Exercise intensity: effect on postexercise O₂ uptake in trained and untrained women

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In weight reduction programs the energy cost of exercise is generally assessed from the O₂ uptake (VO₂) during the activity, with the well-documented elevation of metabolism after exercise, variously referred to as O₂ debt (22), recovery O₂ (37), or, more recently, excess postexercise O₂ consumption (EPOC) (17) ignored.

Hill et al. (22) and Margaria et al. (28) found increased resting metabolism for several hours after exercise but considered the long-term effect to be something other than the recovery process and eliminated it from their recovery O₂ measurements. Research on O₂ debt since then has concentrated on the two phases described by Margaria et al. (28) as the rapid lactic acid phase and the slower lactacid phase, which was purportedly due to the removal of lactic acid and resynthesis of glycogen. Other authors have since ascribed at least part of the EPOC to the increase in rate of chemical reaction for each 10°C increase in temperature (Q₁₀) effect of elevated body temperatures (7, 19). None of these studies, however, has considered the long-term elevation of metabolism referred to by Hill et al. (22) and confirmed by many other authors (3, 6, 13-15, 20, 27, 34).

Little is known about the factors associated with the prolonged elevation of metabolism after exercise. In the past, studies have found that resting metabolic rate (RMR) was restored within 1 h after 3–20 min of exercise at 30–55% of maximal O₂ consumption (VO₂ max) (33, 36), after 40 min of exercise at ventilatory threshold (16), or after exercise to exhaustion at 90% VO₂ max (1). However, other investigators (3, 6, 20, 27) have shown that metabolic rate after 1–3 h of exercise at 50–75% VO₂ max was still significantly elevated above rest 5–12 h after completion of exercise. It would appear from these studies that if exercise duration is too short (even with high intensity) or intensity is too low, no long-lasting calorigenic effect of exercise is observed. Moderate exercise maintained for a sufficient period of time may be more successful in elevating postexercise metabolic rate hours after exercise cessation (11).

Chad and Wenger (10, 11) found that when exercise intensity or total work was equated, elevation of postexercise metabolism was related to exercise duration. Core temperatures returned to resting levels within 2 h, whereas VO₂ remained elevated for 2–8 h. Since then a recent study has been published indicating that, in untrained males, 3-h recovery of VO₂ was positively related to both duration and intensity of exercise (18).

The effect of exercise intensity, independent of duration, on the extended EPOC has not been studied in women. This study investigated the effect of constant duration and varying intensity (and thereby total work) on the postexercise metabolic rate. The effects of 30 min of cycling at 50 and 70% VO₂ max on 3-h recovery of VO₂ were determined. Exercise intensity and duration were chosen as typical of recommendations for aerobic conditioning (2). Because training status influences the intensity of exercise that can be maintained for a given duration, trained female cyclists and untrained females were compared.

Methods

Subjects. Five trained female cyclists (age 25.2 ± 1.72 yr) and five untrained females (age 27.2 ± 0.75 yr) volunteered to participate in the study. The cyclists averaged at least 300 km/wk, and the untrained individuals did no formal exercise. All subjects were eumenorrheic and were not in the menstrual phase of the female cycle during the time of the experiment. Before participation, all subjects...
completed an informed voluntary consent form and a medical history questionnaire. The procedures were approved by the Human Experimentation Ethical Review Committee of the University of Queensland.

**Experimental protocol.** One week before the 1st day of testing, height and skinfold measurements were taken. Seven skinfold sites (triceps, subscapular, suprailiac, umbilical, biceps, thigh, and medial midcalf) were measured to the nearest 0.1 mm with Harpenden calipers, with the sum of the seven being recorded for each subject (38). The weight of each subject was measured on a beam scale and recorded to the nearest 0.1 kg before commencement of each testing session. Before the testing, the subjects were also familiarized with cycle ergometer exercise at a constant pedaling rate while breathing through a mouthpiece and valve that were to be used in all metabolic measurements.

