

Postexercise oxygen consumption and substrate use after resistance exercise in women

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ABSTRACT

BINZEN, C. A., P. D. SWAN, and M. M. MANORE. Postexercise oxygen consumption and substrate use after resistance exercise in women. *Med. Sci. Sports Exerc.*, Vol. 33, No. 6, 2001, pp. 932–938. **Objective:** This study investigated the acute effects of 45 min of resistance exercise (RE) on excess postexercise oxygen consumption (EPOC) and substrate oxidation 120 min after exercise in moderately trained women. **Methods:** Ten RE trained women (age = 29 ± 3 yr; ht = 168 ± 8.3 cm; wt = 59 ± 5.7 kg; $\dot{V}O_{2\max} = 38.3 \pm 4.7$ mL·kg⁻¹·min⁻¹) underwent two trials: control sitting and RE. Subjects acted as their own controls in a random counterbalanced design. A 2-d nonexercise period was established between testing trials. Oxygen consumption ($\dot{V}O_2$) and respiratory exchange ratio (RER) were measured continuously by indirect calorimetry before, during, and after exercise and on a separate control day. RE consisted of 3 sets of 10 exercises at 10-repetition maximum with a 1-min rest period between each set. Fingertip samples of blood lactate concentration [BL] were collected immediately postexercise and every 30 min thereafter until [BL] returned to resting baseline values after exercise. **Results:** The overall 2-h EPOC was 6.2-L (RE = 33.4 ± 5.1 L vs control = 27.2 ± 0.3 L), corresponding to an 18.6% elevation over the control period. RER was significantly ($P < 0.01$) below the control RER from minute 30 to minute 120 postexercise (RE = 0.75 ± 0.01 vs control = 0.85 ± 0.01). During the last 30 min of recovery, $\dot{V}O_2$ and [BL] had returned to control/baseline values and fat oxidation was significantly ($P < 0.0001$) higher (29.2 vs 16.3 kcal) after RE compared with the control trial. **Conclusion:** These data indicate that in young RE trained women, acute RE produces a modest increase in $\dot{V}O_2$ during a 2-h recovery period and an increase in fat oxidation. **Key Words:** RESPIRATORY QUOTIENT, RESPIRATORY EXCHANGE RATIO, FAT OXIDATION, EXERCISE RECOVERY, EXERCISE TRAINING, EPOC

Energy expenditure (EE) associated with exercise includes both the energy expended during exercise and the energy expended during the postexercise recovery period. The postexercise elevation in EE above resting levels in the recovery period after exercise is often referred to as excess postexercise oxygen consumption or EPOC (12). Extensive research examining the effects of steady state aerobic exercise on EPOC indicates that exercise intensity has a greater effect on both the magnitude and duration of EPOC than exercise duration alone (2,4,27). In addition, Tremblay and colleagues (29) have found that high-intensity intermittent exercise may favor increased lipid oxidation during recovery when compared with steady state exercise. Resistance exercise (RE) is considered to be intermittent in nature; thus, it may induce a prolonged EPOC and a greater use of fat during recovery.

Although RE is a popular form of high intensity intermittent exercise, understanding the effect of RE on EPOC and fuel utilization is confounded by the considerable diversity of protocol variables commonly employed with previous RE research (7,10,17–19). For example, there are

differences in the type (i.e., circuit training or multiple sets) and intensity (i.e., weight, sets, repetitions, length of rest period) of the RE program, as well as the duration of EPOC time measured. Thus, to be able to clarify the effects of RE on EPOC and substrate utilization, it is of particular interest to utilize a typical high-volume (i.e., 70–80% 1RM, 60-s rest interval) resistance exercise protocol, which is commonly recommended to healthy individuals seeking to increase muscular strength and hypertrophy (16). Also, high-volume RE has been reported to favorably influence lipid metabolism in men (30) and thus may be the most appropriate protocol to use to estimate substrate oxidation after exercise.

