Excess postexercise oxygen consumption and recovery rate in trained and untrained subjects

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Short, Kevin R., and Darlene A. Sedlock. Excess postexercise oxygen consumption and recovery rate in trained and untrained subjects. J. Appl. Physiol. 83(1): 153–159, 1997.—The purpose of this study was to determine whether aerobic fitness level would influence measurements of excess postexercise oxygen consumption (EPOC) and initial rate of recovery. Twelve trained (Tr; peak oxygen consumption \( V_\text{O2peak} \)) and ten untrained (UT; \( V_\text{O2peak} \)) subjects completed two 30-min cycle ergometer tests on separate days in the morning, after a 12-h fast and an absence from vigorous activity of 24 h. Baseline metabolic rate was established during the last 10 min of a 30-min seated preexercise rest period. Exercise workloads were manipulated so that they elicited the same relative, 70% \( V_\text{O2peak} \) (W70%), or the same absolute, 1.5 l/min oxygen uptake (\( V_\text{O2} \)) (W1.5), intensity for all subjects, respectively. Recovery \( V_\text{O2} \), heart rate (HR), and respiratory exchange ratio (RER) were monitored in a seated position until baseline \( V_\text{O2} \) was reestablished. Under both exercise conditions, Tr had shorter EPOC duration (W70% = 40 ± 15 min, W1.5 = 21 ± 9 min) than UT (W70% = 50 ± 14 min; W1.5 = 39 ± 14 min), but EPOC magnitude (Tr: W70% = 3.2 ± 1.0 liters \( V_\text{O2} \); W1.5 = 1.5 ± 0.6 liters \( V_\text{O2} \); UT: W70% = 3.5 ± 0.9 liters \( V_\text{O2} \); W1.5 = 2.4 ± 0.6 liters \( V_\text{O2} \)) was not different between groups. The similarity of Tr and UT EPOC accumulation in the W70% trial is attributed to the parallel decline in absolute \( V_\text{O2} \) during most of the initial recovery period. Tr subjects had faster relative decline during the fast-recovery phase, however, when a correction for their higher \( V_\text{O2} \) was taken. Postexercise \( V_\text{O2} \) was lower for Tr group for nearly all of the W1.5 trial and particularly during the fast phase. Recovery HR kinetics were remarkably similar for both groups in W70%, but recovery was faster for Tr during W1.5. RER values were at or below baseline throughout much of the recovery period in both groups, with UT experiencing larger changes than Tr in both trials. These findings indicate that Tr individuals have faster regulation of postexercise metabolism when exercising at either the same relative or same absolute work rate.

The existence of a postexercise elevation in resting metabolism is a familiar phenomenon and has been labeled the period of excess postexercise oxygen consumption (EPOC) (12). In attempts to distinguish the EPOC response quantitatively, a number of exercise manipulations have been employed. Variation in exercise duration and intensity (4, 6, 8, 27) has received the most attention, but the effects of exercise mode (25), body core temperature (18), and nutritional status have also been examined (5, 24). Aerobic training status is another variable that can influence EPOC, since the physiological adaptations of trained individuals alter many aspects of exercise metabolism (22, 23).

To date, a consensus has not been reached regarding the effect of physical training on EPOC duration or magnitude (7, 8, 10, 11, 26). In some cases, methodological limitations have prevented a clear conclusion from being made (7, 10). In one such report, subjects were separated into groups based on their ventilatory threshold (10), but these distinctions were unclear, and the exercise intensity during testing was somewhat undefined. No sustained elevations in oxygen consumption \( (V_\text{O2}) \) were detected at 40 min postexercise and beyond. However, metabolic measurements were not recorded between minute 4 and 40, so the exact duration of recovery and any distinction between the “high-” and “low-fit” groups were not possible. Another investigation (7) found no difference in EPOC duration between runners and nonrunners, but as the authors point out, the exercise bout (3.2-km walk at 6.4 km/h) was not of sufficient intensity to differentially challenge the regulation of temperature, substrate utilization, and hormone release between the groups. Similarly, Sedlock (26) has also reported that EPOC duration and magnitude in endurance-trained and sedentary men were not different, but the combination of exercise intensity (50% maximal \( V_\text{O2} \) \( V_\text{O2_{max}} \)) and duration (28–35 min) may be too low to yield distinct responses from the two groups.