All subjects performed a stepwise incremental test (39) to measure $\dot{V}O_2$ max. The $\dot{V}O_2$ max test, performed on a Monark cycle ergometer, consisted of a 5-min warm-up at 100 (trained) or 50 W (untrained), after which time the load was increased by 25 W every 2 min. Pedaling speed was constant at 90 and 60 revolutions/min (trained and untrained, respectively) (39). Verbal encouragement was given to each subject to attain maximal values. Attainment of $\dot{V}O_2$ max was based on the following criteria: 1) a plateau or change of <150 ml/min in $\dot{V}O_2$ subsequent to an increase in work load, 2) the attainment of estimated maximum heart rate (220 - age), and 3) a respiratory exchange ratio (R) >1.00. Pulmonary ventilation, $\dot{V}O_2$ and CO$_2$ output were measured and recorded during the last minute of every 2-min stage. $\dot{V}O_2$ was then plotted against power output to determine the work rates at 50 and 70% $\dot{V}O_2$ max, for each subject. This estimation was confirmed by measurement of $\dot{V}O_2$ at that power output during a 10-min pretest conducted 2 days later.

One week after the confirmation of test work rates, each subject was tested at approximately the same time of day for two trials scheduled 1 wk apart. The protocols for the 2 days of testing were identical with the exception of the exercise intensity (1 at 50% $\dot{V}O_2$ max and 1 at 70% $\dot{V}O_2$ max), with duration held constant at 30 min. Once they reported to the laboratory (after a 12-h fast), the subjects consumed no food or beverage, with the exception of water, until the test was completed. All subjects were requested to maintain a mixed diet 1 wk before and throughout the experiment. This was confirmed from dietary records.

RMR was determined by taking the mean of six 5-min $\dot{V}O_2$ measurements, which were sampled over a continuous period of 30 min to ensure stability of the baseline. The subjects sat quietly on a chair for 1 h before this measurement. They were allowed to read, write, or watch television during this rest period. After RMR was established for 30 min, they were allowed to stand up, sit on a chair, and are familiarized with cycle ergometer exercise at a constant pedaling rate while breathing through a mouthpiece and valve that were to be used in all metabolic measurements. After exercise, subjects returned to the sitting position to read, write, or watch television as in the preexercise phase for a period of 3 h. Subjects were dressed in shorts and T-shirts and wore running shoes throughout the test. The laboratory was air conditioned and held at a constant temperature of 22 ± 0.5°C.

All ventilatory measurements were made by standard open-circuit spirometry. For at least 5 min before sampling, subjects wore noseclips and breathed through a Hans Rudolph low-resistance low-dead-space valve (model 2700) that was connected to a mixing chamber via lightweight tubing. The volume of air inspired during measurement periods was measured by a turbine ventilometer (Morgan Mark 2). Volume of expired air was calculated from this measurement by use of the Haldane transformation. All volumes were corrected to STPD. Morgan CO$_2$ (infrared type 901 MK2) and O$_2$ (paramagnetic model OA 500D) analyzers were used for measurement of respiratory gases. Calibration of the gas analyzers by use of certified gravimetric calibration gas and the ventilometer was conducted before and after the preexercise phase, 15 min after exercise, and, thereafter, immediately before each postexercise measurement. The Monark cycle ergometer was calibrated before each exercise bout. Heart rate was recorded from chest electrodes by a Rigel cardiac monitor (model 302).

Five-minute ventilatory measurements were taken during the rest (preexercise) and exercise periods. In the postexercise phase, a 5-min ventilatory sample was taken from 10 to 15 min and, thereafter, every 40–45 min for the next 3 h, which was selected as the predetermined recovery period because of fasting and time constraints.

To seek an explanation for the unexpected results, $R$ values were subsequently used to estimate the proportional contribution from fat and carbohydrate in total energy yield. Absolute fat metabolism was also estimated. Because the work performed was predominantly aerobic, the proportions of O$_2$ consumed in the oxidation of fatty acids and carbohydrate were calculated with a nonprotein $R$ assumed and by use of the accepted values for fatty acid (R = 0.70) and carbohydrate (R = 1.00):%
fatty acid = (1 - R)/0.3 X 100% and %carbohydrate = (R - 0.7)/0.3 X 100%. By use of palmitic acid as a typical fatty acid, yielding an $R$ = 0.7, the absolute quantity of fatty acid oxidized may be calculated from the equation:

$$
\text{Oxidation of } 1 \text{ mol of fatty acid requires } 23 \text{ mol of } O_2 \text{ or } (23 \times 22.4) \text{ liters because } 1 \text{ mol of a gas occupies } 22.4 \text{ liters (STPD).}
$$

