Comparison of energy expenditure elevations after submaximal and supramaximal running

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Laforgia, J., R. T. Withers, N. J. Shipp, and C. J. Gore. Comparison of energy expenditure elevations after submaximal and supramaximal running. J. Appl. Physiol. 82(2): 661–666, 1997.—Although exercise intensity has been identified as a major determinant of the excess postexercise oxygen consumption (EPOC), no studies have compared the EPOC after submaximal continuous running and supramaximal interval running. Eight male middle-distance runners [age = 21.1 ± 3.1 (SD) yr; mass = 67.8 ± 5.1 kg; maximal oxygen consumption (VO2max) = 69.2 ± 4.0 ml·kg−1·min−1] therefore completed two equated treatments of treadmill running (continuous running: 30 min at 70% VO2max; interval running: 20 × 1-min intervals at 105% VO2max with intervening 2-min rest periods) and a control session (no exercise) in a counterbalanced research design. The 9-h EPOC values were 6.9 ± 3.8 and 15.0 ± 3.3 liters (t-test: P = 0.001) for the submaximal and supramaximal treatments, respectively. These values represent 7.1 and 13.8% of the net total oxygen cost of both treatments. Notwithstanding the higher EPOC for supramaximal interval running compared with submaximal continuous running, the major contribution of both to weight loss is therefore via the energy expended during the actual exercise.

excess postexercise oxygen consumption; indirect calorimetry

THE ELEVATION in O2 consumption (VO2) above the resting level after exercise, which Gaesser and Brooks (15) have called the excess postexercise oxygen consumption (EPOC), was initially thought to contribute significantly to the energy cost of exercise (11, 21). This finding is often used to enhance the attractiveness of exercise as an integral component of weight-reduction programs. However, neither study quantified the intensity and duration of the exercise, and there is minimal information on the controls (24) that should have been observed when the baseline VO2 data were collected. Bahr and Maehlum (4), accordingly, concluded that the reported large sustained increases in the postexercise VO2 may be more an artifact of the experimental design than the exercise stimulus.

Although there have been improvements in the experimental design of more recent EPOC studies, only five (7, 14, 18, 25, 33) of them have examined the VO2 after quantified weight-bearing exercise by using experimental designs that accounted for the diurnal variation in resting metabolic rate (RMR). The study by Gore and Withers (17) is the most comprehensive because the treatments ranged from a 20-min walk at 30% maximal VO2 (VO2max: 6.8 km/h) to an 80-min training run at 70% VO2max (13.4 km/h). The maximal 8-h EPOC, which occurred after 80 min at 70% VO2max was 14.6 liters (−297 kJ). Withers et al. (33) also reported an 8-h EPOC of 32.4 liters (−594 kJ) after a 35-km road run [−70% VO2max, time = 164.1 ± 14.0 (SD) min], which is by far the most exhausting bout for any of the EPOC studies. They concluded that even after a 35-km run, which is well beyond the capacities of sedentary persons, the contribution of the postexercise increase in metabolism to weight loss is relatively minor when compared with the net energy expenditure during the run.

Bahr and Sejersted (6) have reported an exponential relationship between exercise intensity and the EPOC for prolonged exercise. Furthermore, Gore and Withers (17) demonstrated that exercise intensity was the major determinant of the EPOC because it explained five times more of the EPOC variance than either exercise duration or total work completed. This may be pertinent to athletes who perform supramaximal exercise (intensity > 100% VO2max) during interval training. Bahr et al. (2) measured the EPOC after supramaximal cycling. However, the treatment (three 2-min intervals of 108% VO2max administered to their untrained subjects) is well below the training loads of competitive cyclists. There is also scope for their experimental design to be extended to determine the effect of intensity per se by an attempt to match the total work performed during the high-intensity interval training with that accomplished during lower intensity continuous cycling. No studies were located that examined the EPOC associated with supramaximal running. The purpose of this research was therefore to examine the EPOC difference between submaximal continuous running (30 min at 70% VO2max) and supramaximal interval running (twenty 1-min runs at 105% VO2max with intervening 2-min recovery periods). On the basis of the work by Gore and Withers (17), it was hypothesized that the EPOC of supramaximal interval running would be greater than that for the matched work of submaximal continuous running. If the EPOC difference between these two types of training is physiologically significant, then this may have implications for the energy requirements of high-performance athletes (29) and the design of exercise protocols for weight loss.

