Effect of exercise intensity and duration on postexercise metabolism

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GORE, C. J., AND R. T. WITHERS. Effect of exercise intensity and duration on postexercise metabolism. J. Appl. Physiol. 68(6): 2362-2368, 1990.—Data are reported on the net recovery O_2 consumption (VO_2) for nine male subjects (mean age 21.9 yr, VO_2max 63.0 ml·kg\(^{-1}\)·min\(^{-1}\), body fat 10.6%) used in a 3 (independent variables: intensities of 30, 50, and 70% VO_2max) × 3 (independent variables: durations of 20, 50, and 80 min) repeated measures design (P < 0.05). The 8-h mean excess postexercise O_2 consumption (EPOCs) for the 20-, 50-, and 80-min bouts, respectively, were 20.48, 53.20, and 84.23 liters at 30% VO_2 max (6.8 km/h); 3.14, 5.19, and 6.10 liters at 50% VO_2 max (9.5 km/h); and 5.68, 10.04, and 14.59 liters at 70% VO_2 max (13.4 km/h). The mean net total O_2 costs (NTOC = net exercise VO_2 + EPOC) for the 20-, 50-, and 80-min bouts, respectively, were 20.48, 53.20, and 84.23 liters at 30% VO_2max; 38.95, 100.46, and 160.59 liters at 50% VO_2max; and 58.30, 147.48, and 237.17 liters at 70% VO_2max. The nine EPOCs ranged only from 1.0 to 8.9% of the NTOC (mean 4.8%) of the exercise. These data, therefore, indicate that in well-trained subjects the 8-h EPOC per se comprises a very small percentage of the NTOC of exercise.

<table>
<thead>
<tr>
<th>Intensity (% VO_2max)</th>
<th>Duration (min)</th>
<th>20-min EPOC</th>
<th>50-min EPOC</th>
<th>80-min EPOC</th>
</tr>
</thead>
<tbody>
<tr>
<td>30</td>
<td>20</td>
<td>20.48</td>
<td>53.20</td>
<td>84.23</td>
</tr>
<tr>
<td>50</td>
<td>5.68</td>
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</tr>
<tr>
<td>70</td>
<td>1.01</td>
<td>1.43</td>
<td>1.04</td>
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</tbody>
</table>

excess postexercise oxygen consumption; rectal temperature; respiratory exchange ratio; heart rate

On cessation of activity, O_2 consumption (VO_2) declines exponentially toward the resting baseline level. This postexercise VO_2, which is additional to that required to support resting metabolic processes, was formerly known as the O_2 debt (14) but has more recently (9) been called the excess postexercise O_2 consumption (EPOC). It is frequently stated in the lay press that this EPOC is a substantial percentage of the net energy expenditure of exercise, which would therefore be underestimated if, as is normally done, the duration of exercise is multiplied by the "steady-state" VO_2 minus VO_2 for the resting metabolic rate (RMR). This reasoning is therefore used to enhance the attractiveness of exercise as a weight-reduction modality.

