Fat-free mass and gender influences the rapid-phase excess postexercise oxygen consumption

Linda S. Lamont, Ph.D.
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Abstract: The purpose of this study was to determine the influence of gender dimorphism and body composition on post-exercise oxygen consumption during the rapid recovery phase. We compared the rapid-phase excess postexercise oxygen consumption (EPOC) in men and women matched for age (32.1 years), physical activity status, and maximal oxygen consumption (44.7 mL·kg⁻¹·min⁻¹), but not for body mass or fat-free mass (FFM). All subjects exercised for 1 h at 50% of their peak capacity. Although there were differences between genders in the magnitude of the absolute oxygen consumption and EPOC during the rapid phase of recovery, there were no differences found when EPOC was corrected for FFM. We conclude that the gender differences in the absolute O₂ consumption and EPOC are related to the size of the FFM.

Key words: rapid-phase EPOC, postexercise metabolism, fat-free mass, recovery, H₂[¹⁸O].

Introduction

Much is known about the effects of intensity, duration, and type of exercise for postexercise oxygen consumption (V̇O₂) (Børsheim and Bahr 2003), but little is known about the effects of gender or whole-body fat free mass (FFM). Ovarian hormones are known to influence metabolism during both exercise and recovery (Lamont et al. 1987, 2001; Lamont 2005), and menstrual cycle variations in ovarian hormones have been found to alter the excess postexercise V̇O₂ (EPOC) in 1 study (Matsuo et al. 1999), but not in another (Fukuba et al. 2000). Gender differences have been reported for absolute oxygen uptakes post exercise, but EPOC was not measured (Berg 1991). The duration of EPOC was found to be longer for men than women, but these differences were eliminated when corrections were made for body mass (Smith and McNaughton 1993).

It is unknown if there are differences in the magnitude of EPOC or if gender differences in FFM are responsible for the differences reported in past investigations. In addition, studying the influence of gender dimorphism and the effects of body composition on metabolism will help to assess those factors that influence energy expenditure during recovery. This is timely because there are recent proposals to estimate postexercise energy expenditure (as 15% of the exercise energy expenditure) and add it to the physical activity expenditure to calculate total energy expenditure (Institute of Medicine 2005).

Our study was designed to assess the rapid phase, as opposed to the prolonged phase, of EPOC. The rapid phase is defined as the sum of the components that decay within the first hour of exercise recovery; the prolonged recovery phase decays mono exponentially with a half-life of several hours. The work intensities used in this study are associated with a return to baseline within 1 h and, therefore, the rapid phase was assessed. We compared recovery in men and women matched for age, habitual physical activity, and peak oxygen consumption (V̇O₂ peak) to assess gender differences. In addition, FFM was measured using a tracer dilution method so that the relationship between body composition and the magnitude of the rapid phase of EPOC could be determined. Lastly, we assessed if there was a gender differential in the


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contribution of EPOC to the energy costs of physical activity as measured by the net total VO₂.

Materials and methods

Participants

Fourteen healthy, active subjects (7 female, 7 male) participated in this investigation. The characteristics of these participants are shown in Table 1. The male subjects were each paired with a female according to age and physical activity status. Training status and training quantity (both volume and intensity) were determined from verbal self reports and were confirmed with maximal oxygen consumption (VO₂ max) testing. As can be seen in Table 1, the use of gender matched pairs also allowed us to compare groups exercising at similar relative intensities because VO₂ peak was not different when expressed per kilogram of body mass or per kilogram of FFM (p = not significant (ns)).

Physical examinations were performed by a physician prior to exercise testing and all 14 participants were found to have normal heart sounds, electrocardiograms, and resting blood pressures. In addition, the menstrual cycle phase was determined for our female participants using a monoclonal antibody self-test kit (Ovukit, Quidel, San Diego, Calif.). None of the female subjects were ovulating and 6 were studied during the follicular phase of the menstrual cycle and 1 was using an oral contraceptive. We also collected 24-h urine volumes, which were analyzed for urea nitrogen content as an indirect measure of protein catabolism. This measure was included because the menstrual cycle and exercise alters protein catabolism, which is an energetically demanding process (Lamont et al. 1987).

