Excess Postexercise Oxygen Consumption After Aerobic Exercise Training

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Literature examining the effects of aerobic exercise training on excess postexercise oxygen consumption (EPOC) is sparse. In this study, 9 male participants (19–32 yr) trained (EX) for 12 wk, and 10 in a control group (CON) maintained normal activity. VO₂max, rectal temperature (Tₑ), epinephrine, norepinephrine, free fatty acids (FFA), insulin, glucose, blood lactate (BLA), and EPOC were measured before (PRE) and after (POST) the intervention. EPOC at PRE was measured for 120 min after 30 min of treadmill running at 70% VO₂max. EX completed 2 EPOC trials at POST, i.e., at the same absolute (ABS) and relative (REL) intensity; 1 EPOC test for CON served as both the ABS and REL trial because no significant change in VO₂max was noted. During the ABS trial, total EPOC decreased significantly (p < .01) from PRE (39.4 ± 3.6 kcal) to POST (31.7 ± 2.2 kcal). Tₑ, epinephrine, insulin, glucose, and BLA at end-exercise or during recovery were significantly lower and FFA significantly higher after training. Training did not significantly affect EPOC during the REL trial; however, epinephrine was significantly lower, and norepinephrine and FFA, significantly higher, at end-exercise after training. Results indicate that EPOC varies as a function of relative rather than absolute metabolic stress and that training improves the efficiency of metabolic regulation during recovery from exercise. Mechanisms for the decreased magnitude of EPOC in the ABS trial include decreases in BLA, Tₑ, and perhaps epinephrine-mediated hepatic glucose production and insulin-mediated glucose uptake.

Keywords: EPOC, endurance exercise, hormones, rectal temperature, blood lactate

Excess postexercise oxygen consumption (EPOC) is the elevation in oxygen consumption above the resting value during recovery from exercise. The concept that the body will continue to expend a significant amount of energy after a bout of exercise would appear to be very attractive for individuals who exercise to reduce their body mass. Therefore, along with the effects of various independent variables such as exercise intensity (Chad & Quigley, 1991; LaForgia, Withers, & Gore, 2006; Phelain, Reinke, Harris, & Melby, 1998; Sedlock, 1991b), exercise duration (Borsheim & Bahr, 2003; Chad & Wenger, 1988; Quinn, Vroman, & Kertzer, 1994), exercise mode (Borsheim & Bahr, 2003), upper body exercise (Lyons et al., 2007; Sedlock, 1991c; Short, Wiest, & Sedlock, 1996), resistance exercise (Braun, Hawthorne, & Markofski, 2005; Crommett & Kinzey, 2004; Schuenke, Mikat, & McBride, 2002; Thornton & Potteiger, 2002), intermittent exercise (Lyons et al., 2006; McGarvey, Jones, & Petersen, 2005), menstrual status (Fukuba, Yano, Murakami, Kan, & Miura, 2000), spinal-cord injury (Sedlock, Schneider, Gass, & Gass, 2004), and nutritional status (Bahr & Sejersted, 1991; Borsheim, Kien, & Pearl, 2006; Sedlock, 1991a), several studies have been performed to investigate the effects of exercise training on EPOC responses.

Similar to the way training adaptations alter many aspects of metabolism during exercise, oxygen consumption during recovery from exercise is also affected by training and fitness level. However, the effects of training on the magnitude and duration of EPOC have been sparsely investigated, and a consensus has not been reached regarding the likely outcome. Although some researchers observed a faster recovery in trained versus untrained individuals (Frey, Byrnes, & Mazzeo, 1993; Girandola & Katch, 1973; Hagberg, Hickson, Ehsani, & Holloszy, 1980; Short & Sedlock, 1997), others reported either no difference (Brehm & Gutin, 1986; Freedman-Akabas, Colt, Kissileff, & Pi-Sunyer, 1985; Imamura et al., 1999; Sedlock, 1994) or a greater EPOC response in trained participants (Chad & Quigley, 1991; Lecheminant et al., 2008).
Inconsistent results of previous studies may have resulted from methodological shortcomings or discrepancies. For instance, exercise duration differed between trained and untrained participants in one study (Frey et al., 1993), whereas duration of the EPOC measurement was limited to 15 min in another study (Girandola & Katch, 1973). In a study reporting no significant difference between fit and unfit participants (Sedlock, 1994), intensity and duration of exercise may have been too low to yield a distinct difference in EPOC between the two groups. Therefore, a well-designed and prospective study appears to be needed.

