Effect of exercise intensity on 24-h energy expenditure and nutrient oxidation

EDWARD L. MELANSON, TERESA A. SHARP, HELEN M. SEAGLE, TRACY J. HORTON, WILLIAM T. DONAHOO, GARY K. GRUNWALD, JERE T. HAMILTON, AND JAMES O. HILL

Center for Human Nutrition, University of Colorado Health Sciences Center, Denver, Colorado 80262

Received 6 July 2001; accepted in final form 10 November 2001

Despite a plethora of investigations that have examined the acute effect of exercise at different intensities on energy expenditure (EE), we have little understanding of the effect of exercise intensity on 24-h EE. It has been reported that, compared with low-intensity exercise, postexercise EE and resting metabolic rate measured the next day are acutely enhanced by high-intensity exercise (1, 12, 26, 33). This would suggest that high-intensity exercise should lead to a greater overall daily EE. However, spontaneous physical activity in the postexercise period may be affected by exercise intensity. For example, using the doubly labeled water technique, Goran and Poehlman (16) demonstrated that total daily EE was unchanged after a period of high-intensity aerobic training in a group of elderly subjects. Although it is possible that the age of this cohort affected their activity during the nonexercise period of the day, these data demonstrate that increases in total daily EE with higher intensity exercise cannot be inferred from acute increases in EE due to a bout of exercise. The only manner by which the acute effects of exercise intensity on 24-h EE and the components of daily EE can be determined is by using whole room indirect calorimetry.

The effect of exercise intensity on 24-h nutrient oxidation is also unclear. It has been well documented that the relative contribution of fat oxidation to total EE is greatest at lower exercise intensities (21, 28, 29, 36). For example, during exercise at 25% of maximal oxygen consumption (VO2 max), nearly all of the EE is derived from fat (28). As exercise intensity increases, the proportional contribution of fat oxidation to total EE decreases (6, 15, 21, 28, 29). These studies have too often been misinterpreted by fitness practitioners to suggest that lower intensity exercise is best for maximizing fat oxidation and fat loss. However, it may be that postexercise nutrient oxidation is affected differently by the intensity of exercise. Indeed, there is evidence of a greater reliance on fat during the postexercise period when high-intensity is compared with low-intensity exercise (4, 37). However, in these studies, EE and substrate oxidation have only been measured for a few hours after the exercise bout. To completely understand the implications for body weight regulation, the prolonged effects of exercise on nutrient oxidation must be considered. It could be that any increases in fat oxidation during the exercise period are offset by decreases in fat oxidation during the remainder of the day. Furthermore, most studies of the effect of exercise intensity on EE and substrate utilization have not matched the energy cost of the exercise bout. Another consideration is that few of these short-term studies have controlled both preexercise energy intake and exercise. This is important for ensuring that subjects are in a similar state of energy balance and fuel repletion at the time of testing, because overand underfeeding have profound effects on nutrient oxidation.
oxidation (18). The only way to evaluate the effects of exercise intensity on nutrient oxidation is to study subjects over a 24-h period by using whole room indirect calorimetry, with the application of suitable pre-study dietary and exercise controls.

There is evidence that muscle enzymatic capacity is related to 24-h substrate oxidation. For example, under nonexercise conditions, Zurlo et al. (41) found a highly significant relationship \((r = -0.75)\) between \(\beta\)-hydroxyacyl-CoA dehydrogenase (HADH) activity (a key \(\beta\)-oxidation enzyme) and 24-h respiratory ratio (RQ) measured by using whole room calorimetry. With regard to exercise, it is well known that, as exercise intensity increases, more type II muscle fibers are recruited (14) and that these fibers have high levels of glycolytic enzymes, so it is reasonable to hypothesize that 24-h substrate oxidation is related to skeletal muscle enzymatic profile. Whether the skeletal muscle enzymatic profile is related to 24-h substrate oxidation under exercise conditions has not been considered.