Research on the effect of RE on EPOC has usually focused on men. Women have less muscle mass and can lift less volume of weight during RE than men. Thus, their metabolic and EE responses may be different than those reported from men. Although RE is becoming more popular in women for its potential for increasing bone density, lean body mass, and metabolism, few studies have reported the effect of RE on EPOC in women (10,19). Additionally, these studies had small sample sizes (N range = 5–7), did not control for menstrual status, and were not consistent in RE protocol design (circuit training, heavy resistance). Finally, few studies have monitored blood lactate and/or bicarbonate fluctuations that can impact substrate oxidation measures using indirect calorimetry. Thus, the influences of

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a standardized high volume RE protocol, similar to what is typically recommended (16) for strength development, on EPOC and substrate utilization in women remains unknown. The purpose of this study was to determine the acute effect of 45 min RE on EPOC and substrate oxidation in moderately trained women during the same time of their menstrual cycle while monitoring blood lactate concentration.

METHODS

Design/experimental protocol. Subjects performed an initial assessment of $\dot{V}O_{2\max}$ and maximal strength for descriptive purposes and to design the RE protocol. Then each subject participated in two EE trials on separate days. EE measures consisted of three time periods: pretrial (20 min of baseline), trial (45 min of RE or controlled sitting), and posttrial (120-min recovery to assess EPOC). A 2-d nonexercise period was established between trials. Typical dietary intake was determined before exercise testing and participants were then asked not to change their food intake throughout the testing days. Subjects acted as their own controls in a random counterbalanced design.

Subjects. Twelve healthy premenopausal women aged 24–34 yr, who had regularly participated in weight lifting exercise ≥ 1 yr were recruited from the University and the surrounding community. Subjects were nonsmokers, nonobese (body fat $\leq 28\%$), eumenorrheic (self-reported regular menstrual cycle every 28–35 d for the past year), and weight stable (defined as no more than a 2-kg weight fluctuation within the previous 6 months). All regularly participated in weight lifting exercise (2–3 times/wk for 1–1.5 h per session). All subjects were informed of the procedures and risks of the study and signed a written informed consent in accordance with the policies and procedures of the University Human Subjects Institutional Review Board. To ensure that the subjects had no preexisting metabolic or health problems that would influence the results of the study, all subjects completed personal health and medical history, exercise training history, and dietary history questionnaires before participation.

Anthropometric measurements. Body height and mass were measured to the nearest 0.10 cm and 0.10 kg, respectively. Body density was estimated from a seven-site skin-fold assessment (14) obtained at standardized sites according to Harrison et al. (13). Total body fat was calculated from body density using the equation of Siri (28).

Aerobic capacity test. All exercise testing procedures complied with the guidelines for exercise test administration as recommended by the American College of Sports Medicine (1). Initial cardiovascular fitness ($\dot{V}O_{2\max}$) was determined by indirect calorimetry using a modified Balke graded walking protocol on a motorized treadmill (Quinton 4000, Seattle, WA). Metabolic measurements during exercise were obtained by using a two-way nonbreathing valve with the mouthpiece interfaced with a MAX-1 metabolic cart (Physiodyne Instrument Corporation, Quogue, NY). The oxygen and carbon dioxide analyzers were calibrated before and after each test by nitrogen and

two primary standard gases accurate to 0.01%. The pneumotachometer was calibrated using a 3-L syringe to deliver fixed volumes at variable flow rates. The testing protocol consisted of 2-min stages where the subject walked at a brisk walking pace (80–100 m·min⁻¹) with the treadmill grade increasing by 2.5% every 2 min. The subject was asked to continue walking until she achieved volitional exhaustion. A heart monitor (Polar CIC, Inc., Port Washington, NY) was used to monitor heart rate throughout exercise. Considerable encouragement was given to each participant to help facilitate achieving a “true maximal” effort. Oxygen consumption values were considered maximal if there was no longer a rise (≤ 150 mL·min) in oxygen consumption as workload increased. Alternatively, $\dot{V}O_{2\max}$ was assumed if two of the following three criteria were met: respiratory exchange ratio (RER) was over 1.15, blood lactate concentration [BL] was over 8 mM and/or heart rate was within 10 beats of age predicted maximal value (25). Using these criteria, all the subjects achieved a true maximal $\dot{V}O_2$.