Borsheim and Bahr (2003) point out that the effect of training status on EPOC is difficult to examine because comparing groups of different fitness levels exercising at the same absolute intensity means that they are working at different relative intensities (and vice versa), which has been shown to influence EPOC. Participants exercised at the same absolute and relative intensities in only two previous studies, one using a cross-sectional design (Short & Sedlock, 1997) and the other examining VO2 kinetics rather than the total EPOC response (Hagberg, Hickson, et al., 1980). Therefore, a study examining the effects of exercise training on EPOC when measured after exercise at the same absolute and the same relative intensity is warranted.

Mechanisms responsible for changes in EPOC resulting from chronic exercise also remain to be examined further. A greater EPOC response in trained individuals was hypothesized to result from increased fat utilization during recovery (Chad & Quigley, 1991), whereas when a smaller EPOC response was observed in trained participants (Short & Sedlock, 1997), it was explained by lower catecholamine concentrations and a subsequent decline in the rate of muscle glycogen utilization, that is, a sparing effect on muscle glycogen. Training could also influence other mechanisms involved in the elevated VO2 response during recovery, such as blood lactate removal (Frey et al., 1993), ventilation rates (Newsholme, 1978), or core body temperature (Hagberg, Mullin, & Nagle, 1980).

Although several researchers have attempted to elucidate the effect of chronic exercise on EPOC, their findings have been contradictory, and the mechanisms responsible for any changes observed consequent to chronic exercise training are currently unclear. Therefore, this study was designed to further examine the effects of endurance exercise training on EPOC. We measured EPOC after exercise at both the same absolute and the same relative intensities after training to provide a comprehensive examination of EPOC. In addition, we measured pertinent hormones and substrates to gain additional insight into the mechanisms responsible for metabolic alterations during recovery consequent to training. We hypothesized that training would decrease the EPOC response after exercise at the same absolute intensity but would not affect EPOC after exercise at the same relative metabolic intensity.

**Methods**

**Participants**

Twenty men age 18–32 years volunteered to participate. They were apparently healthy as determined by a medical history questionnaire, were nonsmokers, had had no significant changes in body mass for 6 months before beginning the study, and were not engaged in any systematic exercise training. They were randomly assigned to one of two groups: an exercise intervention group (EX; n = 10) or a nonintervention control group (CON; n = 10). One of the 10 participants in EX completed the intervention but did not participate in any of the postintervention (POST) testing. Therefore, data from 9 participants in EX and 10 participants in CON were used in the final analyses. All participants provided written informed consent. The procedures used in this study were approved by Purdue University’s institutional review board.

**Experimental Design**

Participants attended an orientation session, preintervention (PRE) testing, a 12-week intervention, and POST testing. During the orientation session, participants were familiarized with the procedures to be used throughout the study period. Height, body mass (BM), body composition, and maximal oxygen consumption (VO2max) were measured PRE and POST. For the EX group, these measurements occurred approximately 48 hr after the last training session. Approximately 72 hr after the VO2max test, EPOC was measured PRE after a 30-min bout of treadmill exercise at 70% VO2max. Two EPOC trials were administered to EX in a counterbalanced order during POST, that is, after exercise at the same absolute (ABS) VO2 as PRE and after exercise at 70% of POST VO2max—the same relative (REL) metabolic intensity. These two EPOC trials were separated by approximately 48 hr. One EPOC trial at 70% VO2max during POST served as both the ABS and REL exercise challenge for CON because those participants did not engage in exercise training and there was no significant change in their VO2max from the PRE value.

**Experimental Procedures**

**Body Mass and Height.** Participants reported to the laboratory in a postabsorptive state at approximately 8 a.m. Height and BM were determined using a balanced beam scale with an attached stadiometer, with participants wearing shorts and a T-shirt.

**Body Composition.** Relative body fat (BF), fat mass (FM), and fat-free mass (FFM) were determined by hydrostatic weighing. A chair was suspended from a Chatillon scale in the shallow end of a swimming pool. The participant was fully submerged, and mass was recorded after a maximal expiration. Ten trials were performed, and underwater mass was recorded as the average of three trials within 0.1 kg. Residual volume
was determined by oxygen dilution (Wilmore, Vodak, Parr, Girandola, & Billing, 1980) with the participant seated and leaning forward to mimic body position used during hydrostatic weighing. Body density was converted to BF using the equation of Siri (1961). FM and FFM were calculated from BM and BF.