Preliminary testing

The subjects’ VO₂ peak were assessed with a graded cycle ergometry protocol (Monark, Varberg, Sweden) using a metabolic cart (model 2900, Sensormedics, Yorba Linda, Calif.). During the maximal exercise test, these subjects exhibited a plateau in VO₂ and an r value >1.0 at peak work (female: 196 W ± 13.87; males: 269 W ± 24.51; p = ns). There was no difference between groups in the peak heart rate attained during the graded exercise test (females: 178.3 ± 3.9 beats-min⁻¹; males: 183.6 ± 3.9 beats-min⁻¹; p = ns). The VO₂ peak data was used to determine the workload (50% of VO₂ peak) that each subject would exercise at ~4 days later. Choosing the same relative work intensity for both groups represented a similar metabolic challenge for the males and females. Although the relative exercise intensity was matched among groups, there were absolute differences in work output. After the VO₂ peak test, each subject was instructed to eat a standardized diet for 1 week prior to the EPOC measurement. A dietician instructed them on this diet, and it was designed to be isocaloric and nutritionally balanced by providing 30% fat, 10%–12% protein, and 58%–60% carbohydrate. Total calories consumed were 1893 ± 113 kcal (1 kcal = 4186.8 J) for females vs. 2442 ± 87 kcal for males; p < 0.003). These subjects were also asked to refrain from vigorous physical activity for 48 h prior to the EPOC measurements. Lastly, they were told to refrain from smoking and caffeine ingestion prior to testing, but none of these subjects were smokers. Compliance with these dietary and activity procedures was assessed by reviewing each subject’s dietary record and by conducting verbal interviews prior to the metabolic measurements.

EPOC measurement

Subjects were driven to the laboratory on the morning of the EPOC measurements and rested in a supine position for 3 h in a fully awake and relaxed state. Each subject arrived at the laboratory in a postabsorptive state with their last meal being consumed the previous evening. They were not permitted to eat or drink caffeine beverages the morning of their test. After the establishment of resting conditions (3 h of supine rest), they exercised for 1 h at 50% of their predetermined peak aerobic capacity to insure that we were comparing the recovery response to a similar metabolic challenge. Although the relative exercise intensity was matched, this procedure did not ensure a similar total of work performed over the hour of exercise. Exercise was performed with a constant-load pan weight cycle ergometer, and the flywheel resistance was periodically checked to ensure accuracy. Recovery was monitored for 1 h after exercise (during the rapid phase of recovery) with the subjects in a supine position; therefore, we measured the magnitude and not the duration of the rapid-phase EPOC.

Resting expired air samples were collected at 30, 60, 90, 120, 150, and 180 min using a Hans Rudolph face mask (Hans Rudolph Inc., Kansas City, Mo.) that was interfaced with the metabolic cart. The mean of the 6 continuous oxygen uptake measurements was used to ensure a stable baseline for the resting data (Lamont et al. 1997). Besides VO₂, the resting air samples were analyzed for carbon dioxide production VCO₂, and VC[¹⁸O₂], which is the ¹³C enrichment of the expired CO₂ at steady-state.

Body composition was assessed during the resting phase of the study using a stable isotope of water H₂[¹⁸O] and the tracer dilution method that has been previously described (Schoeller et al. 1980). An isotopic plateau for expired Cl[¹⁸O₂] was found during the 3-h rest period. This body composition technique assumes that the total body water is a fixed fraction (73.2%) of the FFM (Schoeller et al. 1980). Air samples were collected throughout the 1-h sub peak exercise and for 1 h into recovery. To compute EPOC, the mean of the resting VO₂ was subtracted from the mean of the recovery VO₂ (Quinn et al. 1994) and an average was determined. In addition, the net total oxygen cost (NTOC) of this exercise (NTOC = exercise + recovery VO₂ – resting VO₂) was calculated. This research protocol was approved by the Human Experimentation Review board at Case-Western Reserve University School of Medicine (Cleveland, Ohio), and an informed consent was signed by each subject prior to testing.