The first report of an elevated resting metabolism after physical activity was published by Benedict and Carpenter in 1910 (3). They noted mean increases of 7.7 and 14.5% 7-13 h after severe work for two subjects sleeping in a respiration calorimeter. Another subject registered a mean increase of 23.2% over a period of 1 4 h after very severe work. Herxheimer et al. (13) subsequently noted that the VO_2 of five untrained subjects did not return to base line until 36-48 h after exercise, and Edwards et al. (7) lent further support to this hypothesis by reporting a 25% increase in metabolism 15 h after cessation of activity. These two studies are frequently quoted to support the concept that the net energy cost of exercise is underestimated because no allowance is made for the sustained increase in postexercise metabolism. However, the validity of many of the early studies can be questioned because there was a general nescience that RMR is affected by such variables as time of day, temperature, food and caffeine intake, prior uncontrolled exercise, habituation, and stress. Furthermore, nobody attempted to determine the magnitude of the EPOC. Also, in many cases the intensity and duration of the exercise were not quantified. Nevertheless, better-designed studies have been conducted recently in an attempt to elucidate the nature of the EPOC. Bahr et al. (1) indicated that the 12-h EPOCs after a cycle ergometer was pedaled at 70% of maximum VO_2 (VO_2max) for 20, 40, and 80 min comprised 19.8, 12.2, and 13.8%, respectively, of the exercise VO_2. Other studies (4, 17, 18) demonstrated no prolonged thermogenic effect from less-intensive activity (30-55% VO_2max). There may therefore be a critical threshold for the intensity and/or duration of exercise before the EPOC comprises a physiologically significant component of the total energy expenditure. Knowledge of such a threshold would be of great assistance to those persons who wish to maximize exercise energy expenditure with a view to weight loss. The aim of this study was therefore to determine the magnitudes of the EPOCs after treadmill exercise bouts of 20-, 50-, and 80-min duration at intensities of 30, 50, and 70% VO_2max.

METHODS

Subjects. Nine males (mean values ± SD: age 21.9 ± 2.2 yr, height 179.2 ± 7.0 cm, mass 69.56 ± 4.36 kg, VO_2max 63.0 ± 5.7 ml·kg\(^{-1}\)·min\(^{-1}\), body fat 10.6 ± 3.1%) participated in the study. The weekly training loads of the subjects over the 3 mo before the start of the experiments are presented in Table 1. All the subjects were lifetime nonsmokers. Informed consent was obtained in accordance with the established protocol for human subjects.

Densitometry. The subject controls and methodology for measuring body density by underwater weighing have been described previously (25). The Siri equation (23)
was used to transform the body density to percent body fat.

**Determination of VO₂max and treatment work loads.** All VO₂ measurements were conducted by use of the indirect calorimetry system described by Sainsbury et al. (21). The Beckman LB-2 CO₂ analyzer and Electrochemistry S 3A O₂ analyzer were calibrated every hour with gases that had been authenticated by Lloyd-Haldane analyses. Although the Morgan Mark 2 turbine volume transducer was likewise checked hourly by use of a 1 liter syringe in accordance with the manufacturer’s instructions, it had been previously calibrated throughout a range spanning rest to maximum exercise with sinusoidal artificial lungs that were fabricated in our medical school’s Biomedical Engineering Department. The system was also checked daily for leaks.

The subjects visited the laboratory on a day before the VO₂max test to be familiarized with running on the treadmill, operating the emergency stop lever, and breathing through the Hans Rudolph R2700 respiratory T valve while a noseclip was attached. A 3-min warmup at 7.5 km/h and 0% grade was followed by a treadmill speed of either 12 or 15 km/h with the grade increased by 2%/min until the subject was too fatigued to continue. VO₂max was considered to occur when the VO₂ for successive increments differed by <2 ml·kg⁻¹·min⁻¹. The treadmill speeds to elicit 30, 50, and 70% VO₂max at 0% grade were determined individually on a separate day. Serial VO₂ measurements ensured that the steady state had been attained for the selected speeds.

**Heart rate.** This variable was monitored continuously during all VO₂ measurements by a B-D Electrodyne electrocardiogram by use of a CM-5 electrode placement.

**Body temperature.** Except for the VO₂max tests, the core and skin temperatures were monitored continuously during rest and exercise by customized equipment, which has been described elsewhere (10). The mean skin temperature was estimated by weighting observations from the chest, arm, thigh, and calf (19). This facilitated the estimation of the mean body temperature by the formula proposed by Burton (5).