One liter of O$_2$, therefore, accounts for the oxidation of 1000/(23 X 22.4) liters or 1.94 mmol of fatty acid. Millimoles of fatty acid are converted to milligrams by multiplying by the gram molecular weight, in this case 256, yielding ~500 mg fatty acid/l O$_2$ consumed in the oxidation of fatty acid.

**Data analysis.** The physical characteristics of the trained and untrained subjects were compared by Student's t test for unpaired samples.

For resting and exercise $\dot{V}O_2$ and R values differences between means of six 5-min samples were analyzed across fitness group (trained vs. untrained) and test day (50% $\dot{V}O_2$ max vs. 70% $\dot{V}O_2$ max) by a two-way analysis of variance with repeated measures on test day.

Postexercise comparisons were made by three-way analysis of variance across fitness level, exercise intensity, and time into recovery (15, 60, 105, 150, and 180 min), with repeated measures on exercise intensity and time. Differences between trained and untrained groups, between exercise intensities at each individual sampling
time in recovery, and among sampling times were assessed by Student's t test for unpaired and paired samples, respectively, with a Bonferroni correction for multiple comparisons. $\bar{V}O_2$ values were analyzed as excess postexercise $\bar{V}O_2$ by subtracting resting levels before analysis. Comparisons between the 50 and 70% $\bar{V}O_{2\text{max}}$ recoveries were not altered when the analysis was performed on total rather than excess $\bar{V}O_2$ values.

Results are expressed as means ± SD. $P < 0.05$ was accepted as the level of significance for the analyses of variance and as the alpha level from which Bonferroni corrections were made for multiple comparisons.

RESULTS

Physical characteristics of the subjects. The physical characteristics of the subjects in each group are presented in Table 1. The trained individuals were significantly lighter in weight ($P < 0.05$) and had smaller skinfold measurements ($P < 0.01$) than the untrained individuals. No significant difference in age or height was observed between the two groups. The mean $\bar{V}O_{2\text{max}}$ of the trained group was substantially higher ($P < 0.01$) than that of the untrained group.

Rest. No significant difference was observed in RMR between the 2 days of testing. The data were highly reproducible, with no individual's mean resting $\bar{V}O_2$ varying by >0.01 l/min on the two occasions. The trained group had significantly lower resting $\bar{V}O_2$ values (l/min) than the untrained, but this was largely related to the difference in body weight, and the difference was not significant when $\bar{V}O_2$ was expressed as milliliters per kilogram per minute, $2.92 ± 0.11$ and $3.06 ± 0.10$, respectively.

The resting R values of the trained ($0.85 ± 0.01$) and untrained ($0.84 ± 0.01$) groups were not significantly different, indicating that both groups were metabolizing approximately equal amounts of fat and carbohydrates in the resting state. The mean value for each group did not vary, and individual values were all within 0.01 on the two test days.

Exercise. For the 30 min of exercise, the trained group worked at a mean $\bar{V}O_2$ of $51.1 ± 0.9$ and $71.03 ± 1.95\% \bar{V}O_{2\text{max}}$, and the untrained group worked at $53.02 ± 1.9$ and $72.56 ± 0.79\% \bar{V}O_{2\text{max}}$ (no significant difference between groups). Because of the higher $\bar{V}O_{2\text{max}}$ of the trained subjects, their mean work rate, and hence their $\bar{V}O_2$ values were significantly higher ($P < 0.01$) than those of the untrained subjects during each exercise period (trained at 70% $\bar{V}O_{2\text{max}}$ 2.21 ± 0.07 l/min, untrained at 70% $\bar{V}O_{2\text{max}}$ 1.28 ± 0.10 l/min, trained at 50% $\bar{V}O_{2\text{max}}$ 1.59 ± 0.06 l/min, untrained at 50% $\bar{V}O_{2\text{max}}$ 0.94 ± 0.09 l/min). During the 50% $\bar{V}O_{2\text{max}}$ exercise bout, the trained subjects worked at 85 ± 15 W, whereas during the 70% $\bar{V}O_{2\text{max}}$ work bout they exercised at 115 ± 15 W. The untrained group worked at 45 ± 10 and 80 ± 10 W, which elicited an $\bar{V}O_2$ equivalent to ~50 and 70% $\bar{V}O_{2\text{max}}$, respectively.

