Effect of duration of exercise on excess postexercise \( \text{O}_2 \) consumption

ROALD BAHR, IVAR ING NES, ODD VAAGE, OLE M. SEJERSTED, AND ERIC A. NEWSHOLME
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Bahr, Roald, Ivar Ingnes, Odd Vaage, Ole M. Sejersted, and Eric A. Newsholme. Effect of duration of exercise on excess postexercise \( \text{O}_2 \) consumption. J. Appl. Physiol. 62(2): 485–490, 1987.—This study was undertaken to determine the effect of exercise duration on the time course and magnitude of excess postexercise \( \text{O}_2 \) consumption (EPOC). Six healthy male subjects exercised on separate days for 80, 40, and 20 min at 70% of maximal \( \text{O}_2 \) consumption on a cycle ergometer. A control experiment without exercise was performed. \( \text{O}_2 \) uptake, respiratory exchange ratio (R), and rectal temperature were monitored while the subjects rested in bed 24 h postexercise. An increase in \( \text{O}_2 \) uptake lasting 12 h was observed for all exercise durations, but no increase was seen after 24 h. The magnitude of 12-h EPOC was proportional to exercise duration and equaled 14.4 ± 1.2, 6.8 ± 1.7, and 5.1 ± 1.2% after 80, 40, and 20 min of exercise, respectively. On the average, 12-h EPOC equaled 15.2 ± 2.0% of total exercise \( \text{O}_2 \) consumption (EOC). There was no difference in EPOC:EOC for different exercise durations. A linear decrease with exercise duration was observed in R between 2 and 24 h postexercise. No change was observed in recovery rectal temperature. It is concluded that EPOC increases linearly with exercise duration at a work intensity of 70% of maximal \( \text{O}_2 \) consumption.

Materials and Methods

Subjects. Six male subjects participated in the study. Their physical characteristics are shown in Table 1. After a medical examination, they were fully informed about all procedures before written consent was obtained. All subjects were physically active, but not engaged in endurance training. Their maximal \( \text{O}_2 \) uptake values are similar to those reported as high average for Norwegian males (15).

Testing procedures. Before the testing started the subjects were familiarized with bicycle exercise at a constant pedalling rate and with breathing through the mouth-piece and the breathing valve used in all metabolic measurements. All testing and experimental exercise procedures were performed on a modified Krogh cycle ergometer. About 2 wk before the experiment started maximal \( \text{O}_2 \) uptake was measured using the criteria of Taylor et al. (29). These results were used to predict a work load corresponding to 70% of maximal \( \text{O}_2 \) uptake in each subject. Finally, this work load was used in a 10-min trial run to familiarize the subjects with their subsequent work intensity. The results from the tests are shown in Table 1.

Experimental protocol. Each subject participated in four experiments. The subjects reported to the laboratory at 7:00 a.m. after an overnight fast. They were transported by car, to avoid any physical activity on the morning of each experiment. They were told not to make any changes in their dietary or exercise habits, except that they were asked not to partake in any exercise for 2 days before days of experiment. No tobacco or alcohol was allowed 24 h before each experiment.

Body weight was measured at the start of each test session. A thermistor (type DU 3S, Elles Instruments A/S, Copenhagen, Denmark) was inserted 10–15 cm into the rectum. After that the subjects rested in bed until the exercise period started.

IT IS WELL-ESTABLISHED that \( \text{O}_2 \) consumption remains elevated for some period of time after exercise and the phenomenon has recently been termed excess postexercise \( \text{O}_2 \) consumption (EPOC) (11). Several aspects of the EPOC are still debated: i.e., estimations of its duration and magnitude, and relationship to the total work performed. In this study the previous work of Hermansen et al. (16) has been extended to investigate the effect of 20, 40, and 80 min of exercise on EPOC.

From previous studies it has not been possible to compare quantitatively the magnitude of EPOC and the total exercise \( \text{O}_2 \) consumption. If EPOC is related to the utilization of some energy store during exercise, the magnitude of EPOC would be expected to be closely related to the total work performed. In this study the variation of some energy store during exercise, the magnitude of EPOC would be expected to be closely related to the total work performed.
On three separate days the subjects exercised for 80, 40, or 20 min. A control experiment without exercise was also performed. Otherwise the conditions were identical to the exercise experiments. Experimental days were separated by ~2 wk and the sequence of the experiments (80, 40, or 20 min of exercise or control) was randomized. The start of exercise was adjusted so that the recovery period always started at 9:30 A.M. Three subjects were unable to complete the 80-min exercise period due to exhaustion, and their exercise times are shown in Table 1. After exercise the subjects rested in bed for 24 h, but they were not allowed to sleep for the first 12 h of recovery.