METHODS

Subjects. Eight male middle-distance runners [age = 21.1 ± 3.1 (SD) yr; mass = 67.8 ± 5.1 kg; VO2max = 69.2 ± 4.0 ml·kg−1·min−1; height = 174.9 ± 5.3 cm; body fat = 9.1 ± 2.3%] participated in the study. Table 1 contains the average weekly training loads during the preceding 12 mo.

Hydrodensitometry. The subject controls and methodology for measuring body density (BD) by underwater weighing have been described previously (32). The Brožek et al. (9) equation (% body fat = 497.1/BD − 451.9) was used to estimate percent body fat from BD.

Determination of VO2max and treatment workloads. These measurements were conducted with the automated indirect calorimetry system (Deltatrac II; Datex-Engstrom, Helsinki).
calorimetry system described by Sainsbury et al. (27). The Beckman LB-2 CO2 analyzer (Anaheim, CA) and Ametek S-3A O2 analyzer (Pittsburgh, PA) were calibrated before testing and checked for drift at the end of the test by using three gases that had been authenticated by Lloyd-Haldane analyses. Inspired volume was measured by a P. K. Morgan MK2 turbine-volume transducer (Rainham, Kent, UK) that was calibrated before and after testing by using a 1-liter syringe in accordance with the manufacturer’s instructions. The accuracy of the turbine had previously been established throughout the range spanning light to maximum exercise (20). The system was checked daily for leaks. Before data collection, a VO2max-reliability trial (20) produced an intraclass correlation (ICC) of 0.98 and a coefficient of variation (CV) of 1.5%.

Subjects visited the laboratory before the VO2max test to be familiarized with running on the Quinton treadmill (model 18–60; Seattle, WA), operating the emergency-stope lever, and breathing through the Hans Rudolph R2700 respiratory valve (Kansas City, MO) 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 unable to continue. VO2max was held to occur when the VO2 for successive workloads differed by 2 ml·kg\(^{-1}\)·min\(^{-1}\). This criterion is less than two SD for the increments in VO2 that are associated with the step increments of the protocols. The largest VO2 difference between the last two increments of the eight VO2max tests was 1.3 ml·kg\(^{-1}\)·min\(^{-1}\). The 70 and 105% VO2max workloads were subsequently predicted from the regression of steady-state VO2 at ~40, 50, 70, and 80% VO2max respectively, on treadmill speed at 5% elevation.

Recovery and resting VO2. VO2 was measured for the first 25 min postexercise by using the previously described automated system. Subsequent RMR, resting VO2, and recovery VO2 were determined by using the Douglas bag method. Douglas bags (150 liter; Plysu Industrial, Milton Keynes, UK), which had been previously flushed with the subject’s expire, were connected via a two-way straight-through valve to the expiratory port of a Hans Rudolph R2600 respiratory valve. Subjects were connected to the respiratory valve for 2.5 min before the two-way valve was switched into the Douglas bag at the end of an expiration. Collection was completed at the end of an expiration ~10 min later, and the exact collection time was recorded by stopwatch. The volume of expire was determined by using a 350-liter Tissot spirometer (Warren Collins, Braintree, MA) that had been mapped for constant cross-sectional area throughout its elevation. The preexperimental reliability trials for the resting VO2 of six subjects who were measured on consecutive days resulted in an ICC of 0.93 and a CV of 1.8%.

Heart rate. Heart rate (HR) was monitored continuously during all VO2 measurements by an electrocardiogram (Becton-Dickinson, Sharon, MA) by using a CM-5 electrocardiograph.

Rectal temperature. During the treatment and control days, rectal temperature (\(T_r\)) was monitored continuously by customized equipment (18) that was calibrated before data collection against a glass thermometer that had been certified by the National Association of Testing Authorities (Australia).

Experimental design. All subjects participated in a control day and two treatment days that were counterbalanced to eliminate any order effect. Such a design with eight subjects is sensitive enough to detect (\(\alpha = 0.05\) and power = 0.9) an EPOC difference of 5 liters [excess postexercise energy expenditure (EPEE) = −100 kJ] between the two treatments. Subjects were familiarized with the laboratory on three separate occasions before the control and treatments. Two of these visits involved RMR-habituation trials. Subjects ingested a standard dinner (~5,800 kJ; 70% carbohydrate, 15% fat, 15% protein) by 2000 h before the control and treatment days, which commenced at 0720, and they were only permitted to drink water thereafter. On arriving at the laboratory, subjects were asked to void and empty their bowel before being weighed. After subjects were weighed, a rectal temperature probe (18) was inserted and chest electrodes were attached. The subjects then rested quietly on a bed with their shoulders slightly elevated.