Maximal strength test. On a separate day, before the actual testing trials, a multi-station gym (VECTRA Online-1800, Redmond, WA) was used to assess maximal strength (1-RM). Nine exercises: chest press, shoulder press, leg squat, leg extension, leg press, seated row, latissimus dorsi pull-down, biceps curl, and triceps extension were completed. After a brief warm-up, each subject began her single repetition attempt at 50% of subjective predicted maximum, then 75%, 90%, and finally 100% and above if successful. A 2-min period was provided between each successive attempt. The test supervisor was present and discouraged improper procedures such as bouncing the bar on chest, lifting the hips off the bench during bench press, and improper form in the leg squat.

Energy intake assessment. All the women returned two 3-d food records: one before the control trial and one before the RE trial. These food records were analyzed for total energy intake and macronutrient composition (Food Processor, v. 7.0; ESHA Research, Salem, OR). Subjects were encouraged to eat similar diets on the day before each test day to facilitate similar macronutrient profiles before each experimental session.

Metabolic measures. Two EE trials were conducted: a control-sitting trial and a RE trial. The order of each trial was randomized and counter-balanced. Each trial consisted of measuring metabolic variables at three consecutive time periods for a total of 185 min: pretrial resting EE (20 min), during a 45-min RE or control-sitting trial, and for 120 min posttrial (Fig. 1). Measures were made during the follicular phase of each woman’s menstrual cycle (1–7 d after onset of menses), 48 h after the last exercise session and 4–5 h postprandially. Each individual subject was tested at the same time of day although the time of day for testing between subjects varied. Upon arrival at the laboratory, subjects were positioned in a reclining chair and habituated to the open circuit spirometry metabolic analysis apparatus for 20 min. A respiratory mask and two-way, nonbreathing valve (Hans-Rudolph, Inc., Kansas City, MO) was

placed over the subject's face and carefully checked and sealed to prevent air leakage. In addition, a heart rate monitor was placed on the participant. Subjects were advised to remain awake and not to move, "fidget," or talk during the measurement. EE tests were done by indirect calorimetry in a temperature controlled (25–27°C) quiet room. After the 20-min habituation period, resting EE was estimated from a mean of 20 min of continuous gas sampling via indirect calorimetry. Preceding each test, the pneumotachometer and gas analyzers were calibrated as previously described. Resting EE was calculated using the Weir (31) formula.

Resistance exercise/controlled sitting trial. Immediately after the pretrial resting EE measure with the metabolic analysis apparatus still attached, the subject was led through the RE trial or was left to continue sitting for the controlled sitting trial. The metabolic cart was mobile to facilitate changing to different exercise stations with no interruption in measurement. The RE trial consisted of performing three sets of nine different resistance-training exercises (same as noted previously) for a total of 27 sets plus three sets of abdominal crunches within 45 min. Subjects completed 10 repetitions per set at an intensity of 70% 1-RM on the multi-station gym. For each lift, subjects performed the concentric contraction phase in two seconds and the eccentric contraction in 4 s with the test administrator keeping the pace. A 1-min rest period was given between sets.

Upon termination of exercise, each subject resumed the sitting position and expired gas was continuously collected for an additional 120 min into recovery. Metabolic samples were taken in 30-s intervals and then calculations of $\dot{V}O_2$, $\dot{V}CO_2$, and RER were averaged into 5-min time blocks. These are reported at six specific time intervals during recovery: Immediately post exercise (IPE), 5, 30, 60, 90, and 120 min.