The R values during exercise were significantly higher at 70% $\bar{V}O_{2\text{max}}$ than at 50%. The trained subjects had significantly lower R values at both exercise intensities ($P < 0.01$; trained at 70% $\bar{V}O_{2\text{max}}$ 0.94 ± 0.09 l/min, trained at 50% $\bar{V}O_{2\text{max}}$ 0.98 ± 0.01, untrained at 50% $\bar{V}O_{2\text{max}}$ 0.92 ± 0.01, untrained at 50% $\bar{V}O_{2\text{max}}$ 0.94 ± 0.01).

Postexercise. The time course of postexercise $\bar{V}O_2$ is shown in Figs. 1 (trained) and 2 (untrained). $\bar{V}O_2$ at every sample period in the postexercise phase was significantly elevated ($P < 0.01$) above the pretest resting level after the two exercise intensities in both the trained (Fig. 1) and the untrained (Fig. 2) groups. At the end of 3 h of recovery, mean $\bar{V}O_2$ was still elevated by 105 (untrained) and 135% (trained) after 50% $\bar{V}O_{2\text{max}}$ and by 53 (untrained) and 82% (trained) after 70% $\bar{V}O_{2\text{max}}$. In the last 2 h, the mean decline was only 0.04 (untrained) to 0.06 (trained) and 0.02 (untrained and trained) l/min in each case.

From the first sample, 10–15 min into recovery, the $\bar{V}O_2$ after work at 50% $\bar{V}O_{2\text{max}}$ remained significantly higher at every time period than after work at 70% $\bar{V}O_{2\text{max}}$ for both groups and each of the 10 individual subjects. The mean difference throughout was 104 ± 18 ml/min. The high reproducibility of the data can be seen from the low intersubject variability and the consistency of the values throughout the last 2 h of sampling in both groups.

### Table 1. Physical characteristics of untrained and trained subjects

<table>
<thead>
<tr>
<th>Subj No.</th>
<th>Age, yr</th>
<th>Ht, m</th>
<th>Wt, kg</th>
<th>Sum of Skinfolds, mm</th>
<th>$\bar{V}O_{2\text{max}}, \text{l/min}$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Untrained</td>
</tr>
<tr>
<td>1</td>
<td>28</td>
<td>1.73</td>
<td>59.0</td>
<td>12.0</td>
<td>1.73</td>
</tr>
<tr>
<td>2</td>
<td>28</td>
<td>1.52</td>
<td>59.5</td>
<td>13.0</td>
<td>1.74</td>
</tr>
<tr>
<td>3</td>
<td>26</td>
<td>1.54</td>
<td>60.0</td>
<td>14.5</td>
<td>1.80</td>
</tr>
<tr>
<td>4</td>
<td>27</td>
<td>1.67</td>
<td>67.5</td>
<td>16.3</td>
<td>1.96</td>
</tr>
<tr>
<td>5</td>
<td>27</td>
<td>1.64</td>
<td>62.5</td>
<td>15.2</td>
<td>1.60</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>27.2±0.75</td>
<td>1.62±0.08</td>
<td>61.7±3.14</td>
<td>147.9±21.65</td>
<td>1.77±0.12</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Trained</td>
</tr>
<tr>
<td>6</td>
<td>24</td>
<td>1.70</td>
<td>54.0</td>
<td>80.5</td>
<td>2.95</td>
</tr>
<tr>
<td>7</td>
<td>23</td>
<td>1.65</td>
<td>60.0</td>
<td>107.0</td>
<td>3.21</td>
</tr>
<tr>
<td>8</td>
<td>28</td>
<td>1.72</td>
<td>59.0</td>
<td>99.0</td>
<td>2.98</td>
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<td>9</td>
<td>25</td>
<td>1.73</td>
<td>57.0</td>
<td>85.0</td>
<td>3.10</td>
</tr>
<tr>
<td>10</td>
<td>26</td>
<td>1.64</td>
<td>55.5</td>
<td>98.3</td>
<td>3.31</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>25.2±1.72</td>
<td>1.69±0.04</td>
<td>57.1±2.20</td>
<td>92.68±9.33</td>
<td>3.11±0.13</td>
</tr>
</tbody>
</table>

