Postexercise energy metabolism was examined in male subjects age 22–35 years in response to three different treatments: a strenuous bout of resistive exercise (REx), a bout of stationary cycling (AEx) at 50% peak VO₂, and a control condition (C) of quiet sitting. Resting metabolic rate (RMR) was measured the morning of and the morning following each condition. Recovery oxygen consumption (RcO₂) was measured for 5 hr following each treatment. Total 5-hr RcO₂ was higher for the REx treatment relative to both AEx and C, with the largest treatment differences occurring early during recovery. There were no large treatment differences in postexercise respiratory exchange ratio values, except for the first hour of recovery following REx. RMR measured 14.5 hr postexercise for the REx condition was significantly elevated compared to C. These results suggest that strenuous resistive exercise results in a greater excess postexercise oxygen consumption compared to steady-state endurance exercise of similar estimated energy cost.

**Key Words:** metabolic rate, weight lifting, calorimetry

Numerous studies have examined the impact of steady-state aerobic exercise on the magnitude and duration of excess postexercise oxygen consumption (EPOC) (2, 5, 7, 11, 12, 15, 16, 18). Research in this area suggests that only steady-state exercise of extended duration or significant intensity can elevate metabolic rate for several hours after exercise (2, 7, 16). Although it is known that exercise intensity is the major determinant of EPOC, few studies have addressed the impact of intermittent resistance exercise on the postexercise energy expenditure. In two earlier studies (20, 24), we found collegiate wrestlers to exhibit significantly higher resting metabolic rates at 12–15 hr following wrestling practice (which was characterized by non-steady-state exercise with a significant anaerobic component) compared to a control group of nonwrestlers who engaged in low- to moderate-intensity aerobic training.

Recent studies (6, 13, 25, 29) have emphasized the importance of substrate utilization and nutrient partitioning in the development of obesity. A low daily rate of fat oxidation may be a risk factor for subsequent weight gain. Several
authors have reported increased fat oxidation (as determined by reductions in respiratory exchange ratio [R]) during the hours following a significant aerobic exercise bout (2, 5, 7, 11, 12, 16, 18). The reasons for the enhanced rate of fat oxidation following exercise are not clear. It is conceivable that in these studies the subjects were in an energy deficit situation following prolonged exercise, which would confound the effect of exercise on postexercise R values. Negative energy balance could increase fatty acid oxidation independent of the influence of the exercise bout. Goldberg et al. (15) studied postexercise R, correcting for energy balance under exercise and control conditions, and did not observe reductions in R the morning after exercise. Whether the mode of exercise influences postexercise substrate utilization is unclear. Preliminary results from our laboratory suggest that high-volume resistance exercise may elicit prolonged EPOC and may also lead to postexercise reductions in R (19), although no direct comparisons have been made between resistive and endurance exercise within the same subjects.

The objective of this research was to examine both postexercise energy expenditure and postexercise R values in response to two exercise modes (intermittent resistive exercise, and aerobic, steady-state cycling) of similar estimated exercise energy expenditure. The magnitude and duration of the postexercise energy expenditure elicited by these exercise bouts were determined by comparison to a no-exercise control condition performed by the same subjects.

**Methods**

**Subjects**

Ten male subjects (age 22–35 years) were recruited both from the Colorado State University population of students and from local health clubs to participate in two exercise conditions (resistive exercise [REx] and aerobic cycling exercise [AEx]) and a no-exercise control (C) condition. The physical characteristics of the subjects are shown in Table 1. All subjects denied use of anabolic steroids and were regular participants in both weight lifting and aerobic exercise prior to participation in the study, with a minimum of two sessions per week spent in each of the two modes of exercise. Each subject gave his informed consent to voluntarily participate in this study.

Three subjects completed the REx protocol as part of a study that has already been published (19). Their REx data are included here because these

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Physical Characteristics of 10 Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variable</td>
<td>M</td>
</tr>
<tr>
<td>Age (years)</td>
<td>27</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>179</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>81</td>
</tr>
<tr>
<td>Peak VO₂ (ml · min⁻¹ · kg⁻¹)</td>
<td>52</td>
</tr>
</tbody>
</table>
subjects were willing to complete the AEx and C conditions required of this investigation, which compared recovery from the two different modes of exercise used in this study. Participation in the earlier study would not influence the three subjects’ testing responses in the present study, since for both studies combined, they completed the exact same procedures as the other 7 subjects in the present study. This study was approved by the Colorado State University Human Research Committee. Three subjects withdrew from the study before performing the cycling protocol. The results of the AE treatment, therefore, include only 7 subjects, while the results of the RE and C include 10 subjects.