Blood lactate determination. Blood lactate concentration [BL] samples were collected in duplicate at baseline (after resting EE), immediately postexercise, and at 5, 30, 60, 90, and 120 min postexercise (Fig. 1). Fingerprick blood samples were taken within 10-s postexercise to determine [BL] level at $\dot{V}O_{2max}$ (YSI 1500 Sport Lactate Analyzer, Yellow Springs, OH). Elevated [BL] and/or increases in acidity of the blood, an accumulation of CO_2 in the tissues, or the changes in buffering of acids invalidates the use of RER as a measure of substrate metabolism during exercise recovery. To avoid the effects of elevated [BL] on substrate oxidation, [BL] was monitored until it returned to a value that corresponded to the steady state baseline values. The

lactate analyzer was calibrated preexercise and twice during the postexercise period using standards of 5 and 15 mM. Preexercise intra- and inter-assay reliability of the blood lactate measurement procedures were $r = 0.987$ and $r = 0.973$, respectively.

Using the $\dot{V}O_2$ and RER data, estimates of EE and substrate oxidation were calculated during baseline, exercise, and recovery assuming a nonprotein respiratory quotient (6). Because there were no significant differences in EE between the baseline (preexercise) condition and the controlled sitting condition, the controlled sitting EE was deemed as a better comparison with the RE recovery trial than the baseline EE trial. EPOC was determined during recovery by subtracting the controlled sitting EE from the postexercise trial values [i.e., $EPOC(L) = \text{recovery EE} - \text{controlled sitting EE}(L)$].

Statistical analyses. Descriptive statistics were used to identify the subject's physical characteristics. Postexercise recovery data were analyzed using a 2 trial \times 6-time period ANOVA with repeated measures. An alpha level of $P < 0.05$ was set to identify significant differences between trials. When significant differences were indicated Scheffe *post hoc* analyses were conducted to determine within trial differences.

RESULTS

Table 1 indicates the basic subject characteristics. Twelve women were tested but because of excessive fidgeting and difficulty with making a tight seal on the facemask, two subjects were dropped from the study. The remaining 10 subjects were similar in age, height, weight, and body fat. Their $\dot{V}O_{2max}$ values verified that they were moderately trained.

There were no significant differences in energy intake values or macronutrient profile before either trial period (i.e., control = $2072 \pm 360 \text{ kcal}\cdot\text{d}^{-1}$; RE = $2092 \pm 248 \text{ kcal}\cdot\text{d}^{-1}$). Average macronutrient composition profile before both days indicated approximately $59.0 \pm 4.0\%$ of energy intake from carbohydrate, $19 \pm 2\%$ from protein, and $21 \pm 2\%$ from fat.

As expected, the RE trial elicited a higher total EE than the control session. Total $\dot{V}O_2$ during the 45 min of RE was $30.9 \pm 2.9 \text{ L}$, which represented approximately 155 kcal of energy expended during exercise compared with $10.2 \pm 1.0 \text{ L}$ (i.e., approximately 50 kcal) during the same duration of controlled sitting. Table 2 indicates the metabolic measures during the various time periods for

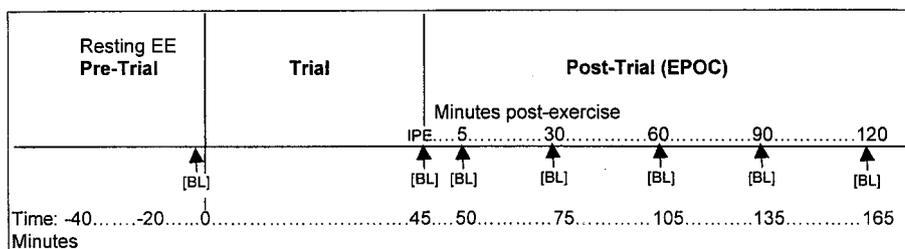


FIGURE 1—Experimental protocol. (EE, energy expenditure; IPE, immediately post exercise; [BL], blood lactate concentration).

the two testing trials. There were no significant differences in baseline and control sitting EE ($\text{kcal}\cdot\text{min}^{-1}$). During the 2-h recovery period, an additional 6.2 L of oxygen (EPOC) was consumed after the RE trial as compared with the controlled sitting trial. Compared with controlled sitting, oxygen consumption was significantly higher after RE at IPE, minute 5 ($P < 0.0001$), and minutes 30 and 60 ($P < 0.05$) (Fig. 2). No significant differences in $\dot{V}O_2$ were found at 90 ($P < 0.06$) or 120-min recovery ($P < 0.128$) compared with the controlled sitting trial.

