Effects of resistance exercise bouts of different intensities but equal work on EPOC

M. KATHLEEN THORNTON and JEFFERY A. POTTEIGER

School of Nursing, Georgia Southern University, Statesboro, GA; and Department of Health, Physical Education and Recreation, Virginia Commonwealth University, Richmond, VA

ABSTRACT

THORNTON, M. K., and J. A. POTTEIGER. Effects of resistance exercise bouts of different intensities but equal work on EPOC. *Med. Sci. Sports Exerc.*, Vol. 34, No. 4, pp. 715–722, 2002. **Purpose:** To compare the effect of low- and high-intensity resistance exercise of equal work output, on exercise and excess postexercise oxygen consumption (EPOC). **Methods:** Fourteen female subjects performed a no-exercise baseline control (CN), and nine exercises for two sets of 15 repetitions at 45% of their 8-RM during one session (LO) and two sets of 8 repetitions at 85% of their 8-RM during another session (HI). Measures for all three sessions included: heart rate (HR) and blood lactate (La) preexercise, immediately postexercise and 20 min, 60 min, and 120 min postexercise; and ventilation volume ($\dot{V}_E$), oxygen consumption ($\dot{V}O_2$), and respiratory exchange ratio (RER) during exercise and at intervals 0–20 min, 45–60 min, and 105–120 min postexercise. **Results:** Exercise $\dot{V}O_2$ was not significantly different between HI and LO, but $\dot{V}_E$, [La], and HR were significantly greater for HI compared with LO. Exercise RER for HI (1.07 ± 0.03 and LO (1.05 ± 0.02) were significantly higher than CN (0.86 ± 0.02), but there were no differences among conditions postexercise. EPOC was greater for HI compared with low at 0–20 min (HI,1.72 ± 0.70 LO_2; LO, 0.9 ± 0.65, LO_2), 45–60 min (HI, 0.35 ± 0.25 LO_2; LO, 0.14 ± 0.19 LO_2), and 105–120 min (HI, 0.22 ± 0.22 LO_2; LO, 0.05 ± 0.11, LO_2). **Conclusion:** These data indicate that for resistance exercise bouts with an equated work volume, high-intensity exercise (85% 8-RM) will produce similar exercise oxygen consumption, with a greater EPOC magnitude and volume than low-intensity exercise (45% 8-RM). **Key Words:** ENERGY EXPENDITURE, FEMALES, OXYGEN UPTAKE, RESISTANCE TRAINING

For health benefits, aerobic exercise has been the primary mode of activity recommended to women (29), but recently, resistance exercise has become popular and recognized as part of a well-rounded fitness program (2). Currently, a low- to moderate-intensity regimen of resistance exercise is commonly recommended for women (15). High-intensity resistance exercise, however, may provide additional health benefits, particularly, as it relates to energy expenditure. Resistance training can enhance energy expenditure directly from the exercise, indirectly from increasing lean mass, and from increasing postexercise energy expenditure (28). Determining the exercise and postexercise energy expenditure from varying intensities of resistance exercise could be useful in developing resistance-exercise programs.

Studies investigating higher-intensity compared with lower-intensity (26,30) and/or intermittent compared with continuous exercise (5) have consistently reported a greater excess postexercise oxygen consumption (EPOC) response for higher-intensity and intermittent exercise. Although comparisons are problematic, studies using a similar estimated exercise energy cost (16) or similar exercising oxygen uptake (11) to equate continuous aerobic exercise and intermittent resistance exercise, have indicated that resistance exercise will produce a greater EPOC response. When comparing aerobic cycling, circuit weight-training exercise and heavy resistance exercise, the exercise energy expenditure per min and EPOC were highest for the heavy resistance exercise (14). When comparing varying protocols of resistance exercise (e.g., circuit vs standard set resistance training) the regimen with the highest work volume also has produced the highest EPOC (22). Only one study has compared high and low-intensity bouts of resistance exercise of equal work volume on EPOC (24). No significant difference was found between the two intensities. However, the difference between the two intensities was narrow (60% 1-RM and 75% 1-RM) and may not have been large enough to elicit a treatment effect. Comparing exercise energy expenditure for high- and low-intensity resistance exercise of equal work volume needs further examination.

With routines and equipment that are typically used for the general public, the intensity of resistance exercise that will produce the best overall effect on energy expenditure needs to be determined, particularly as it relates to total energy expenditure. These data could then be used in developing resistance exercise programs to promote the greatest impact on caloric expenditure. If a high-intensity regimen (higher weight loads with lower repetitions) produces greater energy expenditure than a low-intensity regimen (lower weight load with higher repetitions), high-intensity programs can be recommended to individuals. However, if the two exercise intensities have a comparable EPOC, individuals may then choose which regimen they prefer.

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TABLE 1. Subject characteristics ($N = 14$).