**Procedures**

For this project we utilized a repeated-measures design with subjects completing each of three conditions: a 100-min bout of strenuous weight lifting (RE), a bout of cycle ergometry (AEx) at 50% of predetermined peak VO₂, and a control condition of quiet sitting. At least 7 days separated each condition, and subjects refrained from exercise on the day prior to participation in each condition. Meals were provided the evening before and throughout the day of each treatment. Subjects were acquainted with the laboratory equipment and study procedures prior to their participation. Table 2 displays the experimental protocol followed by the subjects. While the order of the control versus either of the two exercise treatments was randomly assigned, in every case the subjects completed the RE prior to the AEx treatment (the reason for which will be further explained in this section).

At 18:00 hr on Day 1 a standardized meal (meal composition and energy content are described later in this section) was provided and consumed by 18:30. Subjects were allowed to leave the lab for the night but were instructed not to consume anything but water and to avoid activity until they returned in the afternoon on the following day.

**Table 2 Treatment Protocol**

<table>
<thead>
<tr>
<th>Day</th>
<th>Time</th>
<th>Protocol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1</td>
<td>18:00-18:30</td>
<td>Supper</td>
</tr>
<tr>
<td></td>
<td>6:00-7:00</td>
<td>RMR 1</td>
</tr>
<tr>
<td></td>
<td>7:00-7:30</td>
<td>Breakfast</td>
</tr>
<tr>
<td></td>
<td>10:45-11:15</td>
<td>Lunch</td>
</tr>
<tr>
<td></td>
<td>14:00-14:20</td>
<td>Preexercise metabolic rate</td>
</tr>
<tr>
<td></td>
<td>14:20-16:00</td>
<td>Treatment (RE, AEx, or C)</td>
</tr>
<tr>
<td></td>
<td>16:00-18:00</td>
<td>EPOC</td>
</tr>
<tr>
<td></td>
<td>18:00-18:30</td>
<td>Supper</td>
</tr>
<tr>
<td></td>
<td>18:30-21:30</td>
<td>Postprandial EPOC</td>
</tr>
<tr>
<td>Day 2</td>
<td>6:00-7:00</td>
<td>RMR 2</td>
</tr>
</tbody>
</table>

**Note.** RMR = resting metabolic rate; RE = resistance exercise; AEx = aerobic cycling exercise; C = control condition of quiet sitting; EPOC = excess postexercise oxygen consumption.
morning. At 06:00 hr on Day 2, resting metabolic rate (RMR) was measured for 1 hr by indirect calorimetry. Following RMR measurement subjects were provided a standardized breakfast and 3.75 hr later a light late-morning lunch. Each meal was consumed in less than 30 min. Subjects were allowed to leave the lab between meals to attend class and other sedentary business with instructions to consume nothing but water and to avoid any physical activity. At 13:30 each subject returned to the laboratory and the rate of preexercise resting energy expenditure was measured for 20 min beginning at 14:00 hr. The start of the exercise treatment was adjusted so that the measure of recovery energy metabolism always began at 16:00 hr. Approximately 5 min of postexercise VO₂ values were lost between the end of exercise and the onset of postexercise calorimetry while subjects were placed in a respiratory canopy and instrument wash-in occurred. At 18:00 hr subjects were removed from the canopy and consumed a meal identical to the meal provided the evening before (evening meals were identical for each treatment). At 18:30 hr subjects returned to the canopy, and postexercise, postprandial thermogenesis was measured continuously for the ensuing 3 hr. At 21:30 hr subjects were allowed to leave the laboratory with the same instructions as provided the prior evening. At 06:00 hr the following morning (14.5 hr postexercise) subjects returned to the laboratory and RMR was measured again.

**Peak VO₂.** Prior to each subject’s participation in the experiment, his peak aerobic capacity was determined by a continuous multistage exercise test on a cycle ergometer. The resistance applied to a constant 90 pedal rpm was increased in 0.5-kp increments until volitional exhaustion was reached. Data from the VO₂max test were used to estimate how long each subject should cycle at 50% of peak O₂ consumption to expend approximately the same amount of calories as the resistive exercise treatment.