**VO\textsubscript{2max}.** A continuous, graded, multistage treadmill exercise test to exhaustion was used to determine VO\textsubscript{2max}. The protocol was developed based on suggestions of Howley, Bassett, and Welch (1995). The test began with a warm-up at 3.5 miles/hr and 0% grade for 5 min followed by a 1-min rest. For Minutes 1 and 2, speed and grade were 4.0 miles/hr and 0%, respectively. Speed and grade were increased to 4.3 miles/hr and 2%, respectively, for Minutes 3 and 4. After Minute 4, speed and/or grade was increased every minute until Minute 9, when speed was 5.0 miles/hr and grade was 10%. Thereafter, grade remained constant and speed was increased by 0.3 miles/hr every minute until volitional fatigue was reached. VO\textsubscript{2max} was defined as the point at which two of the following criteria were met: <150-ml/min increase in VO\textsubscript{2} with an increase in work rate, heart rate (HR) >90% of age-predicted maximum, or respiratory-exchange ratio (RER) >1.10.

**EPOC Testing.** Participants were transported to the laboratory at 6 a.m. after a 12-hr fast and 48-hr abstention from vigorous physical activity. Ambient room temperature was maintained at 24 ± 1 °C. On arrival, each participant inserted a rectal temperature probe (YSI 400, Yellow Springs, OH) to a depth of 12–15 cm. A telemetric HR monitor and a head-mounted breathing apparatus were attached, and a catheter was inserted into an antecubital vein. After a 20-min habituation period of quiet sitting, VO\textsubscript{2}, ventilation, and RER were measured for 15 min with the average of the last 10 min used as the baseline (BASE). Metabolic data were collected via open-circuit spirometry (Truemax 2400 Metabolic Measurement System, ParvoMedics, Salt Lake City, UT) using a two-way, low-resistance breathing valve and mouthpiece interfaced with the system. HR and rectal temperature (T\textsubscript{r}) were recorded at the end of BASE, followed by the collection of 10 ml of blood. Participants then ran on the treadmill for 30 min at the appropriate intensity (i.e., ABS or REL). Speed and grade for PRE were set at 4.7 ± 0.1 miles/hr and 2.3% ± 0.9% for CON and 4.8 ± 0.2 miles/hr and 2.9% ± 0.5% for EX. Speed and grade for the REL trial in EX after training were 5.4 ± 0.5 miles/hr and 3.5% ± 0.5% (PRE speed and grade were used in EX for the ABS trial POST). VO\textsubscript{2} was measured continuously throughout the exercise and maintained within ±1 ml · kg\textsuperscript{-1} · min\textsuperscript{-1} of the target value by making minor adjustments to treadmill speed or grade if necessary. Immediately after the exercise, participants were seated in a chair while VO\textsubscript{2}, ventilation, and RER were monitored continuously for the first 30 min of recovery and thereafter for the last 10 min of each 30-min period for the following 90 min. HR and T\textsubscript{r} were recorded every 15 min. Ten milliliters of blood were collected immediately after exercise (END) and at 30 min of recovery for the subsequent determination of epinephrine (EPI), norepinephrine (NOR), free fatty acids (FFA), insulin (INS), glucose (GLU), and lactate (BLA). One milliliter was drawn at 10 min of recovery for subsequent BLA measurement, and 5 ml were collected at 60 min (FFA, INS, and GLU) and 120 min (FFA and GLU) of recovery for subsequent additional measurements.

**EPOC Measurements.** Magnitude of EPOC (kcal) was calculated as the sum of the net energy expenditure for each minute of the first 30 min of recovery. The average VO\textsubscript{2} value of the last 10 min of each 30-min period was used for the remaining 90 min of recovery. Magnitude of EPOC was expressed on an absolute basis (kcal/2 hr) and relative to BM and FFM.

**Blood Collection and Storage**

Samples were prepared for EPI and NOR analyses according to the following: 5 ml of blood were collected in a sodium heparinized tube (green top) containing 100 μl of glutathione and EGTA solution. The solution was prepared by mixing 2.5 g of glutathione, 3.0 g of EGTA, and 33.5 ml of distilled water. The blood sample was mixed with the solution by gentle inversion and placed in an ice bath for 5 min, then centrifuged at 4 °C and 600 g (i.e., 1,750 rpm) for 10 min. The plasma sample was transferred to a storage tube and stored at –80 °C until analyzed. Other blood samples (5 ml) were collected in plain tubes (red top) for subsequent determination of FFA, INS, GLU, and BLA. An aliquot (20 μl) was deproteinated by mixing with 100 μl of 0.1-N perchloric acid (HClO\textsubscript{4}) and centrifuged at 4 °C and 960 g (2,800 rpm) for 15 min, and the supernatant was removed and stored at –80 °C for subsequent measurement of BLA. The remainder of each blood sample, after being allowed a minimum of 30 min to clot in an ice bath, was centrifuged at 4 °C and 960 g for 15 min. Serum samples were transferred to storage tubes and stored immediately at –80 °C.