Characteristic	Mean ± SD
Age (yr)	26.8 ± 5.0
Height (cm)	164.5 ± 7.3
Body mass (kg)	63.1 ± 4.2
Body fat (%)	17.6 ± 2.8
Maximal oxygen consumption ($L \cdot min^{-1}$)	2.87 ± 0.39

The purpose of this study was to compare the postexercise energy expenditure from bouts of low-intensity resistance exercise and high-intensity resistance exercise that have been equated for work volumes. The primary research questions were: 1) will an acute bout of high-intensity resistance exercise compared to low-intensity resistance exercise of equal work volume, stimulate a greater EPOC; and 2) will variables that help explain EPOC (ventilatory volume ($\dot{V}_E$), blood lactate concentration ([La]), heart rate (HR), and respiratory exchange ratio (RER)) be augmented for postexercise intervals of 0–20 min, 45–60 min, and 105–120 min? These variables are known to explain EPOC and were feasible for measurement in this study. A secondary research question was: Will high-intensity resistance exercise compared to low-intensity resistance exercise of equal work volume, produce similar responses for oxygen consumption ($\dot{V}O_2$), $\dot{V}_E$, HR, [La], RER, and rating of perceived exertion (RPE) during exercise? Although body temperature and hormonal variables would have been useful in examining the mechanisms behind EPOC, they were not included in this study due to limited resources.

METHODOLOGY

Subjects. Fourteen healthy eumenorrheic female volunteers participated in the study. Subjects had at least 6 months experience performing resistance exercise (at least 2 $d \cdot wk^{-1}$ in the 6 months before starting the study), and were familiar with the exercises and equipment used in this study. Exclusion criteria were pregnancy, cigarette smoking, the presence of glandular disorders or hormone therapy, or consumption of medications that could affect metabolism. Subjects taking oral or injectable birth control were included in the study. Subject demographics are presented in Table 1. The study sample was homogenous, composed of young women of average height and body mass, with a higher than average cardiorespiratory fitness and a lower than average percent body fat for their age group (1). Before participation all subjects signed an informed consent form and completed a health and exercise history questionnaire in accordance with guidelines set forth by the Advisory Committee for Human experimentation at the University of Kansas. All subjects were judged healthy according to guidelines established by the American College of Sports Medicine (1).

Research design. Each subject made four visits to the Exercise Physiology Laboratory. During the first visit, body composition and cardiorespiratory fitness level were determined as descriptive measures, and muscular strength was determined for calculating weight loads for the treatment sessions. The next three visits involved a no exercise control testing session (CN) and two treatment sessions: low-intensity resistance exercise (LO) and high-intensity resistance exercise (HI). Although all subjects were not tested the same time of day, each subject completed these three sessions at the same time of day. The CN session was done first, one treatment session was completed the following day and the second treatment session was completed 3–7 d later. The treatment and control sessions occurred between days 3 and 11 of the subject's menstrual cycle as calculated by calendar day estimation (32). The two exercise treatment sessions were randomly assigned to subjects in a counterbalanced order. The control session was completed first so that the exercise sessions could easily be completed within the specified time period of the menstrual cycle. Treatment sessions involved performing two sets of nine resistance exercises with an equated work volume but different exercise intensity. Subjects were instructed to be well hydrated, 3 h postprandial having refrained from caffeine for 24 h, and strenuous exercise for 48 h before all testing sessions.

Body composition. Hydrostatic weighing was performed to determine body density. Subjects were weighed on an electronic scale in their bathing suit with weight recorded to the nearest 0.1 kg. Eight trials of underwater weight were performed with the mean of the three heaviest trials used to calculate body density. Residual lung volume was determined using the nitrogen dilution technique (35). Body density was calculated (9) and percent body fat was computed using the Siri equation (31).

Cardiorespiratory fitness testing. Using the Bruce protocol (10) subjects performed a graded exercise test to exhaustion on a treadmill ergometer to determine maximal aerobic power. During testing, expired gases were collected using a two-way low-resistance breathing valve (Hans-Rudolph Inc., Kansas City, MO) and analyzed using a SensorMedics 2900 metabolic measurement cart (SensorMedics Corporation, Yorba Linda, CA). The metabolic cart was calibrated before each test according to the manufacturer specifications. RPE was recorded during the last 15 s of each stage using Borg's 15-point scale (7). Heart rate was determined at the end of each stage using a Polar telemetric system (Polar CIC. Inc., Port Washington, NY). The test was considered maximal if the subject met any three of the four following criteria: plateau of the oxygen consumption (<2.0 $mL \cdot kg^{-1} \cdot min^{-1}$) with an increase in exercise intensity; attainment of a respiratory exchange ratio of 1.10 or greater; plateau of heart rate within ± 10 $beats \cdot min^{-1}$ of age predicted maximum heart rate; and exhaustion or an RPE of 18 (18).