**Experimental design.** All subjects were familiarized with the laboratory, the personnel, and the equipment by having their RMRs recorded on two separate days before any experimental data were collected. They were requested to adhere to the following before the nine experimental and two control conditions to control for those factors known to affect the RMR: 1) engage in no exercise during the preceding 36 h; 2) ingest no caffeine, alcohol, or drugs during the preceding 24 h; 3) consume a standardized preweighed meal before 2000 h on the day before a test; water only was drunk thereafter until a standardized preweighed light breakfast was consumed 3–5 h before any measurements were taken; 4) eliminate uncontrolled activity by traveling to the laboratory by car or motorcycle; and 5) report to the laboratory at 0630 h. After 1 h of bed rest, each subject performed one of the nine experimental treatments on the treadmill (30% VO₂max; 20, 50, and 80 min; 50% VO₂max; 20, 50, and 80 min; 70% VO₂max; 20, 50, and 80 min), which were counterbalanced to eliminate any order effect. The subjects then rested in bed for 8 h and ate a standardized 4,900-kJ meal (63% carbohydrate, 23% fat, 14% protein) 3 h postexercise. A 24-h postexercise measurement was also taken, but the subjects were not detained in the laboratory during the intervening period. However, they were instructed to refrain from exercise and consume standardized meals at specified times. Two control conditions of no exercise followed by 8 h of bed rest were conducted before and after the nine experimental treatments for each subject, and the mean was used as the base line. The laboratory temperature in the vicinity of the subject was maintained at 24.0 ± 0.5°C while all the preceding measurements were taken. The subjects were always covered with a blanket when not exercising.

**Computation of the O₂ deficit, net total O₂ cost (NTOC) of exercise, and EPOC.** The trapezoidal rule was used to approximate the integral for the exercise, 8-h postexercise, and control VO₂ values over time. This facilitated the computation of NTOC (exercise VO₂ + 8-h postexercise VO₂ − exercise and postexercise control VO₂) and 8-h EPOC (8-h postexercise VO₂ − 8-h control VO₂).

**Statistical analyses.** The EPOC data were analyzed by a 3 (intensities of 30, 50, and 70% VO₂max) × 3 (durations of 20, 50, and 80 min) factorial design analysis of variance (ANOVA) with repeated measurements across both dimensions. The 0.05 probability level was used when the statistical significance of the two main effects and their interaction were tested. Tukey’s post hoc tests (P < 0.05) were used in the event of any significant F ratios. Checks were undertaken for violations of the assumptions underlying all analyses by Bartlett’s test of sphericity. Dependent t tests (P < 0.05) were used to locate statistically significant differences between the experimental and control days for VO₂, heart rate (HR), rectal temperature (T<sub>r</sub>), and average body temperature (T<sub>b</sub>).

### RESULTS

**Postexercise VO₂.** Figures 1–3 indicate that the 1-h postexercise VO₂ was not significantly different from the mean control value (269 ml/min) for all the 20-min treatments (264, 267, and 276 ml/min), the 50-min bouts at 30 (261 ml/min) and 50% VO₂max (276 ml/min), and the 80-min walk at 30% VO₂max (266 ml/min). The postexercise VO₂ for the 80 min at 50% VO₂max (273 ml/min), 50 min at 70% VO₂max (335 ml/min), and 80 min at 70% VO₂max (299 ml/min) treatments all decreased to the points where they were not significantly different from the time-matched controls after 2 (268 ml/min), 4

### TABLE 1. Weekly training loads

<table>
<thead>
<tr>
<th>Subject</th>
<th>Running</th>
<th>Cycling</th>
<th>Swimming</th>
<th>Other</th>
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<tbody>
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<tr>
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<td>5</td>
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<tr>
<td>JA</td>
<td>110</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>MW</td>
<td>5</td>
<td></td>
<td>3 km interval running (sprints)</td>
<td></td>
</tr>
<tr>
<td>MV</td>
<td>20</td>
<td>3 h squatch</td>
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<td></td>
</tr>
<tr>
<td>BM</td>
<td>50</td>
<td>12 km ski paddling</td>
<td></td>
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</tr>
<tr>
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</tr>
<tr>
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<tr>
<td>PH</td>
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</table>
FIG. 1. Mean postexercise $\dot{V}O_2$ after 20, 50, and 80 min of treadmill exercise at 30% $V_{O2max}$. No control measurements were made at 0.25, 0.50, and 0.75 h postexercise, so dependent $t$ tests could not be conducted for these times. $n = 9$ subjects. $^*P < 0.05$ between treatment and control means.