$n = 10$ women.
After both exercise intensities, the trained group had a significantly higher \( \text{VO}_2 \) than the untrained group, and \( \text{VO}_2 \) remained higher throughout the postexercise period, although a post hoc analysis of the individual samples showed only the first samples at 10–15 min to be significantly different.

Pairwise comparisons across recovery time showed that, after exercise at 50% \( \text{VO}_{2\text{max}} \), significant falls in \( \text{VO}_2 \) occurred between samples in both trained and untrained subjects until 105 min. After exercise at 70% \( \text{VO}_{2\text{max}} \), significant falls occurred in both groups for only 60 min, even though the \( \text{VO}_2 \) values during the exercise were considerably higher than at 50% \( \text{VO}_{2\text{max}} \).

\( R \) was significantly lower after 50% \( \text{VO}_{2\text{max}} \) than after 70% \( \text{VO}_{2\text{max}} \) at every time point for both groups of subjects (Figs. 1 and 2). In the trained group, \( R \) fell below the resting level within the 1st h. In the untrained subjects, \( R \) reached the resting level within 1 h after 50% \( \text{VO}_{2\text{max}} \) but not until 150 min after 70% \( \text{VO}_{2\text{max}} \). \( R \) followed a pattern similar to \( \text{VO}_2 \) in that it fell significantly between samples for 105 min after 50% \( \text{VO}_{2\text{max}} \) and for 60 min after 70% \( \text{VO}_{2\text{max}} \).

**Estimated fat metabolism.** Because fat metabolism seemed to be one of the few possible explanations of a higher \( \text{VO}_2 \) and lower \( R \) after 50% than after 70% \( \text{VO}_{2\text{max}} \), fat metabolism was calculated based on the tentative assumption that \( R \) approximated the true respiratory quotient (see DISCUSSION).

Estimates of resting fat metabolism were consistent on the 2 days for each group: 51 ± 4 (untrained) and 43 ± 3 mg/min (trained). Individual values were all within 7 mg/min on the two test days.

Estimates of fat metabolism during exercise suggested that untrained subjects metabolized approximately twice as much fat at 50% (102 ± 19 mg/min) as at 70% \( \text{VO}_{2\text{max}} \) (53 ± 20 mg/min). The trained subjects appeared to metabolize ~26% more fat at 50% (225 ± 13 mg/min) than at 70% \( \text{VO}_{2\text{max}} \) (178 ± 24 mg/min). The trained subjects seemingly metabolized more than twice as much fat in absolute terms as the untrained subjects at 50% \( \text{VO}_{2\text{max}} \) and more than three times as much at 70% \( \text{VO}_{2\text{max}} \) during exercise at 50% and 75% \( \text{VO}_{2\text{max}} \) in trained subjects (\( n = 8 \)).

**FIG. 1.** Postexercise \( \text{VO}_2 \), respiratory exchange ratio \( R \), and fat oxidation (FAT) after 30 min of exercise at 50% and 75% \( \text{VO}_{2\text{max}} \) in trained subjects (\( n = 8 \)).

* \( p < 0.01 \) 50% vs 70% \( \text{VO}_{2\text{max}} \)
+ \( p < 0.01 \) 50% \( \text{VO}_{2\text{max}} \) vs rest
\( \times \) \( p < 0.01 \) 70% \( \text{VO}_{2\text{max}} \) vs rest

**FIG. 2.** Postexercise \( \text{VO}_2 \), \( R \), and fat oxidation after 30 min of exercise at 50% and 75% \( \text{VO}_{2\text{max}} \) in untrained subjects (\( n = 5 \)).