**Resistance Exercise Condition.** The weight lifting treatment was a single session of high-volume resistance exercise similar to that described by Wallace et al. (26). Subjects performed five sets of 10 different weight lifting exercises for a total of 50 sets. The weight for each lift was initially set at 70% of each subject’s preestablished one repetition maximum. Subjects performed as many repetitions as possible for each set, usually between 8 and 12 repetitions. For several subjects the weight was reduced for the fourth and fifth sets to ensure that a minimum of 8 repetitions was performed. The 10 different lifts were divided into five groups of two lifts emphasizing different muscle groups, and the lifts were serially executed in pairs. A 4-min interval was allowed between the start of one set and the start of the next set of the same exercise. However, within the 4-min interval, subjects performed a set of the other paired lift. Five sets of each of the paired lifts were performed in a 20-min period, with all 10 lifts (5 pairs) performed in 100 min.

The protocol included both upper and lower body lifts with the following order of paired lifts: bench press and bent-over rows, leg extensions and leg curls, military press and sit-ups, biceps curl and triceps extension, and half-squats and lateral raises (arm abductions for deltoids). Subjects were closely supervised and were required to use proper lifting technique to minimize any unfair mechanical advantage and to reduce the possibility of injury. The lifting regimen was similar to the usual routine with respect to workload for the majority of subjects, although some subjects reported that the recovery period between sets was shorter than that to which they were accustomed.
An attempt was made to keep the caloric costs of the cycling and weight lifting treatments somewhat similar. In a preliminary test, two subjects performed the weight lifting protocol wearing a sealed face mask connected to the metabolic cart. Because weight lifting exercise severely disturbs the body’s bicarbonate pools, the R is not always an accurate reflection of whole-body fuel utilization. Furthermore, during exercise that requires significant anaerobic metabolism, oxygen consumption underestimates energy expenditure. Given this scenario as well as the reliance on creatine phosphate and carbohydrate as the major fuels for such exercise, we estimated the caloric equivalent per liter of oxygen to be 5 kcal.

Additionally, we measured oxygen consumption both during the execution of the lift and during the recovery between lifts. The calories expended by each subject, corrected for body size and the amount of weight lifted, were similar between the two subjects. Subject 1 (body weight = 70 kg, exercise volume = 24,160 kg) had a net caloric cost of 534 kcal for the 100-min weight lifting protocol. Subject 2 (body weight = 90.4 kg, exercise volume = 29,383 kg) had a net caloric cost of 695 kcal. Subject 1 expended 11.05 kcal per 500 kg of weight lifted, while Subject 2 expended 11.83 kcal per 500 kg of weight lifted in the 100-min weight lifting session. To estimate the energy cost of the exercise bouts for the 10 subjects in this study, we multiplied the total amount of weight lifted by each of the subjects by the average factor (11.44 kcal per 500 kg) established in the preliminary test.

In 7 of the 10 subjects the R value fell below 0.65 during the first 30 min postexercise, presumably reflecting restoration of bicarbonate liberated during exercise. Since this disallowed use of the Weir equation (27), we determined EPOC magnitude for the first 2 hr after weight lifting by assigning a caloric equivalent per liter of oxygen of 4.8. In every other instance the caloric equivalent per liter of O₂ was determined with the Weir equation (27).

**Aerobic Exercise (Cycling) Condition.** A cycling intensity of 50% of peak VO₂ was used to represent an intensity commonly used for long-duration aerobic exercise. Data from the peak VO₂ test were used to estimate how long each subject should cycle at 50% of peak O₂ consumption to expend approximately the same amount of calories in each exercise treatment. Because we used the estimated caloric cost of the weight lifting session to calculate the cycling time required to expend a similar number of calories, the subjects completed the weight lifting treatment prior to the cycling treatment. During the submaximal cycling exercise, respiratory gases were measured for 5 min of every 15 min of exercise, and small workload adjustments were made to keep the subjects exercising as close as possible to 50% of their peak oxygen consumption.