(326 ml/min), and 8 h (295 ml/min), respectively.

$EPOC$. The 8-h EPOC data are graphed in Fig. 4. The factorial design ANOVA for the EPOCs yielded $F$ ratios for intensity, duration, and interaction that were all significant beyond the 0.0005 level. Tukey’s post hoc tests of the interaction showed no significant differences between the EPOCs for the 20-, 50-, and 80-min walks at 30% $V_{O2max}$ but that, above this intensity, the EPOC increased significantly with duration. However, the increase (2.96 liters) in EPOC after 80 min of running at 50% $V_{O2max}$ compared with 20 min at 50% $V_{O2max}$ was much smaller than that (8.91 liters) after running at 70% $V_{O2max}$ for the same durations. This trend is emphasized by Fig. 4. Although intensity accounted for 45.5% of the systematic variance of the EPOC, duration and the interaction between intensity and duration accounted for only 8.9 and 7.7%, respectively.

Three of the four largest EPOCs were associated with the 70% $V_{O2max}$ treatments, and these data have been graphed in Fig. 3. The mean $V_O2$ was 3,135 ml/min (~64 kJ/min) at the end of the run compared with a control value of 274 ml/min (~5.6 kJ/min). Thus this intensity of exercise caused an 11.4-fold increase in metabolism.

The exercise bouts that corresponded to a short walk (20 min at 30% $V_{O2max}$; 6.8 km/h), a recreational jog (50 min at 50% $V_{O2max}$; 9.5 km/h), and a training run (80 min at 70% $V_{O2max}$; 13.4 km/h) were chosen for Table 2, which shows the separate energy equivalents of the EPOC and NTOC.

$HR$. The recovery HRs for the three 30% $V_{O2max}$ treatments together with the 20- and 50-min bouts at 50% $V_{O2max}$ were not significantly different from the
treatments. Table 3 indicates that $T_b$, had recovered such the control value as from 2 h postexercise for all nine treatments. Table took much longer than that from the other bouts in Table 3 indicate that recovery from the 80-min run. The HRs for the 70% $V_o_2$max at 50% $V_o_2$max from the control value of 56 beats/min at 1 h postexercise, whereas that for the 80-min run at 50% $V_o_2$max was not different (P = 0.54) from the control value until 3 h after completion of the run. The HRs for the 70% $V_o_2$max exercise bouts in Table 3 indicate that recovery from the 80-min treatment took much longer than that from the other two treatments.

$T_b$. $T_b$ was generally not significantly different from the control value as from 2 h postexercise for all nine treatments. Table 3 indicates that $T_b$ had recovered such that it was not significantly different from the control values at 2, 6, and 7 h postexercise for the 20, 50, and 80 min at 70% $V_o_2$max treatments, respectively.

**DISSCUSSION**

The $V_o_2$ data for the two control conditions are presented in Fig. 5. A 4,900-kJ meal increased $V_o_2$ to a maximum of ~23%, which occurred 1 h after ingestion. This thermic effect of food (TEF) is caused by the energy requirements of digestion, absorption, transport, and storage. TEF appears to be similar for the nine treatments and the control, but it is only possible to precisely measure the TEF per se against base lines of exercise-no food and rest-no food. The three largest deviations of the two control days about the mean control were ± 2.46, 2.40, and 1.88%, respectively, whereas the average for all the differences during the two 9.53-h control days was ± 1.19%. We made 11 temporally matched measurements of each subject's RMR, i.e., two control and nine at 24 h postexercise. Because the $V_o_2$ data revealed no significant 24-h postexercise elevation for the nine treatments, coefficients of variation have been calculated for each subject by use of 11 scores. These ranged from 3.2 to 4.6% [mean 4.1 ± 0.5% (SD)]. Furthermore the correlations between control 1 and control 2 at the time equivalents of 0 and 8 h postexercise were 0.920 (SEE = 7.7 ml/min, mean1 272 ml/min, mean2 276 ml/min) and 0.990 (SEE = 2.5 ml/min, mean1 295 ml/min, mean2 295 ml/min), respectively. The preceding analyses are measures of the reliability of our indirect calorimetry system and represent combinations of both within-subject biological variation and methodological error.