Figure 3 shows the typical rise in RER immediately postexercise. RER was significantly ($P < 0.001$) below both the control and baseline values from minute 30 to minute 90 postexercise. At 120 min postexercise the RER (0.770) remained significantly lower ($P < 0.01$) than both baseline RER (0.857) and control sitting values (RER = 0.837).

As illustrated in Figure 4, [BL] was markedly increased at IPE and returned to baseline levels by minute 90 of the recovery period. Estimations of substrate oxidation using $\dot{V}O_2$ and RER were made during the last 30 min when [BL] had returned to baseline (Table 2). Using these data, fat oxidation was significantly ($P < 0.0001$) elevated for the final 30 min of the 2-h recovery period after RE compared with the control trial. Estimated fat oxidation the last 30 min after RE was 29.2 kcal compared with 16.3 kcal of fat used during the control-sitting bout. Thus, although total EE was not significantly different for the last 30 min of recovery (35 vs 33 kcal), 79% more fat was used after the RE bout than during control sitting.

DISCUSSION

The major finding in this study was that after RE total EE remained significantly elevated above resting levels for at least 1 h and that fuel utilization postexercise favored lipid oxidation. During the last 30 min of recovery, which corresponded to the time period when [BL] returned to baseline (minute 90–120 postexercise), fat oxidation was significantly increased after RE as compared with the control condition.

These results are in agreement with previous findings in men (7,17,18) and women (19). Melby et al. (17) reported an 11.7% increase in $\dot{V}O_2$ 2 h after 90 min of RE (70% 1-RM) in a small group of men ($N = 6$). This

increase was associated with an increase of about 34 kcal above the baseline resting values. Melby et al. (18) also found that metabolism remained increased for about 1-h postexercise after a 45-min RE bout accounting for an additional 19 kcal above rest. Additionally, Burleson and colleagues (7) reported that $\dot{V}O_2$ was significantly elevated for up to 90 min after a moderate-intensity (60% 1-RM) circuit weight training bout compared with resting baseline in 15 RE trained men. In women, a recent report by Osterberg and Melby (19) indicated that strenuous resistance exercise increased resting metabolic rate (RMR) for 3-h postexercise and RMR was still elevated the next day (16 h postexercise) by about 4.2%. However, other researchers have not found such an extended period of EPOC. For example, Elliot et al. (10) indicated that metabolism was elevated ($P < 0.05$) for only up to 30 min after a moderate-intensity circuit-training exercise in men and women. Inconsistencies in the duration of recovery time measured, possible differences in dietary influences and the vast variability of the RE protocol design may account for the discrepant findings in the literature and limit the generalizability between studies. Finally, most previous RE studies have utilized a preexercise baseline to determine EPOC rather than a controlled sitting period that replicated the conditions present during recovery. Using a preexercise baseline to determine EPOC has limitations because of possible confounding diurnal fluctuations in metabolic rate and/or variations resulting from an order effect or different time exposures to the measurement apparatus. In the present study, we believe that the random assignment and the use of the control-sitting period allowed for more accurate estimations of EPOC than using a preexercise baseline.

Several possible mechanisms have been suggested to explain the components of EPOC (12). A review by Pohleman et al. (22) indicated that the greater the exercise perturbation, the greater the magnitude of EPOC. Such metabolic perturbations may include elevated blood

TABLE 1. Physical characteristics ($N = 10$).