The primary aim of this investigation was to examine the effect of exercise intensity on 24-h EE and substrate oxidation to test the hypotheses that, compared with a day on which low-intensity exercise is performed, 1) 24-h EE will be greater on a day on which high-intensity exercise is performed and 2) 24-h fat oxidation will be lower on a day on which high-intensity exercise is performed. To achieve these aims, we measured 24-h EE and substrate oxidation during three stays in a whole room calorimeter. On two occasions, subjects expended 1,670 kJ (400 kcal) during stationary cycling at workloads corresponding to 40 and 70% of their maximal aerobic capacity. These data were compared with a sedentary day during which no exercise was performed. A secondary aim was to determine whether skeletal muscle enzymatic profile is related to 24-h substrate oxidation on days on which low- and high-intensity exercise is performed. To achieve this aim, a resting skeletal muscle biopsy was obtained and analyzed for glycolytic and oxidative capacity.

METHODS

Subjects

Sixteen (8 men, 8 women) lean, healthy adults participated in the study. Subjects were nonobese (body fat = 13–22% for men, 19–28% for women), moderately active (3–5 h/wk of exercise, as determined from self-report), and between 20 and 45 yr of age. Smokers or individuals reporting a history of diabetes, cardiovascular disease, or metabolic disorders known to affect intermediary metabolism were excluded. A health history and a physical examination were performed to confirm that there were no medical reasons for exclusion from the study. Descriptive characteristics of the study subjects are presented in Table 1. The study protocol was approved by the Colorado Multiple Institutional Review Board and the Scientific Advisory Board of the General Clinical Research Center (GCRC) at the University of Colorado Health Sciences Center. Because of scheduling constraints, we were unable to standardize the phase of the menstrual cycle in which female subjects were studied.

Preliminary Assessments

Aerobic capacity. Maximal aerobic capacity was determined from \(\dot{V}O_2\) max measured by using standard indirect calorimetry (model 2900, Sensormedics Metabolic Cart, Yorba Linda, CA) during a graded exercise test using a stationary cycle ergometer. Subjects pedaled at a cadence of 70 rpm beginning at a workload of 50 W. The workload was increased by 25 W every minute until volitional exhaustion. Heart rate (monitored by using a 12-lead electrocardiogram), blood pressure, and perceived exertion were measured every minute. \(\dot{V}O_2\) max was determined from the average of the highest three measurements during the final stage of exercise. To be accepted as valid, the test was required to meet two of the following three criteria: 1) a RQ > 1.1; 2) heart rate within 10 beats/min of 85% of age-predicted maximum; and 3) an increase in oxygen consumption in response to the final workload of \(<2.0\ \text{ml kg}^{-1}\ \text{min}^{-1}\).

Body composition. Body composition was determined by hydrodensitometry, with residual volume measured simultaneously by using the open-circuit nitrogen-dilution technique (13). Nitrogen was measured by using a Med-Science 505-D Nitrifier (St. Louis, MO). Percent body fat was estimated from body density (average of 7–10 repeat measurements) by using the revised equation of Brozek (5).

Resting metabolic rate. Resting metabolic rate (RMR) was measured by using indirect calorimetry (model 2900, Sensormedics Metabolic Cart). Measurements were made in the morning after a 12-h fast and 24-h abstention from exercise. After 30 min of rest, RMR was measured for 15–20 min by using a ventilated canopy. Oxygen consumption and carbon dioxide production were used to calculate RMR according to the formula of Weir (39).

Experimental Protocol

24-h EE and substrate oxidation were measured on four occasions, ~1 wk apart, by using whole room, indirect calorimetry. The first calorimeter day served as a baseline day, during which actual 24-h EE was measured without any cycling exercise performed. This allowed us to produce a state of energy balance with greater precision during the experimental days. Subjects were then studied on 3 additional calorimeter days performed in random order: 1) no exercise day (Con); 2) a low-intensity aerobic (40% of \(\dot{V}O_2\) max) exercise day (LI); 3) a high-intensity aerobic (70% of \(\dot{V}O_2\) max) exercise day (HI). The aerobic exercise bouts were performed on a stationary electronically braked bicycle ergometer (Pedal Mate, Warren E. Collins, Braintree, MA). Subjects expended

<table>
<thead>
<tr>
<th>Table 1. Subject characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
</tr>
<tr>
<td>--------</td>
</tr>
<tr>
<td>Male (n = 8)</td>
</tr>
<tr>
<td>Female (n = 8)</td>
</tr>
</tbody>
</table>

Values are means ± SD; n, no. of subjects. BMI, body mass index; \(\dot{V}O_2\) max, maximal oxygen consumption; FM, fat mass; FFM, fat free mass.