It is appropriate to relate our findings solely to those studies that not only standardized the protocol for the measurement of RMR but also accounted for its circadian variation. Pacy et al. (17) exercised four subjects on a cycle ergometer for 20 min at 35-55% $V_o_2$max during each of four consecutive hours and concluded that there was no prolonged thermogenic effect of moderate repeated aerobic exercise because 60 min after exercise the $V_o_2$ was not significantly different from the control value. This group (18) reached similar conclusions when the 24-h RMR was measured by direct calorimetry in three subjects who exercised on a cycle ergometer for 1 h on two occasions at 30% $V_o_2$max. However, although the difference between the exercise and control means was not statistically significant, the EPOC for one of the subjects was equivalent to ~60 liters. Bielinski et al. (4) exercised 10 subjects (mean $V_o_2$max = 62.5 ml·kg⁻¹·min⁻¹) on a treadmill for 3 h at 50% $V_o_2$max, but the postexercise $V_o_2$ ceased to be significantly different from the control values as from 4 h postexercise; the average increase in metabolism up to that time was 9%. The preceding studies are in accordance with our findings because, of the six 30 and 50% $V_o_2$max treatments, only the 1-h postexercise $V_o_2$ for the 80 min at 50% $V_o_2$max exercise bout was significantly greater than its time-matched control value (284 vs. 269 ml/min), and it (273 ml/min) had returned (P > 0.05) to base line (268 ml/min) by 1 h later. Therefore, there does not appear to be any convincing evidence for a sustained elevation in

![Graph](image-url)
TABLE 3. Mean values for physiological variables during control conditions and recovery from the three 70% $V_{O_2 \text{max}}$ treatments

<table>
<thead>
<tr>
<th>Condition</th>
<th>0</th>
<th>0.5</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>24</th>
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<tbody>
<tr>
<td>$R$</td>
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<td></td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>Control</td>
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<td>0.823</td>
<td>0.827</td>
<td>0.822</td>
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<td>0.924*</td>
<td>0.826</td>
<td>0.807</td>
<td>0.780*</td>
<td>0.779*</td>
<td>0.846</td>
<td>0.849</td>
<td>0.862*</td>
<td>0.861</td>
<td>0.835*</td>
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<tr>
<td>50 min</td>
<td>0.910*</td>
<td>0.817</td>
<td>0.819</td>
<td>0.773*</td>
<td>0.769*</td>
<td>0.835*</td>
<td>0.836*</td>
<td>0.835*</td>
<td>0.837*</td>
<td>0.818*</td>
<td>0.808</td>
</tr>
<tr>
<td>80 min</td>
<td>0.902*</td>
<td>0.794</td>
<td>0.809</td>
<td>0.779*</td>
<td>0.796</td>
<td>0.816*</td>
<td>0.815*</td>
<td>0.819*</td>
<td>0.816*</td>
<td>0.810*</td>
<td>0.839</td>
</tr>
</tbody>
</table>

| Control values for 24 h postexercise are identical to those for 0 h. There are no control values for 0.5 h postexercise. * P ≤ 0.05 from control.

FIG. 5. Mean $V_{O_2}$ data for 2 control conditions. n = 9 subjects.