Statistical analysis

Sigma Stat statistical software was used to perform all analyses and the data were reported as means ± SEM. The statistical power for these data at an α = 0.010 was calculated to be 0.998. The test–retest reliability of the indirect calorimetry procedures was also determined (r = 0.92; sample n = 4). A Student’s t test was used for measuring the differences between groups. In addition, a Pearson product
moment correlation was used to define the relationship among variables. In all cases, a $p < 0.05$ was considered statistically significant.

**Results**

The measurements obtained during the 3-h pre-exercise supine rest period are found in Table 2. The mean $V\dot{O}_2$ during this rest reflected the well-established gender difference in resting metabolic rate. During this time period, there was no gender difference in resting respiratory exchange ratio (RER) or heart rate.

$V\dot{O}_2$ was measured post exercise for 1 h. The mean 1-h measurement was recorded as the absolute $V\dot{O}_2$ (APOC), and the resting $V\dot{O}_2$ was subtracted from the APOC to compute EPOC. The APOC was significantly ($p < 0.001$) larger in the male subjects (306.7 ± 12.6 mL $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$) when compared with the females (216.2 ± 10.8 mL $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$). Table 3 indicates that the average EPOC (postexercise $V\dot{O}_2$ – resting $V\dot{O}_2$) was also heightened in the males when compared with the females. These EPOC data indicate that the females expended an additional 4.43 ± 1.5 kcal during the hour of exercise recovery and the males an additional 8.79 ± 1.8 kcal.

Although this rapid-phase EPOC showed a significant gender difference, when corrections were made for FFM the values were similar (Table 3). Thus, FFM had a significant relationship to the magnitude of the EPOC response and the correlation coefficient ($r = 0.70$) between these 2 variables was significant ($p < 0.0075$). Analysis of covariance (ANCOVA) indicates that an $r > 0.60$ is necessary to be considered a significant covariate (Neter et al. 1983). Therefore, the present study found that fat-free tissue mass significantly impacted the size of the rapid-phase EPOC. In addition, FFM accounts for approximately half of the variance in this measure. Lastly, when EPOC was expressed as a percentage of the NTOC (Table 3) for this exercise, there was no difference between genders (0.02% ± 0.007% for females vs. 0.05% ± 0.013% for males; $p = ns$).

There was a difference between gender groups in the 24-h urinary urea nitrogen excretion and hence protein catabolism (5.80 ± 0.50 g $\cdot$ day$^{-1}$ for females vs. 12.50 ± 1.98 g $\cdot$ day$^{-1}$ for males; $p < 0.007$). However, there was no relationship between EPOC and urinary urea nitrogen excretion ($r = 0.15$; $p = ns$).

**Discussion**

A previous review of the postexercise $V\dot{O}_2$ literature highlights the fact that the gender effects on EPOC are still unknown (Børsheim and Bahr 2003). There are a few reasons for this lack of understanding: amount of gender comparison was limited; of those comparisons that have been performed, menstrual cycle phase was not accounted for; and, in some studies, training status was not matched between genders. Our investigation contributes to the literature because menstrual cycle phase was determined and the training status was controlled for by matching subjects prior to testing. Noting menstrual cycle phase is important because resting metabolic rate (RMR) is at its lowest point 1 week prior to ovulation (Solomon et al. 1982) and cycle phase can influence protein catabolism, which is an energetically demanding process (Lamont et al. 1987).

We reported that there are gender differences in resting $V\dot{O}_2$ and, therefore, RMR, and this finding is consistent with the literature. It is a well-documented fact that females have a lower RMR than males (Durnin 1981), but more recent research indicates that β1-adrenoreceptor activity may modulate RMR (Lamont et al. 1997). There are gender differences in catecholamine responsiveness during rest and exercise (Braun and Horton 2001; Lamont et al. 2003). Therefore, future investigations should explore the effect of the interaction between catecholamine responsiveness and gender during the metabolic recovery from exercise.

We reported no difference in aerobic capacity between our gender groups, whether expressed per kilogram of body mass or per kilogram of FFM. Therefore, both groups were compared at similar relative (but not absolute) workloads. Exercise intensity is a key factor in the size of the EPOC, and it has been found to account for nearly half of the variance (45.5%) in this measure (Gore and Withers 1990).