J Appl Physiol • VOL 92 • MARCH 2002 • www.jap.org
~400 kcal (1,670 kJ) above resting levels during each exercise bout. The amount of time and workload required to achieve the desired EE was predetermined by using indirect calorimetry and based on steady-state responses at 40 and 70% of V\text{O}_{2}\text{max}. All exercise sessions were supervised by a technician and workloads controlled via a work integrator external to the calorimeter.

**Prestudy control.** For 3 days before each calorimeter stay, subjects were provided a diet estimated to meet free-living energy requirements (RMR × 1.5–1.8, depending on self-reported physical activity level) and maintain weight stability. The composition of the diet was 30% of energy from fat, 15% of energy from protein, and 55% of energy from carbohydrate. Breakfast was consumed each day in the GCRC, with other food packaged and taken with the subject to be consumed off site. No other food was permitted, and subjects were required to consume all food provided. Two optional food modules [200 kcal (840 kJ) each, with the same macronutrient composition as the total diet] were provided in case subjects were hungry. All food was prepared in the GCRC kitchen at the University of Colorado Health Sciences Center. Body weight was measured daily during this period to confirm weight stability. Subjects were instructed to maintain their normal physical activity and exercise patterns but to refrain from exercise on the day before the calorimeter stay.

**Colorimetry days.** Subjects entered the calorimeter at 0800 and exited at 0700 the next day. The data were extrapolated to 24-h values. On the LI and HI days, stationary cycle exercise was performed at 1000. During all calorimeter days, a standardized walking and stepping protocol was performed each day between 1420 and 1630 to mimic typical activity level outside the calorimeter. Subjects were free to move about the calorimeter during other times of the day, but primarily this time was spent in sedentary behavior (reading, writing, watching television). Subjects were instructed to remain awake and not to nap or perform any exercise other than that prescribed by the protocol. For each subject, meals were the same for each condition and were provided at the same time of day. Subjects were asked to go to bed at the same time during each calorimeter stay. During each stay in the calorimeter, subjects consumed a diet designed to achieve energy balance, as determined from the baseline day. The composition of the diet was 30% of energy from fat, 15% of energy from protein, and 55% of energy from carbohydrate. The energy values of the diets on the exercise days were adjusted (+500 kcal) for the estimated increased EE due to the exercise bouts (400 kcal for exercise + 100 kcal for elevated postexercise EE).

**Measurements**

24-h EE and substrate oxidation. Total daily EE and substrate oxidation were determined from oxygen consumption and carbon dioxide production measured in a whole room calorimeter. Gas concentrations were determined from the flow rate and the differences in carbon dioxide and oxygen concentrations between entering and exiting air by using Hartman and Braun (Frankfurt, Germany) oxygen (Magnos 4 G) and carbon dioxide (Uras 3 G) analyzers. Values were corrected for temperature, barometric pressure, and humidity. Urine was collected for the duration of the calorimetry stay and analyzed for total nitrogen concentration, which was then used to determine 24-h protein oxidation (35). EE and substrate oxidation were calculated from oxygen consumption and RQ based on the equations of Jequier et al. (20). Values for all indexes were averaged over 1-min inter-

**Statistical Analysis**

Statistical analyses were carried out by using SAS (SAS/STAT User's Guide, version 8, 2000, SAS Institute, Cary, NC). Repeated-measures analysis of variance with within-subject factor condition (Con, LI, HI) was carried out by using linear mixed models and a random subject effect (23) with SAS PROC MIXED. These methods provide valid handling of the occasional missing observations (38). Contrasts were used within these models to estimate overall condition effects. The relation between substrate oxidation and skeletal...
muscle enzyme activity was calculated by using Pearson’s coefficient. Results are presented as means ± SE. One subject was excluded from the nutrient oxidation analysis because he did not consume all of the food that was provided during one of the calorimeter stays and was thus in slightly negative energy balance during that condition.