Muscular strength. Each subject performed an 8-RM strength test for 9 exercises on Universal Power Circuit resistance exercise machines (Cedar Rapids, IO). The exercises were performed in the following order, biceps curls, shoulder press, chest fly, bench press, latissimus pull-down, triceps extension, leg curl, leg press, and leg extension. After a warm-up with low weight, each subject performed each exercise beginning with 60% of her perceived 8-RM for 8 repetitions. Incremental increases of 5 kg were completed until the 8-RM was reached. The 8-RM was defined

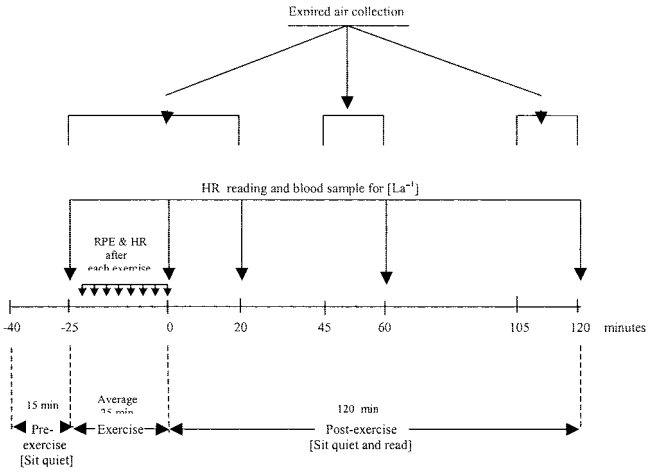

FIGURE 1—Testing schematic.

as the maximum amount of weight that could be lifted through a full range of motion 8 times. The exercises selected are common resistance training exercises used by and recommended for the general public (15).

Resistance exercise sessions. A schematic of the exercise testing is presented in Figure 1. Upon arrival for testing, height and weight were measured. The telemetric heart rate monitor was strapped to the subject at a level just below the breasts and remained in this position throughout testing. Subjects sat quietly for 15 min before exercise to allow for the metabolic rate to stabilize. After controlled light stretching, subjects performed the nine resistance exercises. A mouthpiece was fitted on the subject that was attached to a two-way breathing valve and gas hose connected to a calibrated SensorMedics 2900 (Yorba Linda, CA) metabolic measurement cart. As the subjects moved from one weight machine to another the metabolic cart was rolled along with them. Intensity level was calculated as a percent of the subjects' 8-RM. The HI session involved performing two sets of 8 repetitions at 85% 8-RM, while the LO session involved performing 2 sets of 15 repetitions at 45% 8-RM. Each set required 20–60 s to complete and was followed by a 60-s rest period. The rest periods were determined from pilot testing and were selected to elicit a time period that allowed for recovery between sets but maintained an elevated exercise induced metabolic rate. The entire exercise session lasted approximately 23 min for HI and 26 min for LO. The actual exercise time was approximately 6.9 min for HI and 8.3 min for LO. The primary investigator ensured that correct form was maintained and gave verbal encouragement during exercise. At the end of each exercise, RPE and HR were recorded. When the exercise session was complete, subjects immediately sat in a chair quietly for 20 min while they remained connected to the metabolic cart for expired gas collection. At the end of this 20-min period subjects were disconnected from the metabolic cart and the mouthpiece was removed. The mouthpiece was reapplied and subjects were reconnected to the metabolic cart for expired gas collection 45–60 min postexercise and 105–120 min postexercise. Immediately before exercise, immediately postexercise, and at 20, 60, and 120 min postexercise, heart rate was recorded and blood was collected for lactate analysis. Throughout the postexercise measurement period subjects sat quietly in the laboratory, and during periods between measures the subjects were allowed to read or sit quietly. Measurements were not done the entire 2-h postexercise period to reduce discomfort by subjects and to avoid fidgeting that could falsely elevate EPOC levels (21).

Control testing. A no-exercise control session (CN) was conducted to account for diurnal variations and to avoid the possibility of false elevations of metabolism due to preexercise arousal (21). The CN session was used as a baseline to calculate EPOC. Preexercise, exercise, and postexercise measures during this session were identical to those of the treatment sessions, except that instead of exercising, subjects sat quietly. Our day-to-day variance for the no-exercise measurement of resting metabolism was 3.3%

Determination of EPOC. EPOC was calculated using a computer software program (Matlab for Windows, V 4.2c0.1, The Mathworks, Inc., 1994) that used the average $\dot{V}O_2$ (205.5 ± 29.6 mL·min^{-1}) determined from the CN session. The oxygen consumption was plotted against time and the area under the curve was determined using the trapezoidal rule.

Calculation of caloric values. Strenuous exercise greatly influences blood pH. An increased exhaling of CO_2 and bicarbonate buffering, to help normalize acid-base balance, will affect accurate interpretation of the RER (17). Therefore, the Weir equation for calculating substrate utilization was not used for calorie determination. Instead, an RER value greater than 1.0 during exercise was assumed to be all carbohydrate substrate use and so 5 kcal·LO_2^{-1} was applied to calculate calories expended during exercise. For the postexercise measurements, a mix of carbohydrate and fat substrate use was assumed so 4.8 kcal·LO_2^{-1} was applied to calculate caloric expenditure during this period (25).

