Effect of feeding and fasting on excess postexercise oxygen consumption

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BAHR, ROALD, AND OLE M. SEJERSTED. Effect of feeding and fasting on excess postexercise oxygen consumption. J. Appl. Physiol. 71(6): 2088-2093, 1991.—This study was undertaken to determine the effect of fasting on the magnitude and time course of the excess postexercise O2 consumption (EPOC). Six lean untrained subjects were studied in the fasted state for 7 h after a previous strenuous exercise bout (80 min at 75% of maximal O2 uptake) and in a control experiment. The results were compared with identical control and exercise experiments where the subjects were fed a 4.5-MJ test meal after 2 h of rest. EPOC was calculated as the difference in O2 uptake between the corresponding control and exercise experiments. The total EPOC (0-7 h postexercise) was 20.9 ± 4.5 (fasting) and 21.1 ± 3.6 liters (food, NS). A significant prolonged EPOC component was observed in the fasted and in the fed state. The thermic effect of food (TEF) was calculated from O2 consumption and respiratory exchange ratio as the difference in energy expenditure between the corresponding food and fasting experiments. The total TEF (0–5 h postprandial) was 321 ± 32.0 (control) and 280 ± 37.7 kJ/5 h (exercise, NS). It is concluded that the prolonged component of EPOC is present in the fasting state. Furthermore, no major interaction effects between food intake and exercise on the postexercise O2 consumption could be detected.

MATERIALS AND METHODS
Subjects. Six male students participated in the study (Table 1). All subjects were physically active but not engaged in regular training. After a medical examination, they were fully informed about all procedures before written consent was obtained. Data from related studies on the same subjects have been published elsewhere (3, 4).

Preexperimental procedures. Before the experiments started, all subjects were familiarized with bicycle exercise at a constant pedaling rate and with breathing through the mouthpiece 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 experiments started, maximal O2 uptake was measured using criteria modified for the cycle ergometer from Taylor et al. (26). In this protocol the subjects performed 3-min work bouts with 10-min resting periods and 19-W increments in work load. These results were used to predict work loads corresponding to 75% of maximal O2 uptake in each subject.

Experimental protocol. Each subject participated in four experiments, two exercise experiments (with and without food) and two control experiments (with and without food). Experiments were separated by 2 wk, and the sequence of the experiments was randomized. The subjects reported to the laboratory at 7:00 A.M. after an overnight fast. They were transported by car to avoid unnecessary physical activity before each experiment. The subjects were told not to partake in any exercise for 2 days before an experiment and not to make any changes in their dietary or exercise habits. No tobacco or alcohol was allowed 24 h before each experiment.
The urinary bladder was emptied, body weight was measured, and a thermistor (type DU3S, Elias Instruments, Copenhagen, Denmark) was inserted 10–15 cm into the rectum. Then the subjects rested in bed until the exercise period started. On two separate days the subjects exercised at a constant pedaling rate of 75 rpm for 80 min at 75% of maximal \( \text{O}_2 \) uptake (Table 1). They were allowed a 5 min rest period once every 20th min. After exercise the subjects rested in bed for 7 h. Two control experiments without exercise were also performed and the conditions were identical to the resting period of the exercise experiments. The start of exercise was adjusted so that the recovery period always started at 9:30 A.M. After exercise the subjects rested in bed until 4:30 P.M. but were not allowed to sleep during this period.

In one of the exercise experiments (food exercise) and one of the control experiments (food control) the subjects were given a meal consisting of bread (3 g/kg body wt), jam (2 g/kg body wt), and skim milk (0.5 liters). The average caloric content per meal was 4.5 MJ (5% fat, 81% carbohydrate, 14% protein). The meal was taken 2 h after exercise. All meals were completed within 30 min. In the other exercise (fasting exercise) and control experiment (fasting control) the subjects fasted for the entire recovery period.