**RESULTS**

During the calorimeter stays, the exercise bouts were performed according to prescription. Mean responses during LI exercise were 42.5 ± 0.3% of VO₂_max for women and 41.6 ± 3.4% of VO₂_max for men. During HI exercise, mean responses were 66.9 ± 1.6% of VO₂_max for women and 67.7 ± 1.3% of VO₂_max for men. Female subjects exercised for 111.9 ± 2.5 min on the LI day and 66.3 ± 4.0 min on the HI day. Values for men were 86.1 ± 4.7 and 49.1 ± 2.2 min, respectively. There were no differences in preexercise glucose, insulin, circulating FFA, or NE levels across conditions. Postexercise, plasma glucose was unchanged, but plasma FFA increased more (P = 0.03) after LI (442.9 ± 78.3 μeq/l) than HI (199.5 ± 75.7 μeq/l). Plasma NE increased after the exercise period (HI > LI = Con; P < 0.0001), but the values were highest after the HI exercise bout (761.5 ± 53.1 pg/ml) compared with LI (432.9 ± 34.6 pg/ml) and Con (406.7 ± 61.1 pg/ml).

**EE**

Absolute 24-h EE (Table 2) was significantly elevated above the Con day on both the LI and HI exercise days (P < 0.001) but was not different between exercise conditions. The average increase in EE was 506 ± 34 kcal/day on the LI day and 546 ± 34 kcal/day on the HI day. As expected, the rate of EE (kcal·kg FFM⁻¹·min⁻¹) during the exercise period was elevated with increased intensity (Con < LI < HI, P < 0.001). Immediate postexercise EE (end of exercise to 1230) was also elevated after LI and HI compared with Con (P < 0.001) but was not different between LI and HI. EE during the remainder of the day (1230 to bedtime) and sleeping periods was unaffected by exercise intensity. Subjects were maintained within a tight range of energy balance. Mean energy balance across conditions was 12.5 ± 29.2 kcal for Con, −6.8 ± 42.7 kcal for LI, and −54.5 ± 24.5 kcal for HI (not significant).

**Nutrient Oxidation**

The 24-h RQ was not affected by exercise intensity (Con = 0.86 ± 0.01, LI = 0.87 ± 0.01, HI = 0.87 ± 0.01; P > 0.05). As expected, RQ during the exercise period was significantly elevated compared with the Con day (Con < LI, P = 0.002; Con < HI, P < 0.0001; LI < HI, P = 0.052). However, subsequent analyses revealed that exercise RQ was greater in men than in women during LI (0.89 ± 0.02 vs. 0.86 ± 0.01) and HI (0.93 ± 0.01 vs. 0.88 ± 0.01). During the immediate postexercise period, RQ was the same after LI and HI exercise but was greater in men than in women across all conditions (Con: 0.86 ± 0.02 vs. 0.80 ± 0.01; LI: 0.89 ± 0.06 vs. 0.78 ± 0.02; HI: 0.86 ± 0.06 vs. 0.79 ± 0.03). RQ during the remainder of the day and during sleep was unaffected by exercise intensity and was not different between men and women.

Because of these sex differences in RQ, we decided to analyze the substrate oxidation data for men and women separately. Carbohydrate oxidation over 24 h (Fig. 1A) was significantly elevated over the Con day on both exercise days (Con < LI = HI) and significantly higher in men than in women (P = 0.035). However, 24-h fat oxidation (Fig. 1B) was unaffected by exercise intensity and was not different between men and women. When the rate of substrate oxidation over 24 h was expressed relative to FFM (mg·kg FFM⁻¹·min⁻¹), there were no sex differences in the rate of carbohydrate oxidation (Fig. 1C), but women had a higher rate (24–56%) of 24-h fat oxidation (Fig. 1D) during all conditions (P = 0.02).

During exercise, absolute carbohydrate oxidation was not different during LI (85.2 ± 4.0 g) and HI (98.8 ± 3.8 g). However, fat oxidation was greater (P < 0.0001) during LI (21.4 ± 1.8 g) compared with HI (11.6 ± 1.7 g). During the postexercise (lunch to bedtime) and sleeping periods, substrate oxidation was unaffected by exercise intensity. When the rates of substrate oxidation were expressed relative to FFM, a greater rate of carbohydrate oxidation was observed during exercise in men (P = 0.03; Fig. 2A), but a greater rate of fat oxidation was observed during exercise in women (P = 0.01, Fig. 2B). Women also sustained a higher rate (P < 0.05) of fat oxidation after exercise (lunch to bedtime) on the control day (1.32 ± 0.13 vs. 0.86 ± 0.18 mg·kg FFM⁻¹·min⁻¹), on the LI day (1.18 ± 0.18 vs. 1.05 ± 0.19 mg·kg FFM⁻¹·min⁻¹), and on the HI day (1.52 ± 0.14 vs. 1.03 ± 0.20 mg·kg FFM⁻¹·min⁻¹).