Blood collection and analysis. Blood lactate concentration was determined using a capillary blood sample obtained from the fingertip. Approximately 75 μL of blood was collected into a heparinized capillary tube. Blood lactate concentration was determined using a calibrated YSI 1500 L-Sport Lactate Analyzer (Yellow Springs, OH).

Dietary journal. To account for unexpected fluctuations of nutrient intake, each subject kept a dietary journal of foods consumed 24 h before the control and treatment testing days. On these days, subjects were asked to eat comparable foods in like amounts at similar times to avoid fluctuations that could affect substrate metabolism and metabolic rate. Each dietary record was analyzed for daily caloric intake (kJ) and the percentage of macronutrients using a nutritional software program (Nutrition Data System 2–92, Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN; Food Database version 13A; Nutrient Database version 2B). Daily energy intake was considered essentially equal if the total for each day measured was within $\pm$ 200 kJ.

Statistical analysis. Data were summarized as group means with variance presented as standard deviations. Descriptive statistics of demographic data were evaluated to determine homogeneity of the sample. The level of significance for all tests was set at $P \leq 0.05$. For each multiple analysis of variance (MANOVA) follow-up analysis of variance (ANOVA) and *t*-tests were conducted. All *post hoc* *t*-tests were evaluated using a Tukey test for equal variances, a Dunnet C test for unequal variances, or a Dunnet test when the control group was included in the analysis. To control for Type I error, the Holms Sequential Bonferroni Correction Procedure was used for evaluating the MANOVA tests and the standard Bonferroni was used for the multiple *t*-tests used to evaluate the EPOC. All data were analyzed using SPSS (v9.0).

An ANOVA was conducted for preexercise HR and [La] to test for preexercise differences in the treatment and control sessions. A MANOVA was conducted for exercise and another for postexercise with the variables ($\dot{V}_E$, HR, RER, and [La]) to determine differences between the HI, LO, and CN. Dependent *t*-tests were conducted for exercise RPE and for $\dot{V}O_2$ to determine whether there was a difference between HI and LO.

Independent *t*-tests were conducted to evaluate the EPOC (LO_2), for each time period (0–20 min, 45–60 min, and 105–120 min) after the exercise session for HI and LO. A linear regression was conducted with $\dot{V}_E$, HR, and [La] and RER as predictor variables and EPOC as the criterion variable to determine the contribution of variance accounted for by each predictor. An ANOVA was done to determine whether the $\dot{V}O_2$ at the end of the 2 h postexercise (average $mL \cdot min^{-1}$ of the last 5 min) for HI and LO were significantly different from the control $\dot{V}O_2$ (average $mL \cdot min^{-1}$).

RESULTS

Exercise. There was no significant difference for preexercise HR ($F = 0.858$ $P = 0.432$) and [La] ($F = 0.723$ $P = 0.429$), indicating similar metabolic conditions for each testing session. The exercise MANOVA was significant (Wilks lambda = 0.092, $F = 20.670$, $P = 0.000$), with the multivariate η^2 0.697 quite strong. Follow-up ANOVAs on each variable were significant (HR $F = 98.725$ $P = 0.000$; [La] $F = 64.222$ $P = 0.000$; $\dot{V}_E$ $F = 101.546$ $P = 0.000$; RER $F = 56.198$ $P = 000$). *Post hoc* pairwise comparisons revealed that the exercise HR and [La] were significantly higher for the HI compared with the LO and for both HI and LO compared with the CN (Fig. 2). Although the $\dot{V}_E$ was significantly higher for HI and LO compared with the CN, there was no significant difference between HI and LO (Fig. 3). The mean RPE was significantly greater ($t = 4.098$ $P = 0.000$) for the HI (14.0 ± 1.49) than LO (11.6 ± 1.60). There was no significant difference between exercise $\dot{V}O_2$ for each exercise intensity, but this was expected because the total work volume for the HI and LO exercise condition was equal. The total work volume for HI was 4342.7 ± 754.5 kg and for LO was 4326.5 ± 753.5 kg. The weight lifted per unit of exercise time was 629.4 ± 109.3 $kg \cdot min^{-1}$ for HI, and this was significantly different from the LO condition of 521.3 ± 90.8 $kg \cdot min^{-1}$. The total energy expenditure during exercise and recovery between sets was 63.7 ± 7.0 kcal for HI and 71.7 ± 7.1 kcal for LO. These values were not significantly different.