**Indirect Calorimetry.** The RMR, postexercise energy expenditure, and R values were measured by indirect calorimetry. For measurement of RMR, the subject lay supine on a comfortable bed with his head enclosed in a ventilated canopy constructed of clear plexiglass. The bed was located in a well-ventilated, private, semidarkened room adjacent to the lab with the temperature maintained at 22–23 °C. The canopy was connected to an automated metabolic cart (Horizon MMC, SensorMedics, Yorba Linda, CA) by a hose that passed through a wall and into the lab. The oxygen and carbon dioxide analyzers were calibrated before and after each test using known gas concentrations. The volume transducer was also calibrated using a pump to deliver fixed volumes at three different flow rates,
and temperature was calibrated using a thermometer adjacent to the temperature transducer. All values obtained from indirect calorimetry were standardized for standard temperature and pressure dry (STPD). The reliability of repeated RMR measures in our laboratory has been previously reported with an average coefficient of variation of 3.1 ± 1.3% for RMR and 5.3 ± 2.4% for R (8). For the 1-hr RMR tests, 3-min averages of VO$_2$ and VCO$_2$ from the final 30 min of the test were included for data analysis. Thirty-minute averages were used for data analysis during the 5-hr measured postexercise period.

**Dietary Intake.** Meals were provided to avoid large variations in energy balance and food quotient between treatments. Energy needs were estimated based on body weight and self-reported intakes for each subject. Subjects received an average of 40 kcal · kg$^{-1}$ during the control treatment. Since subjects expended more calories during the exercise compared to control treatments, the additional calories (same composition) for the exercise treatment were provided in the breakfast (average additional 500 kcal) and lunch meals (additional 150 kcal) prior to exercise. Identical evening meals were provided for all treatments, both the evening prior and the day of each treatment, to allow comparisons of the thermic effect of the meal (TEM). Meals were composed of ordinary food and a supplement (Sustacal, Mead Johnson Corp., Evansville, IN). Diet composition was 65% carbohydrate, 15% protein, and 20% fat. The evening meal, provided 2 hr following cessation of exercise or the control condition of quiet sitting, contained an average of 1,300 kcal and reflected 40% of total energy needs during a sedentary day.

**Data Analysis.** The data were analyzed using the Statistical Analysis System (SAS, Cary, NC). For purposes of analyzing the VO$_2$ and R data, the postexercise period was divided into three specific segments: (a) the 2-hr time period immediately following exercise or quiet sitting, (b) the 3-hr time period following ingestion of a meal that was consumed 2-hr following cessation of exercise or quiet sitting, and (c) RMR measured 14.5 hr following cessation of exercise. We examined differences in VO$_2$ and R during the posttreatment periods among the three conditions using a within-subjects repeated measures ANOVA (Condition × Time). Because 3 subjects did not complete the cycling protocol, the analysis was necessarily performed using a univariate approach for unbalanced conditions. The error terms used for the $F$ ratios were selected to account for unequal numbers. For the main effect of treatment, we used the error term for the subject by treatment interaction; for the main effect of time, the subject by time interaction error term was used; and for the treatment by time interaction, the error term used was that of the subject by treatment by time interaction. The level of statistical significance was set at $p < .05$.

**Results**

The mean (±SD) amount of weight lifted by the subjects (Sets × Repetitions × Weight) during the 100-min session of REX was 25,405 ± 5,100 kg, with an estimated mean net caloric expenditure of 588 ± 118 kcal. For the AEx, the subjects averaged 51.5 ± 2.9% of max VO$_2$ for an average total exercise time of 63.6 ± 10.0 min. The average estimated net caloric cost of the stationary cycling for the 7 AEx subjects was 536 ± 77 kcal, which was not significantly different ($p = .52$) from the estimated caloric cost of the REX.
Measures of Postexercise VO₂

Comparison of the 2-hr postexercise VO₂ data for the three conditions is shown in Figure 1. There was a significant time by condition interaction \((f = 11.93, df = 16, 120, p < .0001)\), with post hoc comparisons showing the postexercise VO₂ values to be significantly higher \((p < .05)\) at each of the 30-min time periods for REx compared to control, and for AEEx compared to control at several but not all time points. To determine if treatment differences remained beyond the first hour, we performed an ANOVA only on the second hour’s VO₂ values. There was no significant interaction, but there was a main effect of condition \((f = 12.5, df = 2, 12, p < .01)\). Post hoc comparisons showed the postexercise VO₂ values to be significantly greater \((p < .01)\) for REx than AEEx at 1.5 hr postexercise, but not at 2 hr.