Unlike the findings already mentioned, Chad and Quigley (8) reported that well-trained women maintained higher \( V_\text{O2} \) than an untrained group during 3 h of recovery. Exercise consisted of 30 min of cycling at 50 or 70% of \( V_\text{O2_{max}} \). Trained subjects had a significantly higher rate of postexercise fat utilization that may contribute to EPOC, but a suitable explanation for why this effect was more apparent in trained than untrained individuals was not provided. This study is the first to show that aerobic training may be associated with larger recovery energy expenditure and potentially longer EPOC duration (exact EPOC duration was not determined).

Conversely, Frey et al. (11) have demonstrated that trained women who cycled at either 80% (24 min) or 65% of \( V_\text{O2_{max}} \) (45 min) had shorter EPOC duration than their untrained counterparts. No readily apparent methodological differences are available to explain the disparity in the work of Frey et al. and Chad and Quigley (8). There is support, however, from longitudinal training studies that metabolic recovery is faster in trained subjects (13, 15). Previously sedentary individuals that were examined before and after a 9-wk training program had faster postexercise recovery rates for \( V_\text{O2} \).
heart rate (HR), and pulmonary ventilation in the trained state (15).

The results of Frey et al. (11) are consistent with the hypothesis that aerobic training is associated with faster adjustment of postexercise energetics but they raise additional questions. During exercise, total energy expenditure was held constant, which meant that untrained subjects had to cycle longer (+10 min at 80% \( \text{VO}_2\text{max} \), +15 min at 60% \( \text{VO}_2\text{max} \)) than the trained subjects to reach the 300-kcal limit (11). This raises the possibility that differences in exercise duration may have contributed to the variation in recovery \( \text{VO}_2 \) that the authors ascribed solely to the subjects’ fitness levels. It has been shown that a change in the duration of a vigorous exercise bout (70% \( \text{VO}_2\text{max} \)) significantly alters the EPOC response (4, 9, 14) so there is a need to control both exercise intensity and duration when comparing groups with different abilities. Additionally, Frey et al. (11) report that total EPOC magnitude was not different between groups but that the EPOC accumulated during the fast phase so, in effect, the \( \text{VO}_2 \) at the conclusion of exercise can have a sizable impact on EPOC. This distinction points to the value of controlling the absolute exercise intensity when comparing exercise recovery of trained and untrained subjects (15).

Given the limited number of investigations and the equivocal findings of these reports (7, 8, 10, 11, 26), the purpose of this investigation was to further examine the relationship between aerobic training status and EPOC. Specifically, comparisons were made between the EPOC responses of trained and untrained individuals after exercise bouts of 1) equal relative intensity and duration and 2) equal absolute intensity and duration.