Variable	Mean \pm SD
Age (yr)	29.2 \pm 3.0
Height (cm)	168.8 \pm 8.3
Weight (kg)	59.4 \pm 5.7
BMI ($\text{kg}\cdot\text{m}^{-2}$)	20.8 \pm 1.0
Body fat (%)	15.4 \pm 2.9
Lean body mass (kg)	50.4 \pm 5.0
$\dot{V}O_{2\text{max}}$ ($\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	38.3 \pm 4.7
Blood lactate (rest) (mM)	0.99 \pm 0.02
Volume of weight lifted (kg)	10,843 \pm 1057

IPE, immediately postexercise.

TABLE 2. Estimated metabolic variables at baseline and recovery ($N = 10$).

Variable	Time	Resistance Exercise	Controlled Sitting
Baseline			
$\dot{V}O_2$ ($\text{L}\cdot\text{min}^{-1}$)	-20 to 0 min	0.24 \pm 0.02	0.23 \pm 0.02
RER	-20 to 0 min	0.85 \pm 0.01	0.85 \pm 0.01
Recovery			
$\dot{V}O_2$ total (L)	IPE-120 min	33.4 \pm 5.1*	27.2 \pm 0.3
EPOC (L)	IPE-120 min	6.2 \pm 0.9	—
EE (kcal)	IPE-120 min	157 \pm 12	132 \pm 2
RER	IPE-120 min	0.75 \pm 0.05**	0.85 \pm 0.01
$\dot{V}O_2$ (L)	90–120 min	7.5 \pm 0.6	6.8 \pm 0.3
EPOC (L)	90–120 min	0.65 \pm 0.02	—
RER	90–120 min	0.75 \pm 0.01**	0.85 \pm 0.01
EE (kcal)	90–120 min	35 \pm 1	33 \pm 1
FAT (kcal)	90–120 min	29.2 \pm 1**	16.3 \pm 0.7
CHO (kcal)	90–120 min	6.2 \pm 0.5**	16.7 \pm 0.5

Values expressed as mean \pm SD. IPE, immediately postexercise; EPOC, excess postexercise oxygen consumption; EE, energy expenditure; CHO, carbohydrate; RER, respiratory exchange ratio.

* $P = 0.005$.

** $P < 0.01$.

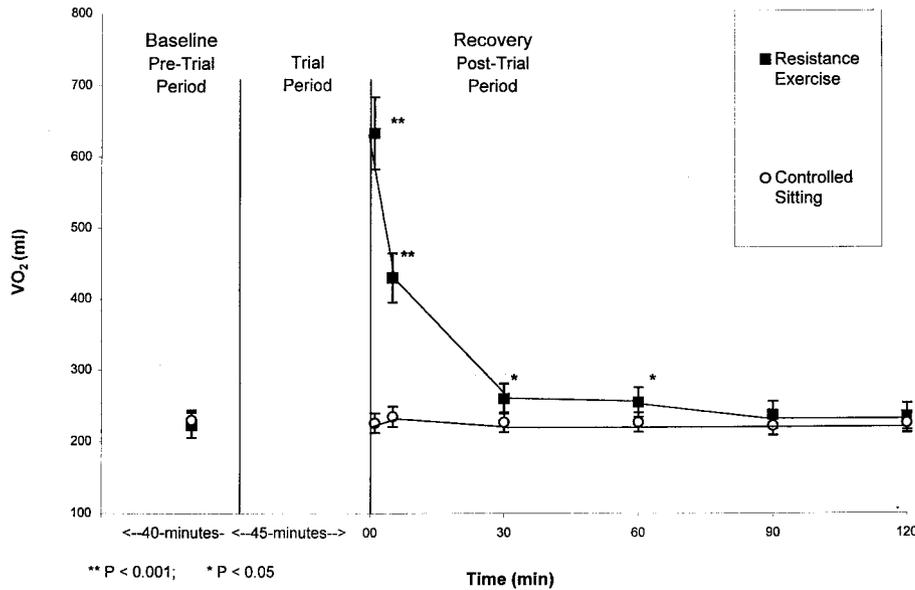


FIGURE 2—Change in total $\dot{V}O_2$ (mL) with time. The pretrial period consisted of 40 min of rest (20-min energy expenditure (EE) measurement), the trial period was either 45 min of resistance exercise (RE) or 45 min of controlled-sitting, and the posttrial period was 120 min of recovery.

lactate levels, resynthesis of glycogen from lactate (15,16), elevated body temperature, phosphagen resynthesis (5,12), elevated catecholamines, and residual hormonal effects (23). An increase in lipid metabolism during EPOC may also contribute to prolonging EPOC (2,8,11). Bahr et al. (3) suggest that the energy cost of the triglyceride-fatty acid cycling rate during recovery may account for close to 50% of the prolonged EPOC.