**Skeletal Muscle Enzyme Activity**

There was no consistent relation between skeletal muscle enzyme activity and 24-h substrate oxidation.

---

**Table 2. Energy expenditure over 24 h and during different segments of the day**

<table>
<thead>
<tr>
<th></th>
<th>Con</th>
<th>LI</th>
<th>HI</th>
</tr>
</thead>
<tbody>
<tr>
<td>24 h, kcal</td>
<td>2,110 ± 75Ψ</td>
<td>2,616 ± 85†</td>
<td>2,656 ± 69†</td>
</tr>
<tr>
<td>Exercise, kcal·kg FFM⁻¹·min⁻¹</td>
<td>0.030 ± 0.001*</td>
<td>0.115 ± 0.003†</td>
<td>0.183 ± 0.006‡</td>
</tr>
<tr>
<td>Immediate postexercise, kcal·kg FFM⁻¹·min⁻¹</td>
<td>0.030 ± 0.001*</td>
<td>0.042 ± 0.004†</td>
<td>0.043 ± 0.002†</td>
</tr>
<tr>
<td>Lunch to bedtime, kcal·kg FFM⁻¹·min⁻¹</td>
<td>0.034 ± 0.001*</td>
<td>0.034 ± 0.001*</td>
<td>0.034 ± 0.001*</td>
</tr>
<tr>
<td>Sleeping, kcal·kg FFM⁻¹·min⁻¹</td>
<td>0.018 ± 0.001*</td>
<td>0.018 ± 0.001*</td>
<td>0.019 ± 0.001*</td>
</tr>
</tbody>
</table>

Values are means ± SE. Con, control; LI, low-intensity exercise; HI, high-intensity exercise. Values with different symbols are significantly different (P < 0.05).
Significant correlations were only found between maximal PFK activity and 24-h fat oxidation on the Con day ($r = -0.55$) and HADH/CS and 24-h fat oxidation on the LI day ($r = -0.54$). We also examined the relation between skeletal muscle enzyme activity and substrate oxidation during sleep and exercise and again found few significant correlations and no consistent relations (data not shown).

Because of the observed sex differences in substrate oxidation, we compared skeletal muscle enzymatic capacity in men and women. Men had significantly greater maximal levels of PFK activity (84.4 ± 4.8 vs. 65.1 ± 9.6 μmol·g⁻¹·min⁻¹; $P < 0.01$), which resulted in a greater PFK/HADH (57.7 ± 10.9 vs. 42.0 ± 5.7; $P = 0.05$). CS activity was slightly higher in women than in men (14.3 ± 5.0 vs. 12.3 ± 1.7 μmol·g⁻¹·min⁻¹; $P = 0.06$). There were no differences between men and women in HADH (1.7 ± 0.9 and 1.8 ± 0.3 μmol·g⁻¹·min⁻¹, respectively) or HADH/CS (0.18 ± 0.05 and 0.13 ± 0.02, respectively).

DISCUSSION

The primary aim of this investigation was to examine the effects of low- and high-intensity exercise on 24-h EE and nutrient oxidation. Exercise intensity had no effect on 24-h EE when the energy cost of the exercise bout was matched. The 24-h EE was increased by a similar amount on the LI and HI compared with the Con day (Table 2). Furthermore, EE in the postexercise period (~10 h) and during sleep was not affected by exercise intensity. Thus we conclude that 24-h and resting EE is not affected by the intensity of acute bouts of aerobic exercise.

The effect of exercise intensity on EE has been controversial. Several studies have shown that, after high-intensity exercise, excess postexercise oxygen consumption is significantly increased over the short term (30 min to a few hours) (12, 26, 33). Some of these studies have not matched the EE of exercise, and many lacked adequate prestudy controls (e.g., exercise the previous day). No previous studies have measured an integrated response of different intensities of exercise, matched for energy expenditure, over a full day (24 h).