**METHODS**

Subjects. Healthy young volunteers were recruited for this study, initially on the basis of their volume of aerobic activity. In the preceding 4–6 mo, untrained (UT) subjects engaged in <2 h/wk of aerobic activity, whereas trained (Tr) subjects exercised ≥5 h/wk. Typical activities were competitive running, cycling, and swimming for Tr and walking or low-impact aerobic dance for UT. All testing procedures and risks were fully explained, and subjects were asked to provide written consent for participation, in accordance with the University policy. Aerobic capacity was then determined, as described below, to verify subject classification. Inclusion in the Tr group required a peak \( \text{VO}_2 \) (\( \text{VO}_2\text{peak} \)) of >45 ml·kg\(^{-1}\)·min\(^{-1}\), whereas \( \text{VO}_2\text{peak} \) in UT was <40 ml·kg\(^{-1}\)·min\(^{-1}\). Training and \( \text{VO}_2\text{peak} \) requirements were established with the intent that two distinct groups would be created and interference from genetic determinants of \( \text{VO}_2\text{peak} \) could be minimized (21). Subjects were also required to achieve a minimum \( \text{VO}_2\text{peak} \) ≥2.0 l/min to ensure that the fixed intensity workload was not too strenuous. Of the initial subject pool, all but four met the qualifications and completed the remaining testing. Final compositions of the groups were five women and seven men in Tr and six women and four men in UT (Table 1).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Trained Group</th>
<th>Untrained Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>23 ± 3</td>
<td>23 ± 3</td>
</tr>
<tr>
<td>Height, cm</td>
<td>168 ± 6</td>
<td>172 ± 10</td>
</tr>
<tr>
<td>Mass, kg</td>
<td>59.9 ± 5.0</td>
<td>70.7 ± 7.1</td>
</tr>
<tr>
<td>( \text{VO}_2\text{peak} ) ml·kg(^{-1})·min(^{-1})</td>
<td>48.6 ± 1.6</td>
<td>56.7 ± 6.4</td>
</tr>
<tr>
<td>HR peak, beats/min</td>
<td>185 ± 4</td>
<td>186 ± 5</td>
</tr>
<tr>
<td>RER peak</td>
<td>1.15 ± 0.05</td>
<td>1.12 ± 0.07</td>
</tr>
<tr>
<td>Aerobic activity, h/wk*</td>
<td>10.5 ± 2.3</td>
<td>9.8 ± 5.1</td>
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</table>

Values are means ± SD. \( \text{VO}_2\text{peak} \), peak oxygen consumption; \( \text{HR} \), heart rate; RER, respiratory exchange ratio. *Significant difference between groups, \( P < 0.01 \); †significant difference between males and females, \( P < 0.05 \).

Procedures. Participants began the study with an assessment of \( \text{VO}_2\text{peak} \) by using a cycle ergometer (Monark Ergomedic B18, GIH, Stockholm, Sweden). After a 2-min warm-up at 50 W, initial work rate was set at 70 W and increased 35 W every 2 min until the subject could no longer maintain the pedaling frequency of 70 revolutions/min. A calibrated digital display was used to monitor pedaling frequency. \( \text{VO}_2\text{peak} \) was determined to be the highest \( \text{VO}_2 \) measured during the test. A minimum of 1 wk separated the incremental exercise test from the EPOC trials.

Two EPOC trials were performed in counterbalanced order and separated by at least 72 h. Work rates during the trials were adjusted to elicit either 70% of \( \text{VO}_2\text{peak} \) (W70%) or a \( \text{VO}_2 \) of 1.5 l/min (W1.5). These tests were designed so that all subjects exercised at the same relative and absolute work rates, respectively. In the 24 h preceding each trial, diet and activity were replicated as closely as possible, and strenuous activity was avoided. Subjects were transported to the laboratory at ~6:30–7:30 AM, at least 12 h postprandial. On arrival, subjects were quietly seated and fitted with a telemetric HR monitor (Polar Vantage XL, Stamford, CT) and a headmount breathing apparatus. After at least 15 min of habituation, the mouthpiece (Hans-Rudolph, Kansas City, MO) was inserted for an additional 15 min of baseline data collection. \( \text{VO}_2 \), respiratory exchange ratio (RER), and \( \text{HR} \) values were continuously measured during this time, but only values obtained during the last 10 min were averaged and used as baseline. Immediately after the rest phase, subjects began the 30-min cycling exercise. When the exercise period was completed, subjects were seated in a chair and remained there for 60 min or until \( \text{VO}_2 \) returned to baseline, whichever was longer.Expired gases and \( \text{HR} \) were continuously monitored for the duration of the recovery period.