**Blood Analysis**

Isolation of catecholamines from the prepared plasma sample was accomplished by alumina extraction using a Chromsystems reagent kit (Alko Diagnostics, Holliston, MA). After extraction, concentrations of EPI and NOR were quantified by high-performance liquid chromatography. A complete Waters (Waters Co., Milford, MA) system consisting of a pump (model 510), WISP autoinjector (model 712) with cooling module, column oven, and an electrochemical detector (model 460) was used. Intra-assay coefficient of variation (intra CV) was less than 1%, interassay coefficient of variation (inter CV) was less than 3%, the correlation coefficient from 9 to 1,000 pg on column was 0.9989, and sensitivity was 5 pg with a signal-to-noise ratio of 5:1. INS was determined from...
the prepared serum samples using solid-phase radioimmunoassays (Coat-A-Count, Diagnostic Products Co., Los Angeles, CA) and an autogamma counter (Cobra II, Packard Bioscience Co., Meriden, CT). Intra CV and inter CV for INS were 1.0% and 5.4%, respectively. FFA and GLU were determined using quantitative enzymatic methods (Wako, Richmond, VA, and Sigma Diagnostics, St. Louis, MO, respectively). Intra CVs for FFA and GLU were 1.4% and 2.0%, respectively, and inter CVs were 0.7% and 1.5%, respectively. BLA was determined using an enzymatic method (Lowry & Passoneau, 1972).

Exercise-Training Program

Participants assigned to EX underwent a 12-week endurance-exercise-training program. Each training session consisted of 10 min of warm-up, the main exercise (jogging/running) performed at an individually prescribed duration and intensity, and 10 min of cooldown. Frequency, intensity, and duration initially were 3 days/week at 60% VO$_{2max}$ for 25 min/session (~230–280 kcal/session). Training was gradually increased so that by the 12th week participants were exercising 4 days/week at an intensity eliciting 80% VO$_{2max}$ for 40 min/session (~520–580 kcal/session). Training was conducted under close supervision by one of the investigators, and compliance was 100%. Participants in CON were asked to maintain their normal daily physical activity and dietary behaviors.

Statistical Analysis

The Statistical Analysis System for Windows, version 8, was used for the analysis. All data are reported as $M \pm SE$. The ANOVA using either a split plot design or a split-split plot design was used to test for differences in the mean values between EX and CON (group), between PRE and POST tests (test), and among the measurement time points (time). Post hoc multiple comparisons using Bonferroni’s adjustments were performed when appropriate. Statistical significance was accepted for all tests at $p < .05$.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>PRE</th>
<th>POST</th>
<th>Group × Test interaction ($p$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>EX</td>
<td>26.2 ± 1.4</td>
<td>26.2 ± 1.4</td>
<td></td>
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<tr>
<td></td>
<td>CON</td>
<td>26.2 ± 0.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>EX</td>
<td>174.8 ± 1.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CON</td>
<td>174.8 ± 1.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>EX</td>
<td>73.8 ± 2.1</td>
<td>73.3 ± 2.0</td>
<td>.0725</td>
</tr>
<tr>
<td></td>
<td>CON</td>
<td>71.2 ± 2.7</td>
<td>71.3 ± 2.6</td>
<td></td>
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<tr>
<td>Body fat (%)</td>
<td>EX</td>
<td>16.4 ± 1.7</td>
<td>15.2 ± 1.8*</td>
<td>.0004</td>
</tr>
<tr>
<td></td>
<td>CON</td>
<td>17.7 ± 1.1</td>
<td>18.1 ± 1.0</td>
<td></td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>EX</td>
<td>12.1 ± 1.4</td>
<td>11.2 ± 1.4*</td>
<td>.0006</td>
</tr>
<tr>
<td></td>
<td>CON</td>
<td>12.8 ± 1.2</td>
<td>13.1 ± 1.1</td>
<td></td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>EX</td>
<td>61.7 ± 2.0</td>
<td>62.1 ± 2.0</td>
<td>.0499</td>
</tr>
<tr>
<td></td>
<td>CON</td>
<td>58.4 ± 1.7</td>
<td>58.2 ± 1.7</td>
<td></td>
</tr>
<tr>
<td>VO$_{2max}$ (L/min)</td>
<td>EX</td>
<td>3.41 ± 0.11</td>
<td>3.73 ± 0.11*</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>CON</td>
<td>3.20 ± 0.12</td>
<td>3.16 ± 0.12</td>
<td></td>
</tr>
<tr>
<td>VO$_{2max}$ (ml · kg BM$^{-1}$ · min$^{-1}$)</td>
<td>EX</td>
<td>46.2 ± 1.2</td>
<td>51.0 ± 1.3*</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>CON</td>
<td>45.1 ± 1.4</td>
<td>44.5 ± 1.3</td>
<td></td>
</tr>
<tr>
<td>VO$_{2max}$ (ml · kg FFM$^{-1}$ · min$^{-1}$)</td>
<td>EX</td>
<td>55.3 ± 1.1</td>
<td>60.2 ± 0.9*</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>CON</td>
<td>54.8 ± 1.3</td>
<td>54.3 ± 1.2</td>
<td></td>
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</tbody>
</table>