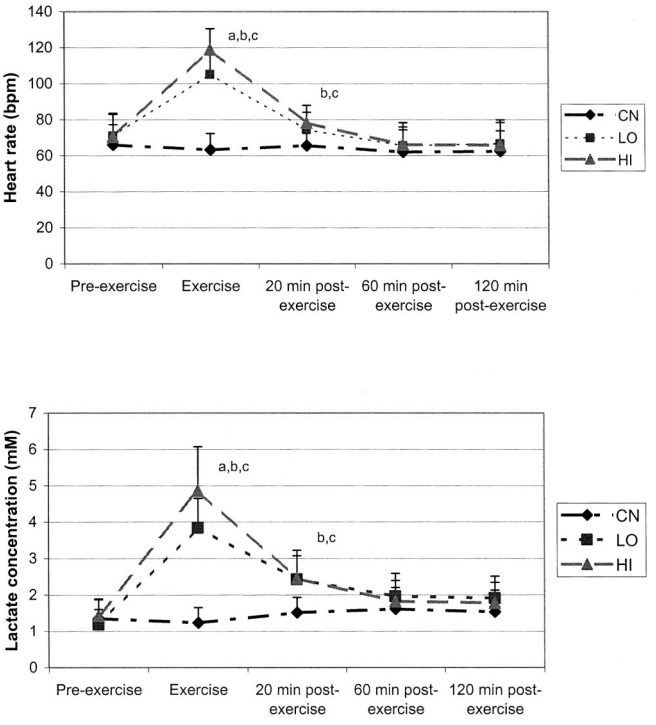

FIGURE 2—Heart rate and blood lactate concentration for high-intensity (HI), low-intensity (LO), and control (CN) sessions preexercise, exercise, and postexercise. Significant differences were observed among the conditions ($P \leq 0.05$); a = HI significantly greater than LO; b = HI and LO significantly greater than CN; c = significant difference from exercise to 0–20 min and from 0–20 min to 45–60 min for HI and LO. Values are means and SD ($N = 14$).

Postexercise. The 0–20 min, 45–60 min, and 105–120 min EPOC values were significantly greater for the HI compared to LO condition (Fig. 4). Because $\dot{V}O_2$ was not measured continuously for the 2-h postexercise period, we are only reporting the approximate energy expenditure from the measured EPOC. The energy expenditure for the EPOC was 11.0 ± 1.9 kcal for HI and 5.5 ± 1.3 kcal for LO.

An ANOVA comparing the CN $\dot{V}O_2$ (average $mL \cdot min^{-1}$) and the postexercise $\dot{V}O_2$ (average of min 115–120 $mL \cdot min^{-1}$) for the HI and LO exercise sessions revealed no significant difference ($F = 0.721$ $P = 0.493$). This indicates that the $\dot{V}O_2$ was not significantly elevated above baseline levels at 2 h postexercise (Fig. 5).

The Wilks lambda was significant (lambda = 0.239 $F = 8.362$ $P = 0.000$, η^2 0.511) for the first postexercise period (0–20 min) for the HR, $\dot{V}_E$, [La], and RER variables. Follow-up tests showed a significant ANOVA for HR ($F = 5.034$ $P = 0.017$, η^2 0.223), [La]($F = 8.980$ $P = 0.001$, η^2 0.339), and $\dot{V}_E$ ($F = 30.854$ $P = 0.000$, η^2 0.638) but not RER ($F = 2.383$ $P = 0.107$, $\eta^2$0.120). The effect sizes were small except for $\dot{V}_E$, which was moderate. *Post hoc* pairwise comparisons revealed differences between the exercise and control sessions, but not between the HI and LO, except for

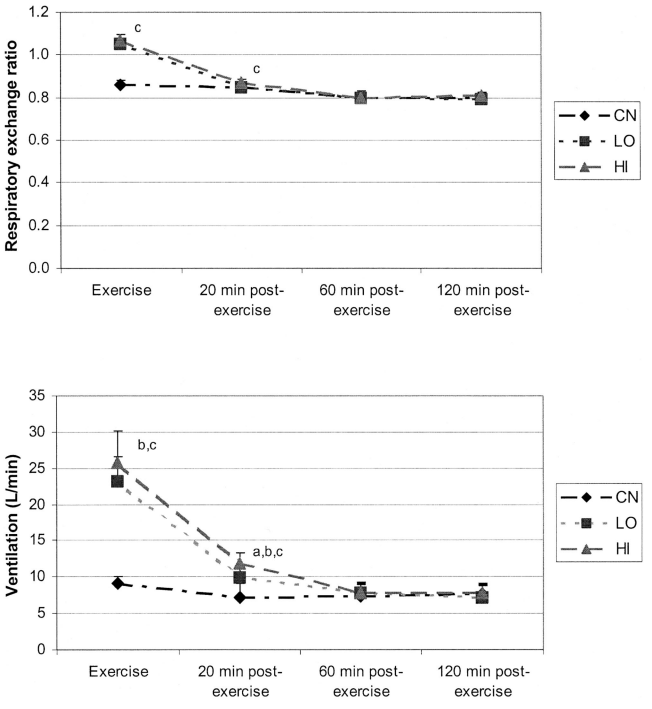

FIGURE 3—Respiratory exchange ratio and minute ventilation for high-intensity (HI), low-intensity (LO), and control (CN) sessions for exercise and postexercise. Significant differences were observed among the conditions ($P \leq 0.05$); a = HI significantly greater than LO; b = HI and LO significantly greater than CN; c = significant difference from exercise to 0–20 min and from 0–20 min to 45–60 min for HI and LO. Values are means and SD ($N = 14$).