During all phases of the testing, gas exchange was measured via open-circuit spirometry. Minute averages of expired air were measured for volume and fractional \( \text{O}_2 \) and \( \text{CO}_2 \) by using an automated system (Horizon MMC, SensorMedics, Yorba Linda, CA) and updated every 30 s. Before all test sessions, the gas analyzers were calibrated with room air and gases of known concentration. The accuracy of the gases used for calibration was guaranteed by the supplier to be within 0.1% of the stated values. EPOC duration was calculated as the time required for a 10-min average of recovery \( \text{VO}_2 \) to equal the baseline value. EPOC magnitude was calculated as
the integrated area between the recovery $\dot{V}O_2$ curve and the baseline. The net energy expenditure of the EPOC period was calculated in a similar manner. Energy expenditure, ($kJ$) was computed by using the standard conversion of $\dot{V}O_2$ from nonprotein RER (17) for each minute. The rate of change in postexercise $\dot{V}O_2$ was calculated as the percent difference between the end-exercise value (assigned a value of 0%) and resting baseline value ($100\%$ recovery) for each measurement time during recovery.

To ensure that the testing environment was appropriately controlled, the laboratory was kept as quiet as possible during all resting measurements. The testing room was maintained at 22.4 ± 0.9°C and 56.6 ± 4.0% relative humidity. Subjects were asked to wear similar clothing (shorts, T-shirt) for both trials.

Statistical analyses. The Statistical Analysis System (SAS; Cary, NC) software package was used to analyze all data. All variables measured during the EPOC trials were subjected to an analysis of variance for repeated measures. Where appropriate, the Tukey post hoc procedure was used to elicit pairwise differences among means. Statistical significance was accepted for all tests at $P < 0.05$.

### RESULTS

Subject characteristics. As shown in Table 1, men had higher body mass (both groups) and $\dot{V}O_2$ peak (Tr only) than women. There were no significant differences between men and women within each group during the EPOC trials so the results have been pooled and presented as means of the Tr and Ut groups.

Baseline. Table 2 shows the baseline measurements obtained at rest before exercise. $\dot{V}O_2$ and RER values were similar for both groups and both tests. $\dot{V}O_2$ measurements were reproducible, with individual measurements differing by <0.028 l/min on the two test mornings (mean ± SD difference = 0.019 ± 0.101/min). The coefficient of variation of these measurements was 5.15%. The only difference between groups was a higher baseline HR for Ut, but even these values were consistent between trials, indicating that subjects arrived at the laboratory in a similar physiological state for each trial.

Exercise. For each variable measured during exercise, a significant group-by-treatment interaction was found, as expected given the types of exercise bouts employed. During the initial minutes of exercise, the workload was adjusted, if necessary, to achieve the appropriate intensity. The values displayed in Table 3 represent the average steady-state values elicited during the majority of the exercise period.

Recovery. Table 4 and Figs. 1–4 display the recovery measurements. The return of $\dot{V}O_2$ to baseline occurred within 60 min in all but two trials (1 Tr and 1 Ut subject) (Fig. 1). The mean EPOC duration was significantly shorter for Tr than Ut in both exercise conditions (Table 4). However, EPOC magnitude of the Tr group was smaller only in W1.5 trial. In the W70% trial, the absolute $\dot{V}O_2$ at the start of the recovery period was significantly higher for Tr subjects because of their higher exercise work rates. To compare the rates of $\dot{V}O_2$ recovery of the two groups in the W70% trial, the $\dot{V}O_2$ curve was normalized to percent change. Figure 2 displays these values during the fast-recovery phase when significant differences between groups were measured. There were no further differences beyond 8 min.