Some well-controlled Scandinavian studies measured the magnitude of the EPOC, thereby facilitating direct comparison with our data. Hermansen et al. (12) initially demonstrated a 12-h EPOC of 48.0 liters in one subject who pedaled a cycle ergometer for 80 min at 75% $V_{O_2 \text{max}}$. $V_{O_2}$ was also still elevated by 5.9% (graph interpolation) 24 h afterward. A subsequent study by Maehlum et al. (16) showed a smaller mean 12-h EPOC of 26 liters in eight subjects (mean $V_{O_2 \text{max}}$ 3.30 l/min or 47.3 ml·kg⁻¹·min⁻¹) who also pedaled a cycle ergometer for 80 min but at 70% $V_{O_2 \text{max}}$. This is much higher than our corresponding value of 14.59 liters reported in Table 2; the authors furthermore stated that the 24-h postexercise $V_{O_2}$ was significantly ($P < 0.05$) higher than the control value. Bahr et al. (1) recently investigated the effect of duration on the EPOC by having six subjects (mean $V_{O_2 \text{max}}$ 4.37 l/min or 54.1 ml·kg⁻¹·min⁻¹) cycle at 70% $V_{O_2 \text{max}}$ for 20, 40, and 80 min. They concur with our finding that the metabolism is not significantly ($P > 0.05$) elevated 24 h after exercise, but their 12-h EPOCs of 11.1, 14.7, and 31.9 liters, which like ours at this intensity increased linearly with exercise duration, are approximately twice as large as those reported for the present investigation. Even if we had continued to measure the EPOC for a further 4 h to make the times comparable, the extra $V_{O_2}$ would have been minimal because the postexercise metabolisms for the 70% $V_{O_2 \text{max}}$ treatments were not significantly different from the control values after 1 h (276 vs. 269 ml/min), 4 h (335 vs. 328 ml/min), and 8 h (299 vs. 295 ml/min) for the 20-, 50-, and 80-min bouts, respectively. It may be that the EPOC after bicycle ergometer exercise is much greater than that after treadmill runs at the same relative intensity and/or that recovery is slower with less well-trained subjects.

Table 2 contains data for the three treatments that represent a short walk (20 min at 30% $V_{O_2 \text{max}}$), a recreational jog (50 min at 50% $V_{O_2 \text{max}}$), and a training run (80 min at 70% $V_{O_2 \text{max}}$). Calculation of the contribution of the EPOC to weight loss has the obvious limitation of ignoring the reduction in the energy cost of a given speed metabolism after exercise of intensities ≤55% $V_{O_2 \text{max}}$ and durations ≤3 h.
of locomotion as weight decreases. Nevertheless, Table 2 emphasizes the extremely small adipose tissue loss caused by the EPOC alone. It therefore appears that the postexercise increase in energy expenditure per se after exercise bouts spanning the range from 20 min at 30% \( \dot{V}O_{2\text{max}} \) to 80 min at 70% \( \dot{V}O_{2\text{max}} \), has a negligible role in weight loss. This is because the EPOC comprises a very small percentage of the NTOC of the exercise. However, it can be argued that our findings apply only to those of well above-average maximum aerobic power because such treatments might produce substantially larger EPOCs in less well-trained subjects. However, Table 2 presents more realistic values regarding the number of exercise bouts equivalent to the energy contained in 1 kg of adipose tissue and emphasizes the role exercise can have in any weight reduction program.

Table 3 shows the \( R \) values for the three 70% \( \dot{V}O_{2\text{max}} \) treatments. There is an inverse relationship between duration and \( R \) at the end of exercise that indicates a greater reliance on fat metabolism as the glycogen reserves are depleted. This trend is even more pronounced 4–8 h postexercise despite a 4,900-kJ meal. Here, the \( R \) values are lower than the controls and suggest that this diminished glucose utilization after exercise may allow more of the exogenous glucose, most of which evades hepatic retention (15), to be used for the repletion of muscle glycogen. It is possible to estimate (4) this carbohydrate storage (i.e., intake minus oxidation) on the basis that 2 mol of ATP are required per 1 mol of glucose transformed into glycogen. This is equivalent to 5.3% of the energy content of the glucose (8). Such calculations show that, compared with the control data over the 3- to 8-h postexercise period, there was an additional carbohydrate storage of ~19 g after the 80 min at 70% \( \dot{V}O_{2\text{max}} \) treatment. This accounted for ~21% (0.84 liters \( O_2 \)) of the 3- to 8-h EPOC of 3.92 liters. The energy requirements of glycogenesis, therefore, appear to comprise approximately one-fifth of the EPOC during this period when the RMR is still elevated.