$\dot{V}_E$. Only $\dot{V}_E$ was significantly elevated for HI compared to LO. The MANOVAs conducted for the second and third postexercise periods did not show a significant difference for any variable between the HI, LO, or CN. This indicates that all postexercise variables had returned to near baseline levels after 20 min but before 1 h. Pairwise comparisons for $\dot{V}_E$, HR, [La], and RER showed a significant difference for both high and low intensity between the postexercise time 0–20 min and 45–60 min but not between 45–60 min and 105–120 min. These findings suggest that these variables had returned to near baseline levels within 1 h postexercise.

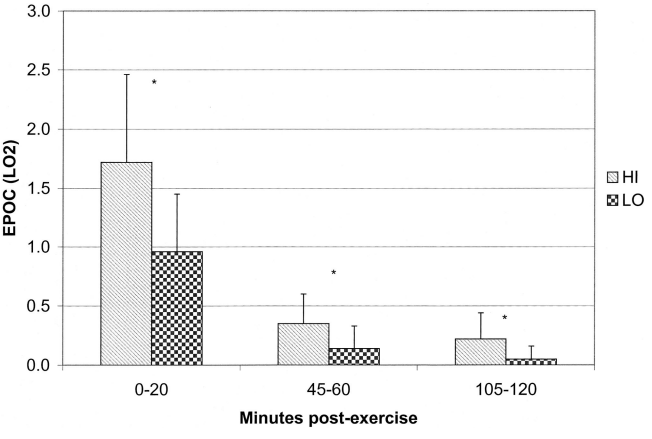

FIGURE 4—Excess postexercise oxygen consumption for the HI- and LO-intensity exercise sessions. *Significant difference between conditions ($P \leq 0.05$). Values are means and SD ($N = 14$).

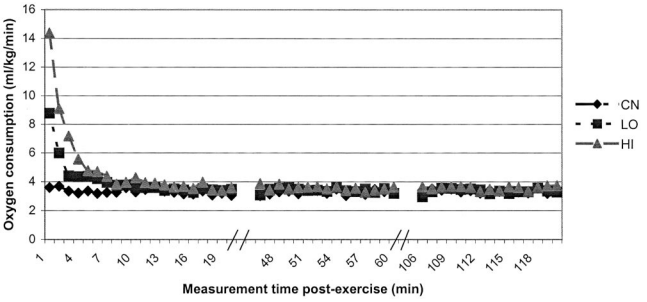

FIGURE 5—Mean oxygen consumption ($mL \cdot kg^{-1} \cdot min^{-1}$) for the 50 min postexercise measurement (0–20 min, 45–60 min, and 105–120 min) for high-intensity (HI), low-intensity (LO) and control (CN) sessions. Values are means, the standard deviation bars have been omitted to provide clarity ($N = 14$).

A linear regression analysis with RER, HR, [La], and $\dot{V}_E$ as predictor variables on EPOC revealed an $R^2 = 0.358$ ($P = 0.002$) with $\dot{V}_E$ being the only significant predictor. The $\dot{V}_E$ variance accounted for 28.8% (R^2 change) of the EPOC, whereas the other variables each contributed less than 1% of the variance accounted for. The standardized beta weights for each variable were $\dot{V}_E$ $B = 0.556$, HR $B = 0.099$, [La] $B = 0.048$, and RER $B = 0.078$.

Total daily energy intake 24 h before each of the exercise and control testing days was within ± 200 kJ (data not reported). This indicates there was no dietary affect that influenced substrate utilization or metabolic rate for each of the testing days.

DISCUSSION

The main finding of this investigation is that EPOC is greater after high-intensity resistance training compared with lower-intensity resistance training when the two sessions are equal in training volume. The EPOC was greater for each postexercise time period with the greatest magnitude occurring during the first 5 min after exercise (Fig. 5). This result occurred even though the exercise $\dot{V}O_2$ was similar for both intensity levels. The increased EPOC following HI was likely caused by a greater metabolic disturbance during exercise requiring increased energy expenditure postexercise to bring various physiologic processes back to equilibrium. The EPOC for the rest intervals between each exercise and each set could not be determined accurately, so it was not possible to establish the true total EPOCs for each treatment session. However, because there was no significant difference in the exercise $\dot{V}O_2$ between the HI and LO conditions the between treatment differences in rest interval oxygen uptake were probably minimal.

Exercise responses. The elevated [La], $\dot{V}_E$, RER, HR, and RPE observed in both exercise sessions were expected due to the physiological challenge of exercise. The greater magnitude produced by the HI exercise for HR, [La], and RPE, however, indicates a greater physiologic stress. This added stress was not reflected in a higher exercise $\dot{V}_E$ or $\dot{V}O_2$ but was evident in the higher magnitude of EPOC in HI during the first 20 min after exercise ended.