The subjects were given three meals consisting of bread, jam, and skimmed milk during the experiment. The average caloric content per meal was 1,200 kcal. The first meal was taken 2 h, the second 7 h, and the last 12 h after exercise. All meals were completed within 30 min. For every subject the amount of food taken at each meal was measured, and he was given an identical diet all four experiments.

**TABLE 1. Physical characteristics and exercise data for each subject**

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Age, yr</th>
<th>Ht, cm</th>
<th>Wt, kg</th>
<th>V̇O₂max, ml·kg⁻¹·min⁻¹</th>
<th>Work Load, W</th>
<th>Exercise V̇O₂, % of max</th>
<th>Exercise Duration on Day 1, min</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>22</td>
<td>188</td>
<td>82.3</td>
<td>59.4</td>
<td>240</td>
<td>72</td>
<td>80</td>
</tr>
<tr>
<td>2</td>
<td>21</td>
<td>181</td>
<td>70.3</td>
<td>57.2</td>
<td>193</td>
<td>73</td>
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<td>3</td>
<td>22</td>
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<td>71.5</td>
<td>62.2</td>
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<td>4</td>
<td>21</td>
<td>183</td>
<td>75.5</td>
<td>50.2</td>
<td>175</td>
<td>69</td>
<td>67</td>
</tr>
<tr>
<td>5</td>
<td>26</td>
<td>197</td>
<td>93.0</td>
<td>55.1</td>
<td>286</td>
<td>73</td>
<td>77</td>
</tr>
<tr>
<td>6</td>
<td>24</td>
<td>197</td>
<td>92.0</td>
<td>50.7</td>
<td>229</td>
<td>70</td>
<td>67</td>
</tr>
</tbody>
</table>

Mean ± SE 22.7±0.8 186.8±3.6 80.8±4.1 54.1±1.5 216.3±17.8 71.3±0.7 69.2±1.3 69.0±2.0 76.3±1.7

Missing value for subject 1 is due to an accidental technical malfunction. V̇O₂max, maximum O₂ uptake.

The postexercise period was divided into three intervals for statistical analysis of the data. The first interval lasts from the end of the exercise period until the first meal (0–2 h). The analysis of this interval provides information on the exclusive effect of exercise on EPOC. The data obtained during the next interval (2–7 h) is influenced not only by exercise, but also by any change the exercise period might induce on the thermic effect of food. The third interval (7–12 h) is identical to the second, except that it is an additional 5 h removed from the exercise period.

A linear relationship was observed between exercise duration and O₂ consumption and the increase was statistically significant for all three time intervals (Fig. 2). No difference was observed between the control and exercise experiments after 24 h of recovery (P > 0.25).

The total O₂ consumption for the first 12 h of recovery was 251.8 ± 10.5, 234.6 ± 7.7, 231.0 ± 8.1, and 219.9 ± 8.1 liters for 80, 40, and 20 min of exercise and the control experiment, respectively. The excess postexercise O₂ consumption (12-h EPOC) was calculated as the increase in O₂ consumption above control values for the first 12 h of recovery. Twelve-hour EPOC was 14.4 ± 1.2, 6.8 ± 1.7, and 5.1 ± 1.2% (SE) for 80, 40, and 20 min of exercise, respectively. A linear relationship was observed between the magnitude of 12-h EPOC and exercise duration (Fig. 3).

We have also calculated the 12-h EPOC as a percent of the total exercise O₂ consumption (EOC); this value was 15.2 ± 2.0% (mean for all experiments). For different exercise durations, values of 12-h EPOC were 13.6 ± 1.5, 12.2 ± 3.0, and 19.8 ± 4.7% of EOC after 80, 40, and 20 min of exercise, respectively (NS).