For the 3-hr postprandial period, there was not a significant condition by time interaction \((p = .12)\). However, there was a main effect of condition \((f = 35.3, df = 2, 15, p < .0001)\) and a main effect of time \((f = 43.3, df = 5, 51, p < .0001)\). The significant condition effect and the lack of an interaction suggest that the slopes of the VO₂ responses were not different and that the condition differences remained fairly consistent during the 3-hr period. Post hoc comparisons revealed differences at several time points (see Figure 1). To test whether the treatment differences remained significant at the end of the measured recovery period, we averaged the last hour of VO₂ data during this period (4.5–5.5 hr postexercise; i.e., 2–3 hr postprandial) and found the VO₂ values to be 6.8 ±

![Figure 1 — Five-hour oxygen consumption (ml/min⁻¹) for the same 30-min time points following resistive exercise (REX), aerobic exercise (AEEx), and quiet sitting (C). Significant differences \((p < .05)\) based on post hoc comparisons at individual time points are denoted as lowercase letters (a, b, c).](image-url)
7.4% higher for REx compared to control \((p < .05)\), and 5.5 \pm 4.7% higher for AEx compared to control \((p < .05)\).

When the 2- and 3-hr postexercise periods were totaled, the analysis showed that the 5-hr EPOC following REx was significantly greater than the 5-hr EPOC following AEx. The average \(\text{VO}_2\) values for the 5-hr recovery period for resistance exercise, cycling exercise, and control were 376 ml \(\text{O}_2\) \(\cdot\) min\(^{-1}\), 349 ml \(\text{O}_2\) \(\cdot\) min\(^{-1}\), and 334 ml \(\text{O}_2\) \(\cdot\) min\(^{-1}\), respectively. The elevation of the postexercise metabolic rate during this 5-hr period accounted for an estimated additional 51 \pm 24 kcal expended for REx and 27 \pm 17 kcal for AEx. These \(\text{VO}_2\) and caloric values did not include the first 5 min following exercise when the differences in postexercise metabolic rate compared to the baseline control condition would be highest.

Figure 2 shows the average changes in RMR from the morning before to the morning after each treatment. The repeated measures ANOVA revealed significant treatment \((f = 5.4, df = 2, 15, p < .05)\) and time \((f = 12.8, df = 1, 10, p < .01)\) effects, but the interaction did not reach statistical significance \((f = 1.4, df = 2, 15, p = .27)\). When RMR was measured the morning following the AEx condition, one of the subjects was severely agitated due to an unfortunate personal difficulty and reported significant emotional distress at the time of measurement. His RMR value was 22% higher than the day before. This high value contributed significantly to the elevation and variability of RMR following AEx. Because of the high RMR variability following AEx, we compared only the REx and C conditions using a separate ANOVA. There was a significant interaction \((f = 9.2, df = 1, 9, p < .05)\), indicating a significantly higher RMR 14.5 hr following resistance exercise compared to the control condition.

Figure 2 — Resting metabolic rates measured the morning before and the morning after a day in which subjects participated in either of three different treatments: resistive exercise (REx), aerobic exercise (AEx), or quiet sitting (C).
Measures of Postexercise Respiratory Exchange Ratio

The postexercise R values for each of the three conditions are provided in Figure 3. During the first 2 hr of recovery there was a significant treatment by time interaction \( f = 8.63, df = 16, 120, p < .0001 \). During this period, R was significantly lower following REx than it was following the AEx treatment and C, although the differences were greatly attenuated by the end of the 2-hr period. During the 3-hr postexercise, postprandial period, there was a condition effect \( f = 3.7, df = 2, 15, p = .05 \), a time effect \( f = 3.9, df = 5, 51, p < .05 \), but no condition by time interaction \( f = 0.7, df = 10, 75, p = .75 \). Post hoc tests revealed a significantly lower R for AEx compared to REx and C only at 4.5 hr postexercise. Because the bicarbonate pools are affected by exercise, which results in R values that do not accurately reflect cellular fuel utilization, fat and carbohydrate oxidation rates were not determined during the 5-hr postexercise period.