Note. Values are expressed as $M \pm SE$ of the mean; $n = 9$ in EX and $n = 10$ in CON. BM = body mass; FFM = fat-free mass.

*Significantly different from EX PRE ($p < .001$).
Results

Physical Characteristics

No significant differences in the pretest values were found between EX and CON with regard to age, height, BM, BF, FM, FFM, and VO$_{2\text{max}}$. After the intervention, significant ($p < .001$) decreases were seen in BF and FM in EX. Training also resulted in a significant ($p < .001$) increase in VO$_{2\text{max}}$ in EX when expressed both in absolute and relative terms. No significant changes in these variables were noted in CON (Table 1).

ABS Trial

Total EPOC during the 120-min recovery period was significantly ($p < .01$) lower POST than PRE in EX both when expressed in absolute terms and relative to BM and FFM (Table 2). EPOC during the first 30 min of the ABS trial also was significantly ($p < .001$) lower POST (22.5 ± 1.3 kcal) than PRE (26.9 ± 1.4 kcal; Figure 1). Net VO$_{2}$ values measured 1, 2, 3, 4 (Figure 2[A] inset), and 5 (Figure 2[A] main figure) min after training were significantly lower than PRE, with no additional significant PRE–POST differences in net VO$_{2}$ noted after 10 min of recovery. No significant changes in EPOC were noted in CON.

Other significant training effects were noted in EX during the ABS trial. RER was significantly lower ($p < .001$) after training at 2, 3, and 10 min of recovery (Figure 2[B] inset). HR after training was also significantly lower throughout recovery, returning to the resting value by 120 min postexercise Figure 3[A]). In addition, $T_{hr}$ was significantly lower POST than PRE throughout the recovery phase (Figure 3[B]). No significant PRE–POST changes were observed for RER, HR, or $T_{hr}$ in CON.

There were no significant group differences in any of the biochemical variables before the intervention period.

Table 2  Excess Postexercise Oxygen Consumption (EPOC) Measured During Preintervention (PRE) and Postintervention Testing (POST) in an Exercise-Training Group (EX) and a Control Group (CON) When Exercise Was Performed at the Same Absolute (ABS) and Relative (REL) Intensity

<table>
<thead>
<tr>
<th></th>
<th>EX PRE</th>
<th>EX POST (ABS)</th>
<th>EX POST (REL)</th>
<th>CON PRE</th>
<th>CON POST</th>
</tr>
</thead>
<tbody>
<tr>
<td>EPOC, kcal/2 hr</td>
<td>39.4 ± 3.6</td>
<td>31.7 ± 2.2*</td>
<td>41.0 ± 2.2</td>
<td>36.8 ± 3.5</td>
<td>38.6 ± 3.1</td>
</tr>
<tr>
<td>EPOC, kcal/kg BM</td>
<td>0.54 ± 0.04</td>
<td>0.43 ± 0.03*</td>
<td>0.56 ± 0.03</td>
<td>0.51 ± 0.04</td>
<td>0.54 ± 0.03</td>
</tr>
<tr>
<td>EPOC, kcal/kg FFM</td>
<td>0.64 ± 0.06</td>
<td>0.51 ± 0.03*</td>
<td>0.66 ± 0.04</td>
<td>0.63 ± 0.05</td>
<td>0.66 ± 0.04</td>
</tr>
</tbody>
</table>

Note. Values are expressed as $M ± SE$ of the mean. BM = body mass; FFM = fat-free mass.
*Significantly different from EX PRE ($p < .01$).

Figure 1 — Excess postexercise oxygen consumption (EPOC) measured for 120 min of recovery from exercise during preintervention testing (PRE) and postintervention testing (POST) in an exercise-training group (EX) and a control group (CON) when exercise was performed at the same absolute (ABS) and relative (REL) intensity. *EX POST (ABS) significantly different from EX PRE.
BASE values for EPI did not change significantly in either group after the intervention period. At END, EPI in EX was significantly lower \((p = .04)\) POST (73.8 ± 10.1 pg/ml) than PRE (102.5 ± 22.4 pg/ml), whereas no significant change was seen in CON. Mean values for EPI returned to BASE by 30 min postexercise in all trials (Figure 4[A]).