Postexercise responses. The most pronounced level of EPOC, the rapid component, occurs within 1 h postexercise. Although the precise cause of this response is not completely known, likely factors that contribute include: restoration of muscle ATP and creatine phosphate stores; replenishment of O_2 stores in blood and muscles; redistribution of compartmental ions (increased sodium-potassium pump activity); tissue damage response, increased heart rate and body temperature, and to some extent, lactate removal (3,11,30). Poehlman (27) has indicated that the greater the metabolic disturbance during exercise the greater the EPOC. Higher-intensity compared with lower-intensity activities may cause an even greater response during the rapid EPOC component because of a greater use of anaerobic energy systems and greater work inefficiency during exercise (20). Treuth et al. (33) have demonstrated that 22% more energy is required to perform the same amount of bicycle work at a high intensity compared with a low intensity. Although it is unclear what mechanisms contribute to the inverse relationship between efficiency and exercise intensity, a potential list includes increased utilization of inefficient fast twitch muscle fibers (12), increased recruitment of stabilizing muscles (19), and increased work of the heart and respiratory muscles (19).

Higher levels of sympathetic nervous system activity may also contribute to an elevated metabolic rate postexercise (8). Epinephrine and norepinephrine are strong stimulators of energy metabolism, and, although not measured in the current study, it is possible that higher levels of catecholamines were present after the HI exercise condition. The plasma concentrations of catecholamines increases exponentially with the exercise intensity and demonstrates the same relationship as that observed between the exercise intensity and EPOC (8).

Although still greater for the HI condition, the EPOC values for the 45–60 and 105–120 intervals were much less pronounced (Fig. 4). During the prolonged component of EPOC (>1 h), processes that restore physiological homeostasis continue but occur at a lower level or do not require as large of O_2 uptake as those processes that occur during the rapid component of EPOC. These processes may include substrate cycling of carbohydrate and fat (4), and the effects of cortisol and growth hormone (3,17).

An increased cardiorespiratory workload also may be a contributor to EPOC during both the rapid phase and slow phase. This concept is supported by the increased $\dot{V}_E$ and HR observed at 20 min postexercise for both exercise sessions compared with the control condition. The greater elevation in $\dot{V}_E$ during HI compared with the LO is further support of the greater disturbance in homeostasis during higher-resistance-intensity exercise. This higher $\dot{V}_E$ also could be interpreted as part of the compensation to normalize the depressed pH caused by elevated levels of lactate incurred during exercise. The similar RER levels observed postexercise for the HI, LO, and CN conditions could be interpreted as indicating no difference in substrate utilization. However, because the $\dot{V}_E$ and the lactate levels were still above baseline levels for nearly the first hour postexercise, valid interpretations of the RER cannot be made due to the normalization of acid-base balance (25).

The exercise energy expenditure and total EPOC energy expenditure were not substantial. Also, the EPOC as a proportion of the energy cost of exercise was not particularly high. Therefore, by using the exercise protocols in this study, the energy expenditure would likely not have a significant impact on weight loss. Although not directly comparable due to differences in protocol, these values are similar to those of other studies when work volume was proportionately similar to that used in this study (18,21,24,25).

Few studies have examined the EPOC magnitude and duration in response to an acute bout of resistance exercise. Two studies have found that resting metabolic rate (RMR) remained elevated for 48 h after a moderate to high-intensity acute bout of resistance exercise (13,34). This finding could be interpreted as the ultra slow phase of EPOC. These responses were hypothesized to be due more to protein turnover and tissue repair, rather than to the various responses that contribute to EPOC 2 h postexercise. Additionally, high-intensity resistance exercise (5 sets of 10 exercises at 70% 1-RM for 8–12 repetitions) compared with a control condition produced a substantively significant EPOC where the $\dot{V}O_2$ remained elevated above baseline 2 h postexercise (21). The elevated $\dot{V}O_2$ at 2 h postexercise at in disagreement with our findings, but the exercise work volume was much higher in the previous study. Our findings of a return to baseline $\dot{V}O_2$ within 2 h postexercise has also been recently supported by Binzen et al. (6). In this study of 10 resistance-trained women, postexercise $\dot{V}O_2$ had returned to baseline during the last 30 min of recovery after a resistance-training exercise session that consisted of 3 sets of 10 exercises performed at the 10 RM.