In the present study, we were cognizant that changes in the acidity of blood, an accumulation of CO_2 in the tissues, or changes in buffering of acids might invalidate the use of RER as a estimate of substrate metabolism. Although we did not measure blood pH or bicarbonate levels, such changes in pH have been shown to parallel lactate concentration (25). Previous studies investigating RE and EPOC have not reported measures of [BL] throughout the recovery period (8,17–19). Thus, the failure to measure [BL] might limit the use of RER to

estimate substrate oxidation and energy expenditure. Phe-lain and colleagues (21), who estimated substrate oxidation after low- and high-intensity (50% vs 75% $\dot{V}O_{2max}$) cycling exercise in women, noted that blood bicarbonate stores had returned to baseline by 35 min after high-intensity cycle ergometry. In this study, [BL] was monitored until it returned to a value that corresponded to the steady state baseline values (minutes 90–120) to avoid the effects of elevated [BL] on substrate oxidation. We assumed that by monitoring [BL] throughout recovery until a time when [BL] was equal to the measurement made during resting baseline, further shifts in bicarbonate were unlikely and that the RER was appropriate to use for estimation of substrate oxidation. Consequently, in this study estimations of fuel oxidation and differences in EE were made during the last 30 min of recovery.

The precise mechanisms responsible for the shift in fuel utilization after RE are not discernible from this

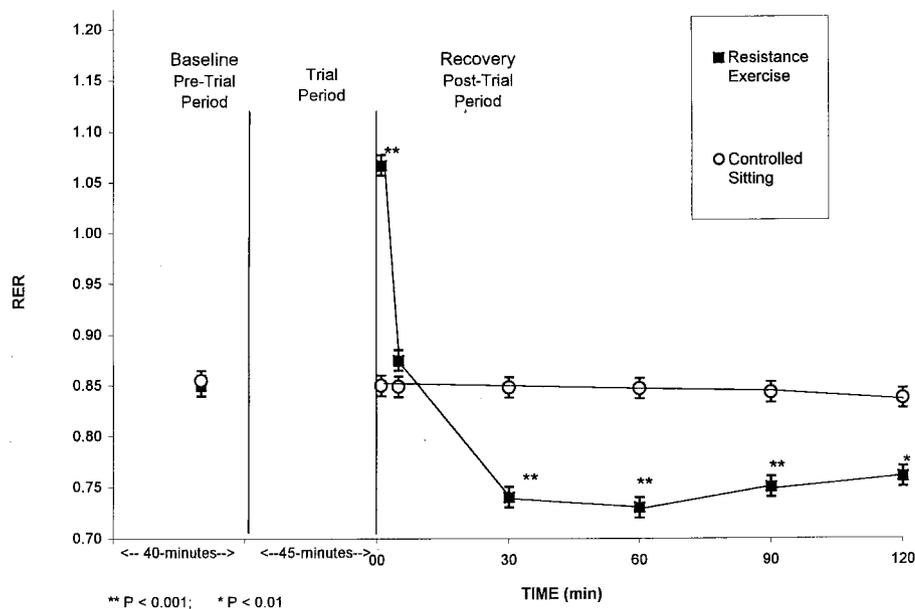
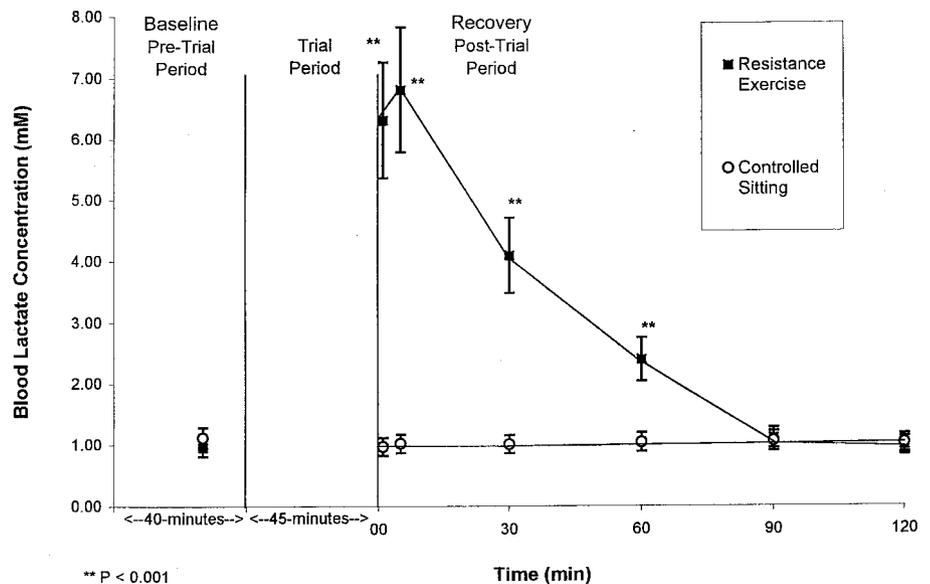