The present study was designed to determine if there are

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**Table 1. Characteristics of the male and female subjects (mean ± SEM).**

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (y)</th>
<th>Body mass (kg)</th>
<th>FFM (kg)</th>
<th>$V\dot{O}_2$ peak (mL $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$)</th>
<th>$V\dot{O}_2$ peak (mL $\cdot$ kg $\cdot$ FFM$^{-1}$ $\cdot$ min$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>30.6±3</td>
<td>59.4±3</td>
<td>48.7±2</td>
<td>42.2±3.3</td>
<td>50.5±3</td>
</tr>
<tr>
<td>Male</td>
<td>33.6±4</td>
<td>79.9±3</td>
<td>71.9±3</td>
<td>47.3±3.3</td>
<td>52.6±3</td>
</tr>
<tr>
<td>$p$</td>
<td>ns</td>
<td>0.01</td>
<td>0.01</td>
<td>ns</td>
<td>ns</td>
</tr>
</tbody>
</table>

**Note:** FFM, fat-free mass; $V\dot{O}_2$ peak, peak oxygen consumption; ns, not significant.

**Table 2. Baseline measurements established during the 3-h pre-exercise rest period (mean ± SEM).**

<table>
<thead>
<tr>
<th>Group</th>
<th>$V\dot{O}_2$ (L $\cdot$ min$^{-1}$)</th>
<th>RER</th>
<th>Heart rate (beats $\cdot$ min$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>0.21±0.008</td>
<td>0.75±0.03</td>
<td>65±5.6</td>
</tr>
<tr>
<td>Male</td>
<td>0.27±0.011</td>
<td>0.83±0.02</td>
<td>61±3.6</td>
</tr>
<tr>
<td>$p$</td>
<td>0.001</td>
<td>ns</td>
<td>ns</td>
</tr>
</tbody>
</table>

**Note:** $V\dot{O}_2$, oxygen consumption; ns, not significant.

**Table 3. Recovery measurements established during the 1-h post-exercise period (mean ± SEM).**

<table>
<thead>
<tr>
<th>Group</th>
<th>EPOC (mL $\cdot$ min$^{-1}$)</th>
<th>EPOC–FFM (mL $\cdot$ kg $\cdot$ FFM$^{-1}$ $\cdot$ min$^{-1}$)</th>
<th>NTOC (L $\cdot$ min$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>15.8±5.3</td>
<td>0.31±0.26</td>
<td>65±5.6</td>
</tr>
<tr>
<td>Male</td>
<td>45.3±11.9</td>
<td>0.62±0.15</td>
<td>61±3.6</td>
</tr>
<tr>
<td>$p$</td>
<td>0.001</td>
<td>ns</td>
<td>ns</td>
</tr>
</tbody>
</table>

**Note:** NTOC = exercise $V\dot{O}_2$ + postexercise $V\dot{O}_2$ – resting $V\dot{O}_2$; EPOC, postexercise oxygen consumption; FFM, fat-free mass; NTOC, net total oxygen cost; ns, not significant; $V\dot{O}_2$, oxygen consumption.
gender differences in the rapid phase of EPOC. We determined that there were significant differences, but this effect appears to be related to the size of the FFM. Others report that men have an EPOC duration that is heightened when compared with females when exercising for 30 min at various exercise intensities (40%, 50%, and 70% of VO₂peak) (Smith and McNaughton 1993). In all of these exercise intensities, the magnitude of the EPOC difference disappeared when adjustments were made for body mass. Although we studied an exercise of longer duration, our intensity was within the intensity range used by Smith and McNaughton (1993). Therefore, our data concurs with theirs and indicates that body composition differences are also critical components to the size of the rapid phase of EPOC.

In summary, this investigation found that males, when compared with females, had a greater APOC and EPOC during the rapid phase of recovery from 1 h of exercise at 50% of peak capacity. The magnitude of this difference was related to the size of the FFM and was found to account for approximately half of the variance. Although gender differences in protein catabolism were found, no relationship was reported for this metabolic process and the EPOC rapid phase.

References