At baseline and throughout the recovery period, the Tr group had a consistently lower HR. To simplify the depiction of recovery HR, values are reported as units of change ($\Delta$) in relation to the preexercise value (Fig. 3). After making this adjustment, the group difference in the W70% HR values was no longer evident, indicat-

### Table 2. Baseline measurements established during preexercise rest

<table>
<thead>
<tr>
<th>Variable</th>
<th>Trained Group</th>
<th>Untrained Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\dot{V}O_2$, l/min</td>
<td>2.459 ± 0.447</td>
<td>2.530 ± 0.437</td>
</tr>
<tr>
<td>$\dot{V}O_2$, ml·kg⁻¹·min⁻¹</td>
<td>63.3 ± 1.7</td>
<td>65.3 ± 1.6</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>159 ± 7</td>
<td>129 ± 7</td>
</tr>
<tr>
<td>RER</td>
<td>0.93 ± 0.05</td>
<td>0.86 ± 0.04</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = 12 (trained) or 10 (untrained). $\dot{V}O_2$, oxygen consumption. Trials consisted of 30 min of cycling at either 70% $\dot{V}O_2$ peak (W70%) or 1.5 l/min $\dot{V}O_2$ (W1.5). *Significant difference between groups, $P < 0.05$.

### Table 3. Physiological measurements of trained and untrained subjects during steady-state exercise

<table>
<thead>
<tr>
<th>Variable</th>
<th>Trained Group</th>
<th>Untrained Group</th>
</tr>
</thead>
<tbody>
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<td>$\dot{V}O_2$, l/min</td>
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<tr>
<td>$\dot{V}O_2$, ml·kg⁻¹·min⁻¹</td>
<td>63.3 ± 1.7</td>
<td>65.3 ± 1.6</td>
</tr>
<tr>
<td>Energy expenditure, $kJ$</td>
<td>1,531 ± 280</td>
<td>943 ± 22</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>159 ± 7</td>
<td>129 ± 7</td>
</tr>
<tr>
<td>RER</td>
<td>0.93 ± 0.05</td>
<td>0.86 ± 0.04</td>
</tr>
</tbody>
</table>

Values are means ± SD. †Different from baseline, $P < 0.05$. a,b,cRow values with same letter do not differ, $P > 0.05$.
ing that HR recovery kinetics were nearly identical in Tr and UT. In the W1.5 trial, however, HR of the Tr group was lower than UT throughout the entire postexercise period, even after correction for baseline differences. In all trials, the HR remained elevated above baseline at the end of the postexercise observation period. End-EPOC HR was similar in both trials for UT, but for Tr the HR elevation at end-EPOC was significantly greater after W70% compared with W1.5.

Postexercise RER is also reported as units of change (Δ) in relation to the preexercise value (Fig. 4). At the start of recovery, there was a transient upward deflection in RER values, followed by a gradual and persistent decline until baseline was reached or surpassed. The difference between recovery RER and baseline in the W70% trial was statistically significant at end-EPOC. In contrast, during W1.5, the mean end-EPOC RER was below baseline only for UT, as Tr had RER values nearly equal to baseline during most of the postexercise period.

**DISCUSSION**

Recovery \( \dot{V}O_2 \). The primary purpose of this study was to examine the effect of aerobic fitness level on postexercise \( \dot{V}O_2 \). Two exercise bouts were selected so that Tr and UT performed at the same relative and absolute intensities for an equal period of time. The W70% condition was chosen to provide a vigorous stimulus for
distinguishing any potential variation during the recovery period, whereas W1.5 trial was used so that all subjects finished the exercise period at the same absolute VO₂. In both trials, the intensity and duration of exercise were constrained by the ability of UT subjects to complete the exercise bout. EPOC duration and magnitude may have been greater with longer or more vigorous exercise (4, 6, 14), but it is clear from these results that differences between Tr and UT could be detected with the exercise bouts employed.

Regular aerobic training is associated with several cardiovascular and muscular adaptations, i.e., increased muscle blood flow and changes in substrate utilization (22, 23), that alter the response to exercise. Considering these and other changes, we hypothesized that training could improve the efficiency of metabolic regulation, resulting in faster recovery times for trained subjects. The present data support that postulation, since Tr had faster initial VO₂ recovery rates and shorter EPOC duration times for both exercise treatments. Even when relative exercise intensity was controlled (W70%), Tr had shorter EPOC duration than UT. One possible explanation for this finding is that W70% imposed a greater stress on UT than Tr subjects because of differences in lactate threshold (20). If this explanation were correct, adjustment of the work rate relative to each individual's lactate threshold would likely increase the homogeneity of the EPOC response. This possibility has yet to be tested.