No significant training-induced change in NOR was seen in EX during the ABS trial (Figure 4[B]). BLA during the
ABS trial in EX was significantly lower after than before training at END ($p < .001$) and 10 min of recovery ($p = .04$). In addition, BLA before training was significantly elevated above BASE at 30 min of recovery in both EX and CON, but this difference was not observed in EX after training (Figure 4[C]).

Mean values for FFA in EX after the intervention at END (0.81 ± 0.14 mmol/L) and 30 min postexercise (0.94...
Figure 4(A) — Epinephrine (EPI) measured at baseline (BASE), the end of exercise (END), and at specific times during recovery during preintervention testing (PRE) and postintervention testing (POST) in an exercise-training group (EX) and a control group (CON) when exercise was performed at the same absolute (ABS) and relative (REL) intensity. *EX POST (ABS and REL) significantly different from EX PRE; #Significantly different BASE in all trials.

Figure 4(B) — Norepinephrine (NOR) measured at baseline (BASE), the end of exercise (END), and at specific times during recovery during preintervention testing (PRE) and postintervention testing (POST) in an exercise-training group (EX) and a control group (CON) when exercise was performed at the same absolute (ABS) and relative (REL) intensity. *Significantly different between EX PRE and EX POST (REL) and between CON PRE and CON POST; #Significantly different from BASE in all trials.

± 0.11 mmol/L) were significantly higher than at END (0.53 ± 0.08 mmol/L; $p < .01$) and 30 min postexercise (0.59 ± 0.07 mmol/L; $p < .01$) during PRE (Figure 5[A]). Mean values for INS were significantly ($p < .001$) lower POST than PRE in EX at BASE, END, and 30 min and 60 min of recovery during the ABS trial (Figure 5[B]). GLU in EX at BASE ($p = .02$), END ($p < .001$), and 30 min ($p < .001$), 60 min ($p < .01$), and 120 min ($p = .01$) postexercise was significantly lower POST than PRE (Figure 5[C]). No significant differences in FFA, INS, GLU, or BLA were found between PRE and POST in CON at any time point.
Figure 4(C) — Blood lactate (BLA) measured at baseline (BASE), the end of exercise (END), and at specific times during recovery during preintervention testing (PRE) and postintervention testing (POST) in an exercise-training group (EX) and a control group (CON) when exercise was performed at the same absolute (ABS) and relative (REL) intensity. *EX POST (ABS) significantly different from EX PRE; #Significantly different from BASE in all trials; $Significantly different from BASE in EX PRE, EX POST (REL), CON PRE, and CON POST.

Figure 5(A) — Free fatty acid (FFA) measured at baseline (BASE), the end of exercise (END), and at specific times during recovery during preintervention testing (PRE) and postintervention testing (POST) in an exercise-training group (EX) and a control group (CON) when exercise was performed at the same absolute (ABS) and relative (REL) intensity. *EX POST (ABS and REL) significantly different from EX PRE.

REL Trial.

No significant PRE–POST differences in EPOC were noted for EX during the REL trial. However, RER was significantly lower ($p < .001$) POST than PRE at 10, 15, 20, 25, and 30 min (Figure 2[B]) of recovery. Similar to the ABS trial, HR in EX during the REL trial was significantly lower POST than PRE throughout the postexercise period and returned to the resting value by 120 min of
recovery (Figure 3[A]). No significant changes were noted for T during the REL trial (Figure 3[B]).

At END, EPI in EX was significantly lower ($p = .04$) POST (76.3 ± 11.2 pg/ml) than PRE (102.5 ± 22.4 pg/ml), whereas NOR at END in EX was significantly higher ($p = .01$) POST (1,416.8 ± 182.5 pg/ml) than PRE (1,114.4 ± 190.6 pg/ml) in the REL trial (Figures 4[A] and 4[B]). No significant PRE–POST change in EPI was seen in CON,
but NOR in CON at END was significantly higher (p = .02) POST (1,300.8 ± 183.6 pg/ml) than PRE (1,047.4 ± 87.7 pg/ml). There were no significant training-induced changes in BLA during the REL trial. In EX and CON, BLA remained significantly higher than BASE through 30 min of recovery both PRE and POST (Figure 4[C]).