When comparing an acute bout of aerobic and resistance exercise with an equated energy expenditure, the resistance exercise produced a significantly higher EPOC (11). The resistance exercise (2 circuit sets of 8 exercises at 60% 1-RM for 8–12 repetitions) was considered to be a higher-intensity activity than the aerobic exercise (cycling at 45% $\dot{V}O_{2max}$ for 27 min). Although comparing aerobic and resistance exercise may be problematic, this study has similarities to our current investigation in that there was an equated energy expenditure during the exercise sessions and that the higher-intensity activity produced a greater EPOC. A similar study conducted by Elliot et al. (14) compared aerobic exercise (40-min cycling 75% $\dot{V}O_{2max}$), circuit resistance exercise (4 sets, 8 exercises, 15 repetitions at 50% 1-RM) and heavy-resistance exercise (3 sets, 8 exercises, repetitions to exhaustion at 80–90% 1-RM). Elliot et al. reported that exercise energy expenditure was highest for the cycling and circuit resistance training, but the heavy-resistance exercise produced the greatest EPOC. Although exercise work volumes were not reported, Elliot et al. postulated that the amount of working muscle mass was the prominent variable that produced the greatest EPOC. It seems reasonable that a higher intensity could be viewed as similar to the increased working muscle mass (increased

motor unit recruitment), and, therefore, our results were similar to those reported by Elliot et al.

Murphy and Schwarzkopf (23) compared two very different protocols of resistance exercises, 3 circuit sets of 6 exercises for 12–10^{-8} repetitions of 50% 1-RM at each circuit and 3 sets of 6 exercises performing repetitions to exhaustion at 80% 1-RM. The work volume of each session was reported to be similar, but the intensity (reported as weight lifted/unit of time) of the circuit exercise was greater. The higher-intensity session produced a significantly higher EPOC, but the $\dot{V}O_2$ returned to baseline levels in 26 min. Although the protocols were quite different, these results are comparable to ours in that higher-intensity resistance exercise produced the greater EPOC at the end of the exercise session.

The magnitude of EPOC in response to resistance exercise depends on the work volume, the resistance load or stress, and the rest interval (17). When rest periods are altered during resistance exercise and all other factors held constant, a longer rest period results in greater exercise energy expenditure, but a shorter rest period results in an elevated EPOC (18). A shortened rest period could be interpreted as increased stress equivalent to increasing intensity. Because the work output of the two resistance exercise bouts was equal, these results are in agreement with our current data in that higher physiological stress (higher intensity or shorter rest periods) causes a greater EPOC than low physiological stress (lower intensity or longer rest periods) with an equal work volume. At intensities $>$ 50% of $\dot{V}O_{2max}$, regardless of the type or mode of exercise, the higher-volume exercise will consistently produce a greater magnitude of EPOC than other variables, but when work volumes are held constant, the higher-intensity exercise will produce a greater EPOC (20).

In a study similar to the current investigation, Olds and Abernathy (24) found no difference in EPOC between a high- and low-intensity resistance exercise bout with equated work volume and $\dot{V}O_2$ returned to baseline in 1 h. These results are in disagreement with the current study, but several issues should be considered when interpreting their findings. The difference between the intensity level was narrow (12 reps at 75% 1-RM and 15 reps at 60% 1-RM) and may have been too small to elicit a differing response. The authors stated that age range (22–55 yr), a high interindividual difference in EPOC (0.7–27 LO_2), and no EPOC measures the first 3.5 min postexercise (when EPOC is highest) may have been factors that affected their results.

Strengths, limitations, and future research. Strengths of this study compared with other studies reported in the literature included using female subjects as their own control and testing during the most stable time of the menstrual cycle. Although all physiological variables appeared to be equal preexercise, only the HR and La were measured to assure this. Because the $\dot{V}O_2$ was not measured preexercise, there is no certainty that resting metabolism before all three sessions was similar and therefore may be a possible limitation of this study. An additional limitation to this investigation may be the inability to separate work and recovery $\dot{V}O_2$ during the exercise phase of the HI and LO conditions. Measuring and separating work and recovery $\dot{V}O_2$ during short-duration intermittent exercise is limited in part by the mechanical ability of the metabolic measurement instrumentation to differentiate between work and recovery $\dot{V}O_2$. However, in the context of the current study the total work completed and the $\dot{V}O_2$ consumed during the exercise component of both conditions was not significantly different. Therefore, at the beginning of the EPOC measurement, the subjects had similar oxygen consumption, and the differences in the EPOC were likely due to reduced high energy phosphate stores, higher levels of muscle and blood lactate, and elevated levels of cardiorespiratory function.

Future study comparing different intensity levels with equal work volume could be conducted using higher work volumes or applying similar procedures as used here with subjects who are not resistance trained, of lower fitness levels, and/or who are overweight. It is possible that these variables will invoke a more substantial and prolonged EPOC in these populations.

In summary, the data from this investigation suggest that when work volume is equal, a high-intensity (85% 8-RM) resistance exercise bout compared with a low-intensity (45% 8-RM) resistance exercise bout in women will produce a similar exercise energy expenditure but a greater magnitude of EPOC, likely due to a greater disturbance in physiological processes. This response is greatest during the first 5 min postexercise. The EPOC difference 20-min postexercise between the two intensities was small and would not add a substantively significant contribution toward weight loss.

Address for correspondence: Jeffrey A. Potteiger, Ph.D., FACSM, Virginia Commonwealth University, Department of HPER, 817 W. Franklin Street, P.O. Box 842037, Richmond, VA 23284-2037; E-mail: jpottei@mail1.vcu.edu.

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