RMR was determined after 50 min of bed rest and was followed by one of two equated treatments (continuous running: 30 min at 70% VO2max, interval running: 20 × 1-min intervals at 105% VO2max with intervening 2-min rest periods) or a control session (no running). The treatments were followed by 9 h of bed rest, during which VO2 was measured frequently during the first hour and thereafter for one 10-min period every hour. A standardized lunch, which was identical to the dinner on the preceding evening, was provided at 1230 on both treatment and control days. The laboratory temperature in the vicinity of the subjects was maintained at 24.0 ± 0.5°C, and they were covered with a blanket.

Statistical analyses. The trapezoidal rule was used to approximate the integral for the exercise, 9-h postexercise, and control VO2 values over time. This facilitated the calculation of the net total oxygen cost (NTOC) of exercise (exercise VO2 + 9-h postexercise VO2 – exercise and postexercise control VO2) and 9-h EPOC (9-h postexercise VO2 – 9-h control VO2). Similar computations determined the 9-h net total energy expenditure (NTEE) and 9-h EPEE after each VO2 data point was converted to an energy equivalent by using the equation of Elia and Livesey (12). Dependent t-tests (\(P = 0.05\)) were used to locate statistically significant between-treatment differences for the EPOC and EPEE data. The VO2, respiratory exchange ratio (RER), \(T_r\), and HR data were analyzed via analyses of variance with repeated measures across both time and treatments/control. In the event of a statistically significant F-ratio (\(P \leq 0.05\)), differences between experimental and control conditions for temporally matched variables were identified via Dunnett’s post hoc test (31).

### RESULTS

Postexercise VO2. The postexercise VO2 was significantly greater than the matched control values for 1 and 8 h after the submaximal and supramaximal treatments, respectively (Fig. 1A). The ~27% increase in VO2 at 4 h for the control and two treatments was ~1 h after the ingestion of the standard meal.

EPOC/EPEE. The submaximal and supramaximal treatments resulted in 9-h recovery VO2 consumption of

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**Table 1. Weekly training loads**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Running, km</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>MJ</td>
<td>49–60</td>
<td>2 h of cycling; 2-h weight session</td>
</tr>
<tr>
<td>FA</td>
<td>120</td>
<td></td>
</tr>
<tr>
<td>SH</td>
<td>60–70</td>
<td></td>
</tr>
<tr>
<td>MM</td>
<td>80–90</td>
<td>2.5 km of swimming</td>
</tr>
<tr>
<td>MP</td>
<td>25–35</td>
<td>1.25 h of swimming; 2.5 h of cycling</td>
</tr>
<tr>
<td>MH</td>
<td>50</td>
<td>2-h weight session</td>
</tr>
<tr>
<td>MDH</td>
<td>55</td>
<td></td>
</tr>
<tr>
<td>NT</td>
<td>95–100</td>
<td></td>
</tr>
</tbody>
</table>

Listed are average weekly training loads for 12 mo preceding experiment.
163.8 ± 12.8 and 171.8 ± 13.4 liters, respectively. These values were significantly greater (P < 0.001 for both treatments) than the control day O₂ consumption of 156.8 ± 10.9 liters. Table 2 indicates that the differences among the submaximal and supramaximal treatments for EPOC (P = 0.001), NTOC (P = 0.001), EPEE (P = 0.007), and NTEE (P = 0.005) were all statistically significant. When both the EPOC and EPEE values were expressed as percentages of the NTOC and NTEE, respectively, the submaximal treatment comprised 7.1 and 6.6% of the NTOC and NTEE of exercise, respectively, whereas the corresponding values were 13.8 and 11.9% for the supramaximal treatment (Table 2).