A comparison of resting R value changes from the morning before to the morning after each treatment showed a significant time effect \( f = 10.5, df = 1, 9, p < .01 \) such that across all conditions, R decreased from Day 1 to Day 2 (Figure 4). Although R decreased to a greater extent following both REx and AEx relative to C, the interaction did not reach statistical significance \( f = 2.0, df = 2, 15, p = .19 \). Macronutrient oxidation rates were calculated from the \( \text{VO}_2 \) and R data obtained during the RMR tests the morning before and the morning after each treatment. Post hoc tests showed that the rate of fat oxidation (as determined from R) significantly increased \( p < .05 \) the morning following REx (morning

![Figure 3](image_url)

Figure 3 — Five-hour respiratory exchange ratio averages for the same 30-min time points following resistive exercise (REx), aerobic exercise (AEx), and quiet sitting (C). Significant differences \( p < .05 \) based on post hoc comparisons at individual time points are denoted as lowercase letters (a, b, c, d).
Figure 4 — Resting respiratory exchange ratio measured the morning before and the morning after a day in which subjects participated in one of three treatments.

prior $3.2 \pm 1.2 \text{ g} \cdot \text{hr}^{-1}$; morning after $4.3 \pm 1.4 \text{ g} \cdot \text{hr}^{-1}$) compared to C (morning prior $3.3 \pm 0.7 \text{ g} \cdot \text{hr}^{-1}$; morning after $3.7 \pm 0.9 \text{ g} \cdot \text{hr}^{-1}$), but the differences in the rate of fat oxidation did not reach statistical significance between REx and AEx (morning prior $3.5 \pm 0.6 \text{ g} \cdot \text{hr}^{-1}$; morning after $4.3 \pm 1.5 \text{ g} \cdot \text{hr}^{-1}$) and between AEx and C. There were no differences in the rate of resting carbohydrate oxidation among groups over time.

Discussion

Major Findings

The present investigation compared the acute changes in energy expenditure and postexercise R following two commonly utilized exercise modes to that of a control condition without exercise. Exercise intensity appears to be the major determinant of the magnitude and duration of EPOC (2). While most studies to date have examined steady-state exercise, Bahr et al. (3) recently reported that 2-min bouts of supramaximal exercise separated by 3-min rest periods produced elevations of postexercise metabolic rate for 4 hr. Drawing on the observations by others that high-intensity resistance exercise is capable of perturbing hormonal milieu (17), reducing skeletal muscle glycogen stores (21), and damaging skeletal muscle cell structure (1), we hypothesized that EPOC following strenuous weight lifting would be of greater magnitude and duration that the EPOC associated with a bout of low-intensity steady-state cycling. We found that resistive exercise of the intensity and duration used in this study is capable of eliciting a higher
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EPOC for at least 1.5 hr following exercise compared to EPOC following a steady-state cycling bout of similar estimated exercise calorie expenditure.

The specific reasons for the greater elevation of postexercise metabolic rate induced by resistive exercise are unclear. Kraemer (17) recently reviewed the endocrine effects of resistive exercise and described elevations in plasma catecholamines, cortisol, growth hormone, and testosterone in response to this type of activity. Exercising involving eccentric muscle contractions has been found by others (1, 9, 14) to induce derangements of skeletal muscle fibrils, membranes, and ion transport. Cannon et al. (10) reported an increased production of the cytokine interleukin-1 in response to eccentric muscle contraction. Any of these perturbations associated with resistive exercise could potentially result in the prolonged elevation of postexercise metabolic rate. The exercise regime used in this study included a significant number of eccentric muscle contractions, which could have resulted in some of the phenomena mentioned previously. However, we made no attempt to identify the influence of eccentric exercise alone on postexercise metabolism.

From a practical viewpoint, our data agree with other studies indicating that the EPOC associated with moderate aerobic exercise is quantitatively small compared to the energy cost of the exercise itself. Clearly, only a minority of individuals participate in resistive exercise of the strenuous nature studied here. In this study the postexercise metabolic response to the resistive exercise bout was not related to the absolute amount of weight lifted by these subjects. Additionally, the metabolic responses varied considerably between individuals, although the relative intensity of the exercise was similar among the subjects.