**Measurements.** In each experiment the fasting baseline \( \text{O}_2 \) uptake was measured for 15 min in the morning before exercise after 30 min of bed rest. The fasting baseline measurement of \( \text{O}_2 \) uptake was repeated on nine separate occasions for each subject, and the coefficient of variation was <6%. During exercise \( \text{O}_2 \) uptake was measured from 15 to 18, 35 to 38, 55 to 58, and 75 to 78 min. Postexercise \( \text{O}_2 \) uptake was measured continuously for the 1st h and thereafter for the last 15 min of every hour for the next 7 h. In the control experiments \( \text{O}_2 \) uptake was measured for 15 min of every hour for the entire experiment. Rectal temperature was recorded continuously during the whole experimental period.

The subjects breathed through a mouthpiece, and expired air was collected in Douglas bags. Volume was measured in a wet spirometer. The bags and spirometer were checked for leaks. Fractions of \( \text{O}_2 \) and \( \text{CO}_2 \) were determined on an automatic system (\( \text{O}_2 \), s3A/I, Ametek, Pittsburgh, PA; \( \text{CO}_2 \), \( \text{CO}_2 \)-analyser, Simrad Optronics, Oslo, Norway). The gas analyzers were calibrated against gases of known \( \text{O}_2 \) and \( \text{CO}_2 \) concentrations before every bag was emptied. All gas volumes were expressed as standard temperature and pressure dry.

EPOC was calculated as the time integral of the difference in \( \text{O}_2 \) uptake between the exercise and control experiments for the postexercise period. Total energy expenditure (EE), which includes the contribution made by protein oxidation, was calculated from \( \text{O}_2 \) uptake (\( \text{VO}_2 \), ml/min) and respiratory exchange ratio (R) according to the formula (12)

\[
\text{EE} = \text{VO}_2 \times (15.480 + 5.550 \times \text{R})
\]

TEF was calculated as the time integral of the difference in energy expenditure between the postprandial and fasted state for the time period after the meal. The rate of fatty acid (FA) oxidation was estimated as described in detail previously (1), assuming that the rate of nitrogen excretion was 7.8 mg/min. This average value was taken from the measured values determined in a similar study on recovery from exercise from our laboratory, where it was shown that there is no change in the rate of nitrogen excretion after exercise compared with a control experiment (1). A 20% error in this assumed value (which exceeds the total range of values in the previous study) would have had no significant effect on the estimated rate of FA oxidation. In estimating the rate of FA oxidation it was assumed that dioleoylpalmitoyl-glycerol (33,976 kJ/mol) represents triglycerides from human fat stores.

**Statistical methods.** The results are presented as means ± SE. The experiment was divided into three intervals for statistical analysis. The first interval (−2 to 2 h) consists of the observations during rest before the meal. The second interval lasts from the end of the exercise period until the end of the experiment (0–7 h), and this interval provides information on the effects of exercise on recovery metabolism. The data obtained in the third interval (2–7 h postexercise, 0–5 h postprandially) provides information on the effects of food intake and the prolonged EPOC component. The data obtained in the different time intervals were compared using analysis of variance for repeated measures using \( t \) tests. Calculated EPOC, TEF, and FA oxidation (time integrals) were compared using a paired \( t \) test. Rejection level was chosen as \( P ≤ 0.05 \).

**RESULTS**

\( \text{O}_2 \) **uptake.** In the control experiments the value for \( \text{O}_2 \) uptake was 238 ± 14 (fasting control) and 248 ± 14 ml/
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In the exercise experiments, O$_2$ uptake increased to 2.79 ± 0.21 (fasting exercise) and 2.86 ± 0.19 l/min (food exercise, NS vs. fasting exercise) at the end of the 80-min work periods. After exercise O$_2$ uptake fell sharply, but postexercise O$_2$ uptake was higher than control values in both the food and fasting exercise experiments (0–7 h; food exercise vs. food control: $F_{50.10} = 71.2$, $P < 0.0005$; fasting exercise vs. fasting control: $F_{50.10} = 86.7$, $P < 0.0005$; Fig. 1).