**Respiratory exchange ratio.** R increased from 0.78 ± 0.01 (mean morning value for all experiments) to 0.92 ± 0.04 (80 min period of exercise) and 0.92 ± 0.02 (for both the 40- and 20-min periods of exercise) at the end of the exercise period. Immediately after exercise, R fell sharply to those of the control experiment and for the interval between the end of the exercise period and the first meal (0–2 h), we observed no significant decrease in R for different exercise durations (Fig. 4). However, after the first (2–7 h) and second (7–12 h) meals we observed a

**RESULTS**

O₃ uptake. On the control day, O₂ consumption was 267 ± 13 ml/min in the morning, and after the first meal it was increased to 334 ± 13 ml/min. O₂ consumption then fell slowly during the next 5 h but was increased to approximately the previous value by the second meal (Fig. 1). Exercise increased the rate of O₂ consumption to 3,219 ± 189 ml/min (for 80 min duration of exercise), 3,076 ± 231 ml/min (40 min), and 3,049 ± 251 ml/min (20 min).

Linear relationships between work time and individual values for statistical analysis of the data. The first interval lasts from the end of the exercise period until the first meal (0–2 h). The analysis of this interval provides information on the exclusive effect of exercise on EPOC. The data obtained during the next interval (2–7 h) is influenced not only by exercise, but also by any change the exercise period might induce on the thermic effect of food. The third interval (7–12 h) is identical to the second, except that it is an additional 5 h removed from the exercise period.

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linear decrease in R with increasing exercise duration (Fig. 5). After 24 h there was still a linear decrease in R as the exercise duration increased ($P < 0.05$).

Rectal temperature. Rectal temperature rose to $38.4 \pm 0.2^\circ$C (80-min period of exercise), $37.8 \pm 0.3^\circ$C (40-min period of exercise) and $37.5 \pm 0.1^\circ$C (20-min period of exercise) at the end of the exercise period. The temperature decreased rapidly and reached control values in $\approx 30$ min on all exercise days. Thereafter, no significant difference was observed in rectal temperature between postexercise and control experiments.

**DISCUSSION**

This study shows that the magnitude of 12-h EPOC is linearly related to the duration of a prolonged submaximal exercise bout. Hence with this kind of exercise 12-h EPOC is a constant fraction equaling 15% of the total O$_2$ consumption during the exercise period.

Because of variability of exercise tasks and differences in experimental design, it is difficult to compare previous findings with those presented in this paper. The present study confirms the recently published findings of Machlum et al. (18) in which the mean excess postexercise O$_2$ uptake for 12 h was 14% above the basal after exhaustive work at a work intensity corresponding to 70% of maximal aerobic capacity. In other studies there is considerably variability concerning the effects of exercise on postexercise O$_2$ consumption. Thus three recent well-controlled investigations (5, 10, 21) failed to show any prolonged thermic effect of exercise, apparently in conflict with the present study. Since two other reports have indicated that an increase in severity of exercise (3, 9) causes an increase in EPOC, the variability in response...
may be explained by differences in exercise intensity, as suggested by Brehm and Gutin (5). The findings of these latter workers suggested the existence of a threshold value of about 50% of maximal aerobic capacity before any significant increase in recovery O$_2$ uptake was observed. It should also be noted that in all three studies (5, 10, 21) O$_2$ uptake was measured for only 1–3 h of the recovery period and in fasting subjects. We were unable to find in the literature any other study on the effect of duration of exercise on EPOC. Hence we conclude that with exercise intensity at approximately 70% of maximal O$_2$ uptake EPOC is clearly demonstrable for at least 12 h, and linearly related to exercise duration.

It is interesting to note that EPOC for 12 h is a constant percent of the total O$_2$ consumption during exercise, when exercise duration is varied. However, in view of Brehm and Gutin’s finding of a nonlinear relationship between exercise intensity and EPOC (5), a study of the effect of intensity of exercise on EPOC could be of considerable interest.

There is additional controversy over both the duration of EPOC and the effect of exercise plus feeding on EPOC. Previously it has been found that, even after 31 h of recovery from exercise, O$_2$ consumption was 8% above normal (9). In the present study, we observed an increase in the 7- to 12-h interval after exercise, but not after 24 h. However, since we have no data for the time period between 12 and 24 h, our values probably represent a minimal estimate of the extra energy consumption. Accordingly, other workers have suggested that the extra energy expenditure after exercise may be considerably greater than presently found during a 12-h period (2).