Mean values for FFA (Figure 5[A]) in EX at END and 30 min postexercise were significantly higher (p < .01) POST (0.78 ± 0.09 and 0.92 ± 0.11 mmol/L, respectively) than PRE (0.53 ± 0.08 and 0.59 ± 0.07 mmol/L, respectively). Mean values for INS measured at BASE, END, and 30 min and 60 min of recovery in EX during the REL trial were significantly (p < .001) lower after than before training (Figure 5[B]). GLU in EX was also significantly lower POST than PRE at END (p < .01), 30 min (p = .02), 60 min (p = .02), and 120 min (p = .03; Figure 5[C]). No significant differences in FFA, INS, GLU, or BLA were found between PRE and POST in CON at any time point.

Discussion

To our knowledge, this is the first prospective study investigating aerobic-training effects on EPOC after exercise at both the same absolute and same relative intensity. In addition, we measured various blood hormone and substrate concentrations to provide insight into mechanisms that may affect training-induced changes in EPOC.

Well-matched assignment of participants into each group with regard to body composition and aerobic capacity is important because metabolic rates can be affected by these factors. In this regard, no significant preintervention differences were found between EX and CON for age, height, BM, BF, FFM, FM, and VO₂max. The training program elicited a substantial cardiorespiratory training effect as evidenced by a significant increase in VO₂max. Absolute VO₂max (L/min), VO₂max per BM, and VO₂max per FFM increased by 9.4%, 10.3%, and 8.9%, respectively. These results are comparable to those of previous studies (Dolezal & Potteiger, 1998) employing a training program similar in duration and intensity to ours. Significant training-induced changes in body composition were also noted. BF and FM significantly decreased, and there was a trend for BM to decrease and FFM to increase.

Training induced a significant decrease in total EPOC when expressed on an absolute basis and relative to BM and FFM during the ABS trial, whereas no significant change was found after the REL trial. Results of the ABS trial indicate that the efficiency of metabolic regulation was improved, resulting in a more economical recovery posttraining than in the pretraining condition. These findings support our previous suggestion (Sedlock, 1991b) that EPOC would vary as a function of the relative metabolic rate of the active musculature. The current results are consistent with previous findings from our laboratory (Short & Sedlock, 1997), as well as findings of Girandola and Katch (1973) and Hagberg, Hickson, et al. (1980), who found that VO₂ during the first 10–15 min of recovery from exercise at the same absolute intensity was less in trained than in untrained individuals. The results also are similar to those of Frey et al. (1993), who reported a smaller EPOC in trained than untrained individuals after a 300-kcal exercise, despite the trained group’s showing a larger fast component of EPOC. In addition, the current results are consistent with our previous findings (Sedlock, 1994) showing no significant difference in EPOC between trained and untrained individuals after exercise at the same relative intensity. Conversely, our results conflict with those of Freedman-Akabas et al. (1985) and Brehm & Gutin (1986), who reported no effect of training status on EPOC after exercise at the same absolute intensity, and Chad and Quigley (1991), who found a greater EPOC response in trained participants.

As mentioned previously, total EPOC decreased significantly after exercise at the same absolute intensity, whereas it remained unchanged after exercise at the same relative intensity after the training program. In several previous studies examining the effects of training status on EPOC, differences in EPOC between trained and untrained individuals were explained indirectly based on RER or HR responses. In only one study (Frey et al., 1993) were NOR, BLA, and T₉ measured to provide a mechanistic explanation for the different EPOC responses resulting from training status; however, the authors were unable to explain the results completely using these variables. Similar to these previous studies, we measured RER, HR, NOR, BLA, and T₉; however, we also measured other hormones and substrates to examine other possible mechanisms responsible for metabolic alterations in EPOC consequent to exercise training.