FIGURE 3—Change in respiratory exchange ratio (RER) with time. The pretrial period consisted of 40 min of rest (20 min energy expenditure (EE) measurement), the trial period was either 45 min of resistance exercise (RE) or 45 min of controlled-sitting, and the posttrial period was 120 min of recovery.

FIGURE 4—Change in blood lactate concentration [BL] with time. The pretrial period consisted of 40 min of rest (20 min energy expenditure (EE) measurement), the trial period was either 45 min of resistance exercise (RE) or 45 min of controlled-sitting, and the posttrial period was 120 min of recovery.



study. It is known that as exercise intensity increases the contribution of fat as a fuel source decreases, resulting in a greater reliance of carbohydrate utilization during exercise (5,12,26). Thus, the performance of strenuous RE is dependent on the anaerobic metabolism of phosphocreatine and glycogen for energy leading to a depletion of glycogen stores (20). During recovery from exercises that result in glycogen depletion, lipid becomes the predominant fuel, indicating a shift toward elevated fat oxidation while sparing carbohydrate to be used for glycogen re-synthesis (5). This shift toward greater fat use during recovery may represent a balancing or counter regulatory mechanism that increases fat utilization to spare carbohydrate. Thus, vigorous, glycogen depleting, “anaerobic” exercise may contribute to a possible lipid deficit, which may then lead to greater lipid oxidation in the postexercise state and an overall change in energy balance.

Resistance strength training programs are becoming more popular with the general public and are receiving more attention from health and fitness agencies (9). RE is being touted for its potential health benefits including increased lean body mass, decreased body fat, increased basal metabolism, and increased bone mineral density (9,24). The results from this study suggest that an acute bout of a typical RE session also facilitates a small increase in fat oxidation in moderately trained, young women. It is necessary to determine whether this effect is only a short-lived acute response or whether it can be sustained with daily exercise training. Also it is important to recognize that the actual

amount of fat oxidized is very small and may be physiologically insignificant in terms of any meaningful change in body fat or body weight regulation.

In conclusion, this study indicated that 45 min of high-volume (70–80% 1-RM, 60-s rest interval) RE can significantly elevate recovery EE for at least 1-h postexercise and that fuel utilization favors fat oxidation. These findings may have important implications in understanding energy balance in exercising women, especially those who participate in strength training. Future prospective research is necessary to determine what clinical role (if any) this shift in the pattern of fuel oxidation during RE recovery may have for programs aimed at altering body fat and facilitating weight control.

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