Assuming a \( Q_10 \) effect of 2.6 (6) and using the van’t Hoff-Arrhenius equation, it was possible to compute the excess \( \dot{V}O_2 \) caused by temperature alone. These indicated that only 2–24% and 11–36% of the 8-h EPOCs could be attributed to the \( T_m \) and \( T_b \) increases, respectively. Nevertheless, these may be conservative estimates, because Saltin and Hermansen (22) demonstrated that the increase in temperature of the vastus lateralis muscle during exercise is greater than that in \( T_m \). There are also other methodological and theoretical limitations associated with the use of thermometry to estimate \( T_b \). These apply particularly during thermal transients.

Some of the EPOC can be attributed to the resaturation of hemoglobin and myoglobin together with the resynthesis of creatinine phosphate. However, these processes require only 0.5–2.0 liters \( O_2 \) (12). The postexercise tachycardia would also contribute to the EPOC because the heart is responsible for ~10% of the resting \( \dot{V}O_2 \) (11). According to Roussos and Campbell (20), the \( \dot{V}O_2 \) of the respiratory muscles at rest is ~0.25–0.5 ml min\(^{-1}\)·liter\(^{-1}\) ventilation, which is equivalent to just 1–2% of RMR. The percentage of the EPOC that could be attributed to increased minute ventilation is, therefore, small. In fact, minute ventilation was only significantly elevated relative to the matched control values during the 1st h of recovery subsequent to 80 min of running at 70% \( \dot{V}O_{2\text{max}} \), and this treatment yielded the largest EPOC of 14.59 liters. Minute ventilation differences between experimental and control conditions for this treatment were thereafter <0.5 l/min. Much of the early work was focused on the lactic acid component of the EPOC. Nevertheless, although postexercise blood lactates were not measured, it is very unlikely that the \( O_2 \) required for the oxidation of excess lactate to pyruvate after the 80 min at 70% \( \dot{V}O_{2\text{max}} \), treatment would exceed 2 liters.

Virtu (21) states that there is a decreased protein metabolism during exercise that is compensated by an augmented rate of protein turnover during the postexercise period. The resultant enhanced protein turnover would therefore contribute to the EPOC. Furthermore, some persons (1, 2, 12, 16) have speculated that part of the EPOC could be due to futile cycling, a mechanism that involves ATP turnover without net metabolic flux, but its importance to heat generation in humans remains unsubstantiated.

In conclusion, the elevated metabolism after such typical exercise loads as a 20-min walk at 6.8 km/h (30% \( \dot{V}O_{2\text{max}} \)), a 50-min jog at 9.5 km/h (50% \( \dot{V}O_{2\text{max}} \)), and an 80-min run at 13.4 km/h (70% \( \dot{V}O_{2\text{max}} \)) is of little physiological significance for weight loss unless the exercise is undertaken on a very regular basis, when the EPOC would have a cumulative effect. Moreover, extrapolating the results of the 80-min run at 70% \( \dot{V}O_{2\text{max}} \) to long-term weight loss in sedentary and/or overweight individuals is quite tenuous given their poor compliance with exercise programs. However, 79.1, 15.4, and 6.5, respectively, of the aforementioned exercise bouts would have to be performed before each of their cumulative net total energy expenditures is equivalent to the energy content of 1 kg of adipose tissue. This emphasizes the important role that exercise has in any weight-reduction program.

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