We also hypothesized that exercise would lead to greater lipid oxidation during recovery and that this would be more apparent following resistive exercise. We formulated this hypothesis based on the observations of others that both activities deplete glycogen stores. We speculated that after exercise and the ingestion of a mixed meal, greater amounts of carbohydrate would be used for glycogen synthesis postexercise, and that the greater nonoxidative glucose disposal would lead to enhanced lipid oxidation. Because exercise requiring significant anaerobic metabolism is fueled to a greater extent by glycolysis, we hypothesized that resistance exercise would manifest a more prominent effect. However, our data do not support the hypothesis that resistive exercise enhances lipid oxidation during the hours immediately after exercise. During the first 2 hr immediately postexercise, REx $R$ values were lower apparently due to replenishment of the bicarbonate pool. During the 3-hr postexercise postprandial period there were only small differences in postexercise $R$ values, which we believe have little clinical relevance.

When we compared the substrate oxidation rates collected during RMR measurements the morning of exercise to the rates collected the morning after exercise, under the REx condition, we found that subjects exhibited a significant elevation in the rate of fat oxidized compared to the control condition. This elevation appeared to result from the combination of the increase in resting $V_O_2$ and a slight decrease in $R$. While it is possible that the elevation in fat oxidation occurred as a direct effect of resistive exercise, it is also possible that this effect occurred because the subjects experienced an acute negative energy balance. We attempted to avoid this by providing the subjects with an additional 650 kcal on
exercise days relative to the control days. However, without 24-hr measurements, an acute state of negative energy balance cannot be ruled out as a factor influencing the substrate oxidation rates the morning after REx.

**Limitations**

It is virtually impossible to match the caloric expenditure between resistive and endurance exercise. Relatively few studies have examined the energy cost of non-steady-state anaerobic activities due to the difficulties in using indirect calorimetry for these measurements. We are well aware that, due to the limitations of indirect calorimetry, our goal of producing similar energy expenditures for the two exercise modes may not have been achieved. Our estimates of the energy expenditure associated with this type of activity based on two subjects do, however, agree with the observations of others (23, 28). Scala et al. (23) estimated the energy expenditure of subjects performing a weight lifting protocol to be 11.5 kcal/min for large muscle group exercises and 6.8 kcal/min for small muscle group exercises including the rest periods between sets. In our study, the estimated caloric cost of the 100 min exercise averaged 7–9 kcal/min including the rest period between sets.

Another possible limitation of the study was the lack of randomized order of the testing conditions. For reasons stated earlier, all subjects completed the REx condition first. It is possible that an order effect could have contributed to the higher metabolic rates following REx compared to C. However, in a previous study of men of similar ages (22), we found no evidence of an order effect when multiple measures of RMR were obtained over several days and weeks, and the reliability of these measures was quite high (mean coefficient of variation = 3.0%).

In an attempt to keep subjects in energy balance, for both REx and AEx conditions, we provided the subjects extra calories to compensate for the additional energy expended in exercise. The additional calories were provided in the breakfast and lunch meals. It is possible that these extra calories provided for the REx and AEx treatments resulted in an elevation of the 2-hr postexercise metabolic rate relative to the control condition owing to a residual dietary-induced thermogenesis during the postexercise period. This situation could result in inflated postexercise metabolic rates for REx and AEx. However, most of the additional calories were provided at breakfast, which was consumed almost 9 hr prior to the EPOC measures. Also, the preexercise metabolic rates were no higher for the REx and AEx conditions relative to the metabolic rate measured for 20 min prior to quiet sitting in the control condition. Thus, we doubt that the extra calories consumed on the exercise days contributed significantly to the higher postexercise metabolic rates on these days.

**Conclusion**

This study suggests that a strenuous bout of resistive exercise of the magnitude reported here may produce an elevation of postexercise energy expenditure during a 5-hr recovery period compared to a similar time period following a condition of quiet sitting. Resistive exercise was also associated with a higher RMR measured 14.5 hr following exercise compared to RMR measured during the control
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condition. Compared to steady-state aerobic exercise (stationary cycling), resistive exercise produced a significantly higher EPOC during the first 1.5 hr following exercise, but oxygen consumption values were not different between the exercise treatments after 1.5 hr into recovery. We also found higher estimated resting rates of fat oxidation (as determined from VO₂ and R) on the morning after resistive exercise compared to the control condition.

References


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