Catecholamines are considered one of the major factors related to postexercise VO₂. EPI and NOR have been found to be significantly correlated with magnitude and duration of EPOC and to possibly elevate the recovery VO₂ (Imamura et al., 2004). Howlett, Febbraio, and Hargreaves (1999) suggested that EPI and NOR increase EPOC by increasing hepatic glucose production and triacylglyceride/fatty-acid cycling, respectively, and that EPOC would be decreased by a reduction in sympathetic nervous system activity. In addition, Frey et al. (1993) found that NOC correlated highly (r = .78) with EPOC. In the current study, EPI at the end of exercise was significantly lower after training during the ABS trial, whereas no similar training-induced changes in NOR were found. As per the previously mentioned suggestion of Howlett et al., the lower EPI response after training may be associated with a decrease in EPI-induced hepatic glucose production, perhaps contributing to the smaller EPOC observed posttraining. During the REL trial after training, EPI was also significantly lower at end-exercise, but NOR was significantly higher. The lack of a significant training effect on EPOC after exercise at the same relative intensity may be the result of these differential EPI and NOR responses. That is, a possible training-induced decrease in the potentiating effect of EPI on EPOC might have been compensated for by a training-induced increase in the augmenting effect of NOR on EPOC.
In the current study, mean values for FFA in EX at the end of exercise and at 30 min of recovery during the ABS and REL trials were significantly higher after training, suggesting that fat oxidation had increased. These results are consistent with those of Chad and Quigley (1991), who reported that trained participants metabolized more than 3 times as much fat as untrained participants during exercise at 70% VO$_{2\text{max}}$ and 18% more fat at the end of a 3-hr recovery period after the exercise. INS concentrations during recovery from the ABS trial were significantly lower after training in the current study, which suggests a training-induced increase in INS sensitivity because GLU was also significantly lower throughout recovery in EX after training. These findings are consistent with those of Houmard, Shaw, Hickey, and Tanner (1999), who found that exercise training improved INS sensitivity during and for 60 min after exercise at 75% VO$_{2\text{max}}$ by enhancing INS signal transduction in skeletal muscle. With the possibility of the aforementioned decrease in EPI-mediated hepatic glucose production, the increase in INS-mediated glucose uptake would represent a carbohydrate-sparing effect of exercise training and contribute to the observed reduction in EPOC. Our data for FFA and GLU are also consistent with a training-induced shift toward greater fat (relative to carbohydrate) oxidation during and after a bout of submaximal exercise.

BLA is also known to be strongly related to postexercise VO$_{2\text{r}}$, particularly the fast component of EPOC. Bahr, Gronnerod, and Sejersted (1992) found that EPOC was related to changes in BLA during the first hour of recovery from exercise. Frey et al. (1993) reported a significant correlation ($r = .90$) between BLA and EPOC. In the current study, BLA at END and at 10 min of recovery was significantly lower after training during the ABS trial, suggesting that the training-induced decrease in BLA may in part account for the posttraining decrease in EPOC. These results support previously reported findings of a smaller EPOC response with lower BLA in trained than in untrained individuals (Frey et al., 1993).

Elevated body temperature appears to be partially responsible for the increase in recovery energy expenditure, particularly the slow component of EPOC. The $Q_{10}$ effect of temperature on metabolism may account for 60–70% of the variance of the slow component after exercise at 50–80% VO$_{2\text{max}}$ (Hagberg, Mullin, & Nagle, 1980). In the current study, recovery Tre was significantly lower after training during the ABS trial, whereas no significant change in Tre with training was found during the REL trial. These results suggest that the decrease in Tre after training may partially account for the training-induced decrease in the EPOC response during the ABS trial. These data support previous research by Brehm and Gutin (1986), who found a faster decrease in Tre during a 1-hr recovery in trained than in untrained participants, and Frey et al. (1993), who reported a significantly lower Tre in trained participants during recovery from exercise at the same absolute intensity but no effect of training status on recovery Tre after exercise at the same relative intensity.

In the current study, RER after training was significantly lower during the first 10 min of recovery after exercise at the same absolute intensity and significantly lower between 10 and 30 min of recovery after exercise at the same relative intensity. These results are similar to those of a previous study from our laboratory (Short & Sedlock, 1997) and agree with those of Frey et al. (1993), who reported that RER for the first 10 min of recovery after exercise at both 65% and 80% of VO$_{2\text{max}}$ was significantly lower in trained than in untrained participants. Notwithstanding the fact that an altered RER response may result from changes in ventilation or acidity of the blood (Chad & Wenger, 1988), this adaptation in RER may be attributed to a training-induced decrease in the transient elevation in carbon dioxide production as reported by Hagberg, Hickson, et al. (1980). Therefore, exercise training appeared to improve control of metabolic stress immediately after exercise, possibly contributing to the observed decrease in EPOC. The lower RER found between 10 min and 30 min of recovery during the REL trial may be related to the training-induced decrease in production and utilization of plasma-borne glucose during exercise, which was considered a carbohydrate-sparing effect of training (Coggan, 1997).

**Conclusion**

Aerobic-exercise training resulted in a significant decrease in the magnitude of EPOC after exercise at the same absolute metabolic intensity. Mechanisms appear to include training-induced decreases in BLA and Tre, as well as the possibility of a reduction in EPI-mediated hepatic glucose production and INS-mediated glucose uptake. No similar training-induced attenuation of EPOC was noted after exercise at the same relative intensity. Because the absolute-intensity exercise trial was performed at a lower relative metabolic intensity posttraining (i.e., 63.5% vs. 70% VO$_{2\text{max}}$), this indicates that EPOC varies as a function of the relative rather than absolute metabolic stress.

**References**