There was a significant increase in O$_2$ uptake after meals to a peak value of 308 ± 13 (food control, $P < 0.001$ vs. fasting control) and to 343 ± 17 ml/min (food exercise, $P < 0.001$ vs. fasting exercise; Fig. 1). In the time period after meals, O$_2$ uptake remained higher than in the fasting experiments for the whole observation period (2–7 h; food exercise vs. fasting exercise: $F_{50.4} = 7.32$, $P < 0.0025$; food control vs. fasting control: $F_{50.4} = 6.41$, $P < 0.0025$).

**EPOC and TEF.** The total O$_2$ consumption over the 7-h recovery period after exercise and for the corresponding time period of the control experiments is shown in Table 2. EPOC amounted to ~21 liters over the 7-h period whether or not food was given. The time course of accumulated EPOC is shown in Fig. 2. Postexercise O$_2$ consumption was significantly higher than in the resting condition throughout the periods before (0–2 h) and after the meal (2–7 h). Hence, the prolonged component of EPOC (2–7 h) amounted to 11.8 ± 3.5 liters in the fasted state and 10.4 ± 2.5 liters in the fed state.

TEF amounted to ~13 liters (corresponding to ~300 kJ) in both the exercise and control conditions. The time course of TEF is shown in Fig. 3. Because EPOC was of similar magnitude whether the subjects were rested or had exercised, TEF was also not different between the food and fasting experiments (Table 2).

Table 2 shows that no interaction between food and exercise could be detected. However, the design of the study does not allow us to conclude that such an interaction does not exist. Considering the SE of 3.9 liters, the test power (1 − β) falls below 0.8 at an interaction of ~9.5 liters. We therefore conclude that both major effects, EPOC and TEF, are not significantly changed by the interventions. Increased TEF cannot account for EPOC but, on the other hand, we cannot exclude the possibility that it contributes because the design only allowed detection of major interaction effects.

R increased from 0.78 ± 0.02 (mean morning value in the control experiments) to 0.90 ± 0.02 at the end of the exercise periods (Fig. 4). After exercise R fell sharply below the level of the control experiments, and there was a significant reduction in R in the postexercise interval in the food and fasting experiments (0–7 h; food exercise vs. food control: $F_{50.10} = 5.84$, $P < 0.0005$; fasting exercise vs. fasting control: $F_{50.10} = 5.32$, $P < 0.0005$) (Fig. 4). However, we detected no effect on R in response to meals compared with the corresponding fasting experiments.

**Table 2.** Total O$_2$ consumption (0–7 h) during rest and recovery after exercise in the four experimental conditions

<table>
<thead>
<tr>
<th></th>
<th>Exercise</th>
<th>Control</th>
<th>Difference (EPOC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Food</td>
<td>136.7±5.7</td>
<td>115.6±4.8</td>
<td>21.1±3.6*</td>
</tr>
<tr>
<td>Fasting</td>
<td>128.4±5.5</td>
<td>102.6±5.7</td>
<td>25.8±4.5*</td>
</tr>
<tr>
<td>Difference (TEF)</td>
<td>13.3±6.6*</td>
<td>13.0±1.9*</td>
<td>0.2±3.9†</td>
</tr>
</tbody>
</table>

Values are means ± SE in liters; $n = 6$. EPOC, excess postexercise O$_2$ consumption; TEF, thermic effect of food. * $P < 0.05$; † NS.

**Fig. 3.** Time plot of accumulated excess postexercise O$_2$ uptake (0–7 h). Bars, SE.
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FIG. 4. Time plot of mean respiratory exchange ratio (R; n = 6). Bars, SE.

(2–7 h; food control vs. fasting control: F₀₉₄ = 2.05, P = 0.13; food exercise vs. fasting exercise: F₀₉₄ = 2.84, P = 0.052).