Figure 1, B-D, summarizes the recovery data for RER, Tᵣₑᵣ, and HR, respectively. The pretreatment values for each of these variables were not significantly different from those for the corresponding control values. Although the RER values for the supramaximal treatment were significantly lower than the control values for the first 4 h of recovery and at 8 h, those for the submaximal treatment were lower at 4 h postexercise, which corresponded to the first postprandial measurement. Tᵣₑᵣ returned to control values for both treatments within 2 h postexercise. Although the majority of postexercise HR values for the supramaximal treatment were elevated significantly (P ≤ 0.05), they ranged from only 2 to 6 beats/min above the matched controls after 1 h of recovery. HR returned to control levels after 1 h for the submaximal treatment.

The HR values for both treatments at 9 h postexercise were not significantly different from the control value.

DISCUSSION

This is the first study to investigate the relationship between the EPOC values when supramaximal and submaximal workloads are equated. Table 2 indicates that the supramaximal work produced a significantly greater 9-h EPOC compared with that for the submaximal treatment; the two EPOC values comprised 7.1 and 13.8% of their respective NTOC values. Furthermore, the energy content of 1 kg of adipose tissue is approximately equivalent to 1) 215 EPEE and 16 NTEE for the submaximal treatment and 2) 116 EPEE and 14 NTEE for the supramaximal treatment. Notwithstanding the higher EPEE for supramaximal interval running compared with submaximal continuous running, the major contribution of both to weight loss is therefore via the energy expended during the actual exercise. The 135-kJ greater EPEE for the interval treatment is of little physiological significance to the energy balance of athletes because this amount of energy is equivalent to the kilojoules in only 75 ml of orange juice. However, when exercise for weight loss is utilized, the EPEE would have a cumulative effect when the exercise is...
undertaken regularly. Although the EPEE resultant from supramaximal running in this study would be associated with a greater cumulative effect, the exercise intensity and duration involved would be beyond the capabilities of nonathletes. It has also been reported that exercise programs utilizing intensities >85% \( V_{O2\text{max}} \) are associated with significant increases in dropout rates and injuries (22).

Few researchers (1, 2, 17, 33) have reported the precision of their indirect calorimetry system, and this is a key issue underlying our conclusions. The lack of reliability data, combined with inadequate controls for the factors known to influence RMR, often confound comparisons between studies. In the present investigation, temporally matched measurements of each subject's RMR were conducted on the control day and before each treatment. These three measurements produced a CV of 3% and an ICC of 0.88. The smallest difference between the control and treatment \( V_{O2} \) that achieved statistical significance was \( \sim 5\% \), which exceeds the precision of our \( V_{O2} \) measurement. Garrow (16) also reported that the intraindividual biological variability in RMR is \( \sim 5\% \). This value is greater than our precision data, which include both biological variability and technical or equipment error.

Our results suggest that supramaximal workloads produce more prolonged elevations in recovery \( V_{O2} \) than moderate work intensities (i.e., 70–75% \( V_{O2\text{max}} \)). This is in accordance with the positive linear relationship between submaximal exercise intensity and EPOC that was reported by Gore and Withers (17). After the submaximal treatment, postexercise \( V_{O2} \) was generally not significantly different from control values after 1 h of recovery, whereas recovery \( V_{O2} \) after the supramaximal treatment did not return to baseline until 9 h postexercise. The \( V_{O2} \) recovery pattern for the submaximal treatment falls between that obtained by Gore and Withers (17) for 20 and 50 min of exercise at 70% \( V_{O2\text{max}} \). Quinn et al. (25) reported a significantly elevated recovery \( V_{O2} \) for 3 h after 30 min of treadmill walking at 70% \( V_{O2\text{max}} \) by young trained women. In contrast to the earlier work of Bahr et al. (3), Sedlock et al. (28) reported that \( V_{O2} \) was elevated for only 33 min after the cessation of 20 min of cycling at 74% \( V_{O2\text{max}} \). Smith and McNaughton (30) and Chad and Wenger (10) utilized cycling with young trained men and women at 70% \( V_{O2\text{max}} \) for 30 min and found recovery to be complete within 50 and 128 min, respectively. Bahr et al. (2) are the only other investigators to use supramaximal interval exercise to investigate recovery \( V_{O2} \). Their untrained young male subjects completed one, two, and three 2-min bouts of cycling at 108% \( V_{O2\text{max}} \), which were associated with an elevation of recovery \( V_{O2} \) for 30, 60, and 240 min and with EPOC values of 4.8, 10.4, and 16.6 liters, respectively. Although Brockman et al. (8) also employed interval treadmill running, their maximum workload was not supramaximal (7 \( \times \) 2-min exercise bouts at 90% \( V_{O2\text{max}} \) with 2-min active rest periods). They reported a 12.7% elevation in recovery \( V_{O2} \) after 1 h for their young female distance runners, which is similar to that found in this study for the supramaximal treatment. The difference in recovery times between the preceding studies and our treatments could be attributed to a number of factors, including exercise modality and/or the greater \( V_{O2\text{max}} \) values for our subjects. Furthermore, some of the investigators (8, 10, 29, 31) used a resting \( V_{O2} \) baseline that was extrapolated from a pretreatment measure, and different methods have also been used to determine when \( V_{O2} \) had returned to baseline.