The study of Mæhlum et al. (18) indicated that EPOC was partly related to an effect of meals. However, other investigators have suggested either that exercise has no effect on the thermic effect of food (27) or that it is only increased if individuals had overeaten before the exercise (4, 19, 27, 30). In the present study a considerable part of the EPOC was observed following the first (2- to 7-h interval) and second (7- to 12-h interval) meals; 42% of 12-h EPOC was observed after the first meal and 22% after the second. Proper estimates of the meal effects will require control studies in which subjects are fasted after exercise. No such information is presently available.

The mechanisms responsible for stimulated O$_2$ consumption after exercise have been discussed by Herman et al. (16). Two explanations were proposed: increased rates of substrate ("futile") cycling after exercise and extra energy required for the synthesis of depleted glycogen stores.

Exercise at 70% of maximal O$_2$ uptake, as used in the present study, is known to deplete glycogen stores completely (17). This would, in a subject weighing 80 kg,
correspond to ~1.7 mmol glycosyl units. At an average R of 0.85, the O₂ cost of glycogen resynthesis would be ~20 liters. However, resynthesis of muscle glycogen is a fairly slow process that may take as much as 48 h (22). In the present study, the average EPOC during the first 12 h of recovery after 80 min exercise was 31.9 liters. Thus it is unlikely that the extra O₂ consumption would account for more than half of the EPOC.

There is now considerable indirect evidence to support the view that an increased rate of substrate (futile) cycles may be in part responsible for EPOC (6, 8, 16, 18, 20) and that raised level of catecholamines could be one important factor leading to increased cycling rates. The present findings are consistent with this viewpoint and, furthermore, they suggest that the mobilization of fat from the adipose tissue reserves and an increased rate of cycling between triacylglycerol and fatty acids may be responsible for EPOC.

It has been known for some time that, in normal subjects, ~20 min of exercise is necessary to increase the plasma levels of fatty acids (7) and, at an exercise intensity of ~60% of maximal aerobic capacity, the plasma fatty acid level increases progressively over 180 min of exercise (1). Furthermore, in this latter study, both the plasma levels of fatty acids and catecholamines were markedly elevated 40 min after exercise had ceased. These two factors may be responsible for stimulation of the rate of the triacylglycerol-fatty acid substrate cycle in adipose tissue. Since one turn of this cycle requires a net hydrolysis of eight molecules of ATP to ADP, an increase in the rate of this cycle would result in a marked increase in O₂ consumption after exercise. This cycle is known to occur in adipose tissue of the rat and mouse (6, 26) and may occur in muscle and liver, and catecholamines can increase the rate of this cycle in vivo (6). The postexercise decrease in the value of R (Fig. 4) indicates a greater proportion of fat being oxidized in our subjects and hence a higher plasma fatty acid level. Consequently one factor responsible for the relationship between duration of exercise and EPOC may be the gradual increase in the dependence on fatty acids as a fuel as the duration of the exercise increases. Once exercise has finished, the plasma level of fatty acids further increases, and this increase may be proportional to the rate of fatty acid utilization during the exercise period. One role of the triacylglycerol-fatty acid cycle in adipose tissue may be to decrease gradually and progressively the rate of fatty acid mobilization and, additionally, in other tissues, it may act as a system to buffer the intracellular levels of fatty acid and fatty acyl-CoA (20). Exercise of insufficient intensity or duration to raise the plasma fatty acid and/or catecholamine levels may not result in a marked EPOC.

The current findings may be of considerable practical importance in prevention of obesity and in the treatment of mild obesity. Thus the importance of exercise depends on the extra energy utilized due to the physical activity—not only during the activity per se but when the activity has ceased. Exercise has often been discounted because of the assumption that very long periods of exercise were necessary to produce any significant use of energy (12). The present work demonstrates that as little as 20 min of sustained exercise may be beneficial, since it increases energy consumption for a considerable period of time after the exercise is stopped. Furthermore, the thermic response to food may be increased after exercise and the subjects oxidized more fat after exercise despite the high carbohydrate nature of the diet, suggesting that this fat must be derived from the adipose tissue. These findings indicate that a regimen of daily exercise plus an energy-controlled diet should provide a satisfactory, long-term treatment for obesity.

In conclusion, this study demonstrates a linear increase in EPOC in relation to exercise duration, amounting to ~15% of exercise energy expenditure. In addition to the obvious practical implications in relation to obesity, these results indicate that the use of tables calculating the energy cost of exercise should be reevaluated to include the considerable excess postexercise energy expenditure.

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EXERCISE DURATION AND POSTEXERCISE \( O_2 \) CONSUMPTION

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