Rate of FA oxidation. The rate of FA oxidation (2–7 h postexercise) was 45 ± 5.9 mmol/5 h in the food control experiments and increased after exercise to 92 ± 8.1 mmol/5 h (food exercise) (P < 0.01). In the fasting control experiments the rate of FA oxidation (2–7 h postexercise) was 88 ± 7.1 mmol/5 h, and it increased to 114 ± 6.5 mmol/5 h after exercise (fasting exercise): (P < 0.025).

Rectal temperature. Rectal temperature increased to 38.8 ± 0.11 (food, P < 0.001 vs. control) and 38.6 ± 0.09°C (fasting, P < 0.001 vs. control, NS vs. food) at the end of the exercise period. The temperature decreased rapidly and reached control values in <1 h on both exercise days. Thereafter, no significant differences were observed between the postexercise and control experiments or between food and control experiments.

**DISCUSSION**

The main finding of this study is that the prolonged component of EPOC is present in the fasting state. Furthermore, we cannot detect any major interaction effects between food intake and exercise on the postexercise O₂ consumption.

**Methodological considerations.** It is well known that there are considerable intra- and interindividual variations in the thermogenic effect of a standard meal and that the measurement of the prolonged EPOC component is subject to variations as well (2, 32). This is also evident in the present study, and our ability to detect small differences in EPOC or TEF as a result of food intake or exercise is limited.

Several attempts have been made to study the possible synergistic interactions between exercise and food intake on total energy expenditure, and conflicting conclusions have been reached (10, 19, 20, 23, 27). Several studies have reported an enhanced thermic effect of a meal followed by exercise (9, 21, 22, 35), whereas other studies have failed to document any interaction of food and exercise on energy expenditure (11, 24, 25, 29, 31).

This has been attributed to three flaws in experimental designs (20): 1) that the intensity and duration of exercise was insufficient, 2) that the meal size was too small, or 2) that highly trained individuals who have developed energy-sparing mechanisms were used as subjects. Thus the present study was planned in an attempt to avoid these objections. First, the subjects undertook a nearly exhaustive exercise protocol (80 min of cycling at 75% of maximal O₂ uptake). Second, the meals given (4.5 MJ) were above the size believed to be critical to achieve an enhanced postprandial thermogenesis (~4.2 MJ) (25). The size of meals was adjusted to the body mass of the subjects, and this may have caused some variability in TEF between subjects. However, because the subjects were given identical meals in the control and exercise experiments, the effect of exercise on TEF would not be affected by this variability. Finally, the fitness level of the subjects was below average for young Norwegian males, as indicated by their maximal O₂ uptake (Table 1) (14).

**Mechanisms for the prolonged EPOC component.** The mechanisms causing a prolonged increase in energy expenditure after exercise are not completely understood (2, 13, 15), but it appears that other tissues as well as muscle must be involved because whole body EPOC is much greater than can be accounted for by local muscle events (5). Because the prolonged EPOC component was present in the fasting experiments, it is clear that food intake is not a prerequisite for the elevation of postexercise metabolic rate.

The classic O₂ debt, or rapid EPOC component (<1 h postexercise), is believed to be caused by replenishment of O₂ stores in blood and muscle, resynthesis of ATP and creatine phosphate, lactate removal, increased ventilation, a higher heart rate, and increased core temperature (13). However, these processes are believed to subside within 1 h after exercise, and therefore their contribution to the prolonged EPOC component: can be excluded (3, 4). Some other processes have been suggested for the prolonged EPOC component: increased rates of triglyceride-FA cycling, a potentiation of TEF, and the energy cost of increased glycogen resynthesis in muscle and liver (4).