A further consideration, which has not been previously discussed in studies (2, 8) utilizing interval work, is the contribution to the EPOC of the increased \( V_{O2} \) during the recovery intervals. When the recovery interval \( V_{O2} \), which is in excess of both the control day \( V_{O2} \) and \( O_2 \) deficit incurred during the work intervals, was added to the 15.0-liter 9-h EPOC determined from the cessation of the last work interval, then the overall EPOC is 37.2 liters. However, this represents an inflated EPOC estimate because the subjects were standing and moving their legs to prevent venous pooling during the recovery intervals. It was not feasible to have them lying down during the recovery intervals to replicate the control day conditions from which the \( V_{O2} \) baseline was derived. In our laboratory, results in two subjects demonstrated that moving and stretching the legs while standing required a \( V_{O2} \) that was threefold greater than that on the control day (unpublished observations). Allowance for this elevation led to an overall EPOC estimate of 17.3 liters, which is not markedly different from that of 15.0 liters determined from cessation of the last work interval. However, further work is required with interval treatments when the corresponding control periods replicate the recovery movement patterns of the intermittent exercise.

There is good agreement between the 9-h EPOC for our submaximal treatment and those reported for experiments that measured recovery \( V_{O2} \) until it returned to baseline. The average EPOC of two previous studies that used treadmill running and controlled for

<table>
<thead>
<tr>
<th>Treatment</th>
<th>EPOC, liters</th>
<th>NTOC, liters</th>
<th>EPOC/NTOC, %</th>
<th>EPEE, kJ</th>
<th>NTEE, kJ</th>
<th>EPEE/NTEE, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Submaximal running</td>
<td>6.9±3.8</td>
<td>97.3±10.4</td>
<td>7.1</td>
<td>133 ± 82</td>
<td>2.019 ± 206</td>
<td>6.6</td>
</tr>
<tr>
<td>Supramaximal running</td>
<td>15.0±3.3</td>
<td>108.4±12.2</td>
<td>13.8</td>
<td>268 ± 87</td>
<td>2.256 ± 264</td>
<td>11.9</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = 8 subjects. EPOC, excess postexercise oxygen consumption; NTOC, net total oxygen cost; EPEE, excess postexercise energy expenditure; NTEE, net total energy expenditure.
the diurnal variation in RMR (17, 25) was 7.1 liters compared with our value of 6.9 liters. In the only study of supramaximal exercise, Bahr et al. (2) reported an EPOC of 16.3 liters for 14 h postexercise. However, they utilized untrained men who exercised supramaximally for only 6 min compared with the 20 min used in our investigation, which was associated with a 9-h EPOC of 15.0 liters. It is interesting to note that the Gore and Withers (17) data for 80 min of treadmill running at 70% \( \dot{V}_{O2max} \) which is over double the work performed in our supramaximal protocol, produced a similar 8-h EPOC of 14.6 liters.

The dip in RER values for both treatments before 1 h postexercise (Fig. 1B) is indicative of CO\(_2\) retention after strenuous exercise to replenish the bicarbonate used to buffer lactic acid. The more pronounced fall for the interval treatment was probably due to greater lactate buffering. Recovery RER values were significantly lower than the control values during the first 4 h of recovery for the supramaximal treatment. Muscle glycogen stores would have been depleted to a greater extent during the supramaximal treatment, thereby leading to a greater reliance on fat metabolism in the recovery period. For the same \( \dot{V}_{O2} \) fat yields less energy than carbohydrate: it is therefore logical that the EPOC/NTOC of 13.8% for the supramaximal treatment is greater than the EPEE/NTEE of 11.9% (Table 2). The contribution of elevated fat metabolism toward the EPOC in this case was estimated to be only 0.8 liter. RER values were significantly lower than control values for both treatments for the 1 h postprandial, presumably while repletion of muscle glycogen was occurring. It has been reported (17) that postprandial glycogen synthesis may account for ~1 liter of the EPOC. However, Bahr (1) has suggested that this value should be disregarded because it is probably less than the \( O_2 \) consumed in the control condition when excess carbohydrate is converted to fat as opposed to glycogen. Only 5.3% of the energy content of ingested carbohydrate is required to store it as glycogen, compared with 23–24% for conversion to triglyceride (13).