* \( p < 0.01 \) 50% vs 70% \( \text{VO}_{2\text{max}} \)
+ \( p < 0.01 \) 50% \( \text{VO}_{2\text{max}} \) vs rest
\( \times \) \( p < 0.01 \) 70% \( \text{VO}_{2\text{max}} \) vs rest
the 30-min exercise bout. At 70% \( \dot{V}O_2 \) max, the untrained subjects appeared to be metabolizing almost exclusively carbohydrate for their exercise needs.

 Estimates of recovery fatty acid oxidation showed significantly elevated levels of fat metabolism throughout the 3 h of the measurement period in both tests \((P < 0.01)\). The estimated fat metabolism was significantly greater at every sample after 50% \( \dot{V}O_2 \) max than after 70% \( \dot{V}O_2 \) max (Figs. 1 and 2) for both groups. At the end of 3 h, estimated fat metabolism after 50% \( \dot{V}O_2 \) max was still 56% higher \((133 \pm 9 \text{ mg/min})\) than after 70% \( \dot{V}O_2 \) max \((85 \pm 7 \text{ mg/min})\) in untrained subjects and 45% higher \((147 \pm 4 \text{ vs. } 101 \pm 6 \text{ mg/min})\) in trained subjects. Trained subjects still appeared to be metabolizing 10 and 18% more fat than untrained subjects after 50 and 70% \( \dot{V}O_2 \) max, respectively.

DISCUSSION

 Previous studies equating exercise intensity and varying duration or equating total work and varying intensity and duration \((10, 11)\) have suggested that duration of exercise was more important than intensity in determining the extent of the elevation of metabolism after aerobic exercise. The present study has shown, in young women, that intensity of exercise is also an important factor in the long-term elevation of \( \dot{V}O_2 \) after exercise. With duration held constant at 30 min, cycle ergometer exercise at 50% \( \dot{V}O_2 \) max produced consistently higher \( \dot{V}O_2 \) values after the first 10 min of recovery than at 70% \( \dot{V}O_2 \) max. The situation was the same for all 10 subjects, both trained and untrained, and at every sample period for every subject for a total of 510 data points. The variability of the measures was extremely low at rest, during exercise, and in recovery, indicating that random experimental error was minimal. Within each training group, intersubject variability was also very low. Although a third control condition without exercise was omitted for practical reasons, other studies have found little or no change in resting metabolism of postabsorptive subjects during a similar 4-h period \((6, 27, 33)\). Where changes have been found \((35)\), they have been of considerably lower magnitude than the postexercise elevations found in this study. In comparisons of the two exercise intensities, each subject acted as her own control, and there is no indication that minor variations in the very consistent resting \( \dot{V}O_2 \) values could have had any substantial part in explaining the findings. The evidence appeared incontrovertible.

These findings are not explained by the previously held views on the mechanisms postulated for the postexercise \( \dot{V}O_2 \), i.e., creatine phosphate replenishment, lactic acid removal, glycogen resynthesis, and the effects of increased core and muscle temperature \((17)\), because these mechanisms would increasingly come into play as the intensity level of exercise increases. However, in the past decade research has shown that these factors may not be the only determinants of postexercise \( \dot{V}O_2 \) because of the following: lactic acid has been shown to be an unlikely cause of the increased metabolism observed several hours after exercise cessation; increased temperatures return to resting levels before RMR has been reached \((11)\); creatine phosphate replenishment may only represent a small amount of the total postexercise \( \dot{O}_2 \) cost \((20)\); and, because of the slow process of the resynthesis of muscle glycogen \((23)\), it is questionable whether this mechanism can be used entirely to explain the elevation in postexercise \( \dot{V}O_2 \). It is possible, therefore, that although these mechanisms may be predominant early in the recovery phase and still remain active during the slow component of recovery, an additional mechanism must assume greater importance in the long-term postexercise \( \dot{V}O_2 \).