Our findings are in agreement with those of Frey et al. (11), who reported that trained female cyclists had shorter EPOC duration than a group of untrained women. Three other studies were not able to detect a difference in EPOC duration between groups with different fitness levels (7, 10, 26). In two of those cases (7, 26), however, insufficient exercise intensity and/or duration (~30 min at ≤50% VO₂max) was used and the third did not monitor the entire recovery period (10). In contrast to other published reports and the present investigation, Chad and Quigley (8) found that aerobically trained women sustained a higher postexercise VO₂ elevation than sedentary women during 3 h of observation. Although it cannot be stated with certainty, since the EPOC period was not followed until its completion in that study, the recovery trends of the two groups suggest that the trained women had longer recovery times, an unexpected finding that is yet to be replicated.

Both exercise intensity (6, 14, 27) and duration (4, 9, 14) are important determinants of EPOC. The effect of exercise intensity is demonstrated in this investigation by the reduction in recovery times in W1.5, which required less effort for most subjects vs. W70%. The duration of EPOC was typically <60 min, with only two individuals requiring up to 80 min after W70%. These values are considerably lower than some previous reports that used the same exercise bout as W70%, i.e., EPOC ≥2 h (8, 9). In comparison, after only 20 min of cycling at 70% (14) or 75% VO₂max (27), EPOC duration was <1 h. Combined results from these studies indicate that some type of interaction between exercise intensity and duration occurs to prolong metabolic recovery in selected cases (14). It should be pointed out that methods of measuring baseline and EPOC duration may not be consistent among investigations, and comparisons should be made with this in mind. In some cases, the baseline and recovery data are collected with the subject in the seated position (7–9, 11, 24–27), whereas in others the subjects lie in bed (2–6, 10, 14, 16). Data from our laboratory (28) indicate that longer EPOC occurs in the supine position vs. upright sitting as a result of the slight reduction in resting metabolic rate (29). The seated rest position was used in this investigation because we have found it to be comfortable and convenient for both subject and researchers. The key papers for comparison have also used a chair during the rest phases (7, 8, 11, 26). Some authors have used statistical comparisons between the recovery and baseline VO₂ curves (means of all subjects) at discrete time points as the criterion for establishing the end of EPOC (1–6, 8, 9, 11, 14). However, in our laboratory (24–27) and in others (7, 18), expired gases are collected continuously during the postexercise period, and recovery VO₂ is required to equate with baseline for all individual trials at end-EPOC. Use of the latter method may extend EPOC duration because of its higher stringency, but practical differences should be negli-
The importance of the absolute V˙O2 during the fast phase.

between the two groups, but it was used to show the variance in relative exercise intensity, particularly if the pace weight-bearing activity (walking at 3.2 km/h) would have produced differences in exercise V˙O2 among subjects, whereas in this study V˙O2 was closely controlled.

Comparison of EPOC magnitude values measured in this study to previously reported values is not easy, given the methodological differences that exist. The trend in most studies, including the present investigation, is a positive relationship between exercise intensity and EPOC magnitude when exercise duration is held constant (6, 7, 11, 14). However, Chad and Quigley (8) demonstrated that a low-intensity bout (50% V˙O2max) yielded larger EPOC magnitude over a 3-h period than a high-intensity bout (70% V˙O2max). Variation in exercise duration has also been correlated with differences in EPOC magnitude (4, 14).