In the present study the prolonged EPOC component was present whether or not food was given. It is well established that the rate of glycogen resynthesis is low when no exogenous carbohydrate is supplied (6, 16, 17), as was the case in the present fasting experiments. Biopsies were not taken to determine the rate of glycogen resynthesis in the present study. However, use of an exercise protocol nearly identical to that described here has shown that the rate of glycogen storage is 1.5–2.5 μmol·g wet wt⁻¹·h⁻¹ in the fasted state compared with 6–8 μmol·g wet wt⁻¹·h⁻¹ when carbohydrates are provided in sufficient amounts (8, 16, 17). The theoretical O₂ cost of storing muscle glycogen at these rates can be estimated to 0.2 l/h in the fasted state and 0.8 l/h in the fed state, a total estimated difference of ~3 liters for the 5-h period after the meal compared with an EPOC of 10–12 liters. Thus the theoretical contribution from the O₂ cost of increased rates of glycogen resynthesis is about one-fourth of the prolonged EPOC component. It remains an interesting possibility that other energy-requiring pro-
cesses are stimulated and contribute relatively more to EPOC when the rate of glycogen resynthesis is low. This hypothesis requires a different experimental approach.

Because the prolonged EPOC component was present in the fasted state it may be concluded that this component is not caused by a potentiation of TEF. Also, no interaction between food and exercise could be detected (Table 2). However, the design of the study does not allow us to detect an effect on TEF of less than ~9 liters. We therefore cannot exclude the possibility of a small but undetected potentiation of TEF contributing to EPOC in the fed state.

It should be noted that fat metabolism may be of importance for EPOC through the energy cost of increased triglyceride-FA substrate cycling after exercise (1, 33). In the present study R was significantly reduced after exercise compared with the control experiment in the absorptive state, as observed in previous studies (2, 18). Because there appears to be a weak relationship between the rate of triglyceride-FA cycling and a reduction in R after exercise (1), it is possible that the observed substrate shift is accompanied by an increased rate of cycling. Substrate cycles of potential importance during recovery from exercise also exist in the processes of glycolysis and gluconeogenesis. There may be an increase in the Cori cycle after exercise, i.e., an increase in the rate of conversion of ingested glucose to lactate within muscle and the subsequent recycling in the liver. It has recently been shown that there is no increase in the rate of the glucose-glucose-6-phosphate cycle after exercise (30).

Interaction between food and exercise on resting metabolic rate. There is some evidence for such an effect in the literature. Most studies have examined the effect of a meal given before or during exercise with the TEF at rest. In the present study a different experimental approach was used, because we have compared the thermic effect of a meal given at rest after exercise with that of a meal given in a control study. This approach was inspired by two recent studies that suggested that a potentiation of TEF may be a possible mechanism for the prolonged EPOC observed after strenuous exercise (2, 18). In these studies a large portion of EPOC was observed after meals given 2 and 7 h postexercise. Similar observations have been made by Bielinski et al. (7), Young et al. (34), and Treadway and Young (28), who observed an increase in the thermic effect of a meal ingested during recovery from exercise. In these studies postexercise O2 uptake was close to control levels before the meal was ingested. It may be argued that because fasting experiments were not done and there may still have been a small undetected increase in O2 uptake due to exercise before meal ingestion, it is not possible to separate EPOC from TEF.

A study design similar to that described here with fasting experiments was used by Segal et al. (22), who studied postexercise oxygen expenditure in the fed vs. the unfed state and concluded that TEF was the same whether the subjects performed light exercise for 30 min immediately before or after ingestion of the 3.2-MJ test meal. It appears that there is no detectable effect on TEF whether the subjects are resting or performing light or strenuous exercise. However, in making this conclusion, there is a possibility of making a type 2 error, and differently designed studies could still reveal a small effect. We therefore conclude that the prolonged EPOC component is present without any contribution from TEF.

Conclusion. The present study shows the presence of a prolonged EPOC component in fasted subjects. The prolonged component observed in the fasted state must be caused by processes other than increased rates of glycogen resynthesis or a potentiation of TEF.

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