One of the few possible explanations for increased \( \dot{V}O_2 \) after lower exercise intensity is the metabolism of fat, a mechanism suggested recently by other authors \((3, 27)\). The respiratory quotient has commonly been taken to indicate the proportion of fat and carbohydrate metabolized under steady-state conditions. The use of \( R \) as an estimate of the nonprotein respiratory quotient is not without risk because a change in the acidity of the blood, a relative hyperventilation, or a change in dissolved CO

\( \text{d}^- \) uptake observed during the last 20 min of the exercise tests. Furthermore, the shape of the recovery curve of \( R \) was similar for both exercise intensities and both groups. Any effect of acidity or hyperventilation would be expected to occur in the samples at the end of exercise and 10–15 min into recovery, resulting in a more steeply sloping curve after 70% \( \dot{V}O_2 \) max. No such effect was apparent. \( R \) values were consistently lower after exercise at 50% \( \dot{V}O_2 \) max and went below the preexercise resting level sooner, suggesting that fat metabolism was the major energy source after the lower exercise intensity. Estimation of the absolute fat metabolism supported the view that more fat was metabolized after 50% than after 70% \( \dot{V}O_2 \) max. The alternative that \( R \) was consistently reduced by a net uptake of \( \text{CO}_2 \) in the tissues seems unlikely over a 3 h period.

The results from the present study once again confirm the view that postexercise metabolism may be elevated for several hours, depending on the duration and intensity of the exercise being performed. There is a long and somewhat neglected history of similar findings \((6, 13–15, 20, 22, 27, 28, 34)\). All these studies support the view that the elevation of metabolism after exercise extends beyond the replenishment of creatine phosphate and \( \text{O}_2 \) stores, removal of lactate, and \( R \) effects.

Much of the confusion and conflict apparently existing in the literature on \( \dot{O}_2 \) debt is related to differences in
experimental design and methods. Most studies have restricted their interest to the first two of the three components of postexercise elevation of $V_{O_2}$. In these cases, the measurement of recovery $V_{O_2}$ has generally been limited to short periods, often with exercise baselines, with the specific intention of eliminating the long-term elevation of metabolism.

Pacy et al. (33) concluded that there was no prolonged thermogenic effect of moderate repeated aerobic exercise in weight-maintaining lean subjects. The lack of significance may have been due to the small sample size and the increased variance due to the mixed-gender sample and the range of exercise intensities. The exercise loads were reported to represent 55-60% $V_{O_2\text{max}}$, although no mention was made as to how this was determined.

Gore and Withers (18) also found that in well-trained male subjects EPOC was of little physiological significance. It is possible that the observed differences in the two studies were either gender or experimental-design related. Although the ≥8-h measurement of RMR was performed by Gore and Withers (18) to account for circadian variations, the effect of increased thermogenesis on metabolic rate may have elevated the "resting" state and hence underestimated EPOC.

The weight of accumulating evidence suggests that postexercise metabolism may be raised for several hours depending on the duration and intensity of exercise. Exercise of sufficiently short duration or low intensity may minimize the effect. In the present study, exercise for 30 min at 50% $V_{O_2\text{max}}$ produced a greater extended EPOC than after 70% $V_{O_2\text{max}}$ in both trained and untrained females. Trained individuals had significantly greater extended EPOCs than untrained. R values were lower after 50% $V_{O_2\text{max}}$ and in trained subjects, suggesting fat metabolism as a possible mechanism. Estimates of fat metabolism support this view both in this study and in two recent studies (6, 27). Subsequent studies in this laboratory have also supported this hypothesis. When fat metabolism was stimulated by the ingestion of caffeine (9) or by dietary manipulation (8), the extended EPOC was higher than in control conditions. The precise mechanism for increased fat metabolism in recovery is not yet clear. Increased substrate cycling, as proposed by Newsholme (31, 32), is one possibility. The conversion of fat to carbohydrate is also less for fat than for carbohydrate metabolism. The conversion of fat to carbohydrate stores glycogen stores has also been considered but does not appear to be a major factor (6, 27). Subsequent studies were either gender or experimental-design related. Although the 28-h measurement of RMR was performed by Gore and Withers (18) to account for circadian variations, the effect of increased thermogenesis on metabolic rate may have elevated the "resting" state and hence underestimated EPOC.

The present results suggest that 30 min of moderate exercise may produce a distinct postexercise elevation of metabolism, possibly that of fat; this may be important in weight loss, particularly in middle-aged people, for whom high-intensity exercise may constitute an increased cardiac risk.

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