\( T_r \) for both treatments had returned to control levels within 2 h. It is therefore unlikely that \( T_r \) contributed to the significantly greater EPOC associated with the supramaximal treatment. The sum of the \( T_r \) differences between the control and treatments over the entire postexercise period for each subject did not correlate significantly with the EPOC. These correlations were 0.30 and 0.13 for the submaximal and supramaximal treatments, respectively. \( T_r \) therefore accounted for only 9 and 2% of the EPOC variance. Maehlum et al. (23), Bahr et al. (2), and Gore and Withers (17) also reported that \( T_r \) only accounted for a small proportion of the EPOC. It has been suggested (17) that muscle temperature may be more closely correlated with the EPOC, but this was not measured.

Although HR was significantly elevated above control levels for much of the supramaximal recovery period, the physiological significance of this is doubtful. All the postexercise HR values after 1 h of recovery for the supramaximal treatment ranged from 2 to 6 beats/min more than the matched control values. Given that the heart consumes ~10% of the resting \( \dot{V}_{O2} \) (19) and the elevations in HR beyond 1 h of recovery were low (2–6 beats/min), the contribution of extra myocardial \( \dot{V}_{O2} \) to the EPOC would be negligible.

Several other factors have been proposed to contribute to EPOC. These include the potentiated thermic effect of feeding (TEF), elevated ventilation (Ve), lactate metabolism, hormonal influences, substrate cycling, and glycogen synthesis from ingested carbohydrate. Bahr and Sejersted (5) reported that a 4.5-MJ test meal 2 h after cessation of 80-min cycling at 75% \( \dot{V}_{O2max} \) did not potentiate the TEF. Hence, it is unlikely that any of the EPOC differences in this study can be attributed to a 5.8-MJ meal 3 h after the cessation of exercise. Ve for the treatments in this study was elevated above the control Ve by ~9% at 1 h postexercise but had returned to control levels for both treatments by 2 h postexercise. The \( \dot{V}_{O2} \) of the respiratory muscles at rest is 1–2% of the RMR (26); it is therefore likely that the modest elevation in Ve before 2 h postexercise would have a negligible effect on the EPOC. The impact of the other factors on the EPOC have been reviewed by Bahr (1) and lactate metabolism could possibly explain some of the EPOC difference between the two treatments used in this study, but plasma and muscle lactate were not measured. Bahr et al. (2) estimated that 4 liters of \( O_2 \) were required to synthesize glycogen from 50% of the lactate generated from 3 × 2-min bouts of cycling at 108% \( \dot{V}_{O2max} \). This value only represents a little over one-half of the difference between the EPOC values in our study, but, given our more strenuous (20-min) supramaximal protocol, it is possible that glycogenesis from lactate could account for the EPOC difference between the two treatments. However, estimates of \( \dot{V}_{O2} \) in relation to lactate metabolism need to be treated with caution. The determination of lactate kinetics is difficult because plasma lactate is not indicative of the total lactate produced during exercise. Furthermore, extrapolation of the lactate concentration in a single muscle biopsy to the amount of lactate in an estimated active muscle mass may be erroneous. Moreover, uncertainty exists in relation to what proportion of lactate is channeled into glycogenesis, which is the component of lactate metabolism that contributes to the EPOC.

In conclusion, this study has demonstrated that the EPEE is significantly greater for supramaximal running compared with submaximal running when there is an attempt to equate the amounts of work performed. Notwithstanding the higher EPEE for supramaximal interval running, the major contribution of both treatments to weight loss was via the energy expended during the actual exercise. The EPEE is therefore of negligible physiological significance as far as weight loss is concerned, unless the exercise is undertaken regularly when the EPEE would have a cumulative effect.

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