Recovery HR. For all subjects, HR remained elevated during the entire postexercise period. Although HR measures are not always reported in the EPOC literature, extended HR elevations have been recorded (11, 24, 25) and in some cases may last up to 12 h (16). During the exercise portion of the W70%, HR values of the two groups were similar, yet it was expected that Tr would have lower end-EPOC HR, since baseline HR for that group was also lower. Throughout recovery, Tr did have lower mean HR at all time points, but end-EPOC HR was not different from UT as a result of the difference in EPOC duration. As shown in Fig. 3, the rate of HR recovery was nearly identical for the two groups in W70%. By making the comparison to W1.5, where the groups differed both in rate of change and actual HR, the influence of exercise intensity on recovery HR is made quite clear. The results of Hagberg et al. (15), in contrast, indicated that HR recovery of trained individuals was faster when either the same pretraining absolute or relative workloads were employed.

Because HR remained elevated throughout the period of V˙O2 recovery, it is obvious that recovery mechanisms for HR and V˙O2 differ somehow, but there is no present explanation for this phenomenon. For brief periods of exercise, short-term recovery HR dynamics have been closely correlated to plasma norepinephrine (NE) levels (19). Elevated NE was associated with the postexercise V˙O2 (r = 0.78) in previous work, but a correlation with HR was not provided (11). Others indicate a relationship between NE and prolonged EPOC periods, but the exercise bouts used in those studies have been considerably longer and sufficient HR data were not given (3, 30). The extended increase in HR could be due to several factors including altered circulatory dynamics, prolonged sensitivity of cardiac tissues to elevated temperature, hormones or metabolites, or mild psychological arousal induced by the activity. Unfortunately, these ideas remain speculative, since there are no reports to date that have taken all of the appropriate measures for long periods after vigorous exercise.

Recovery RER. There was a brief increase in RER immediately after the conclusion of exercise. The transient upward deflection represents a type of relative hyperventilation. During this time, the decline in V˙O2 exceeds the rate of adjustment in ventilation (15), so that more CO2 is released from the lungs. Thereafter, ventilation rates fall into line with oxygen demands, and the drive to restore acid-base balance causes CO2 retention and a rapid fall in RER (1). This pattern is most noticeable after high-intensity exercise bouts that elicit high ventilatory rates and put greater stress on acid-base regulation (4, 6). After both exercise conditions, the RER peak was not as large for Tr, indicating less transient CO2 shift and better control of the metabolic stress during and immediately after exercise (15). Once again, this could be related to difference in the work rates of each group relative to lactate threshold (20).

Following the peak, RER declined steadily for 15–20 min before stabilizing near or below baseline in each trial. The RER immediately after exercise may not be an accurate representation of fuel utilization for the reasons already mentioned, but the adjustments in CO2
balance should be complete within several minutes. Thus the decrease in RER during during the majority of EPOC is evidence of increased fat oxidation. This occurrence is noted after a range of exercise conditions (2, 4, 6, 8, 16, 24). The RER decline tends to be greater after higher intensity bouts (4, 6), although the opposite has also been reported (8). Increased fat oxidation and a concomitant increase in cycling of triacylglycerols and fatty acids have been shown to make significant contributions to postexercise energy expenditure (3, 30). Trained subjects are known to have higher oxidative capacities in trained muscle groups (23), which might be expected to result in greater fat utilization and lower RER after exercise (8). These data are not in support of this idea, however, as RER tended to be the same or lower for UT than Tr in both trials. Because exercise RER was higher for UT even when relative exercise intensity was controlled, the metabolic response to the exercise was not the same for each group, and this difference may account for the postexercise responses.

Summary. After exercising for 30 min at either the same relative or absolute work rate, trained subjects had shorter EPOC duration and faster initial rates of recovery than untrained subjects. This report is in agreement with others that have demonstrated shorter EPOC and faster recovery in trained subjects (11, 13, 15). Additionally, the strong influence of exercise intensity on the magnitude and duration of EPOC and the recovery patterns of RER and HR have been confirmed. These findings demonstrate that aerobic training is responsible for metabolic adaptations that allow more efficient adjustment of energy expenditure during exercise recovery.

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