In the present study, the EE of exercise was matched and carefully controlled, and subjects were studied under conditions of strict prestudy dietary and physical activity control.

With regard to nutrient oxidation, we observed no differences in 24-h fat oxidation on LI or HI days when exercise was matched for EE. The increase in 24-h EE was primarily supported by an increase in the amount of carbohydrate oxidized. Carbohydrate oxidation was increased by a similar amount on the LI and HI conditions.
pared with the Con day (Fig. 1A), but the amount of fat oxidized was not significantly different between Con, LI, and HI (Fig. 1B). We therefore conclude that exercise intensity has no effect on 24-h fat oxidation. Previous investigations have reported that during recovery from exercise there is a shift toward greater fat oxidation in the postexercise period (2, 19, 40) and that postexercise fat oxidation is greater after higher intensity exercise bouts (2, 26, 27). In the present study, we found no differences in fat oxidation after low- and high-intensity exercise. A likely explanation between the discordant results of the present and previous studies is the experimental conditions. In the present investigation, postexercise measurements encompassed the lunch meal; this was by design, because the calorimeter experiments are designed to mimic a typical free-living day. It is possible that hormonal responses (e.g., elevated insulin levels) attenuated fat oxidation in the postexercise period by blunting lipolysis.

A popular dogma that exists amongst fitness practitioners is that low-intensity exercise will produce a greater amount of fat oxidation compared with higher intensity exercise. Although the proportion of fat oxidation to total EE is greatest at very low exercise intensities (21, 28, 29, 36), the total amount of fat oxidized will be dependent on both the duration and intensity of exercise. At higher exercise intensities, total fat oxidation may be greater because EE is greater. In a practical sense, the amount of time that an individual devotes to exercise on a daily basis could be a limiting factor as to how much exercise an individual does. In this instance, if the desire is to maximize fat oxidation, then the prescribed intensity should be one that maximizes the total amount of fat oxidized and not one that maximizes the relative contribution of fat oxidation to total EE. Furthermore, the prolonged effects of exercise on nutrient metabolism must be understood before solid recommendations can be made on prescribing exercise intensity for body weight and body fat regulation. In light of these findings, we would suggest that the exercise program that best maximizes 24-h fat oxidation is one that produces the greatest amount of EE. Given that time is a limiting factor for most individuals, we would also suggest that, if the goal of exercise is to maximize fat oxidation to better regulate body fat mass, then exercise should be performed at the highest intensity that can be comfortably maintained.

We observed few significant correlations between maximal glycolytic and oxidative enzyme activity and 24-h substrate oxidation, either on the Con day or the exercise days. Zurlo et al. (41) reported a significant inverse correlation between maximal HADH activity and 24-h RQ measured in a whole room calorimeter, suggesting that those subjects who had the highest HADH activity also had the highest rates of 24-h fat oxidation. We did not find a relation between HADH activity and 24-h fat oxidation. The reasons for the discordant findings are not readily apparent. However, we did find a significant negative relation between maximal PFK activity and fat oxidation, suggesting that those with the highest glycolytic capacity had the lowest 24-h fat oxidation rates. Thus our data are somewhat in agreement with those of Zurlo et al. Surprisingly, we observed no consistent relationship between enzymatic profile and substrate oxidation on the exercise days. Perhaps this is due to the wide variability in enzyme activity among individuals (24, 41). Thus we conclude that skeletal muscle enzymatic profile is not strongly related to 24-h substrate oxidation, suggesting that other factors (e.g., circulating hormones) are more important determinants of 24-h substrate oxidation.

Although exercise at low and high intensities had similar effects on 24-h EE and substrate oxidation, there appeared to be subtle differences between the sexes in nutrient oxidation during and after exercise. Although small in magnitude, women sustained a greater rate of fat oxidation well into the postexercise period, as indicated by higher fat oxidation rates in the period from lunch to bedtime. The sex differences in fat oxidation during and after exercise appeared to be greatest during the HI exercise condition. This is consistent with the findings of Horton et al. (19), who observed no sex differences in nutrient oxidation immediately after 2 h of low-intensity (40% \(V_{\text{O}}_{2\text{,max}}\)) cycling. To date, no studies have examined sex differences in substrate oxidation across a range of exercise intensities. Although not an a priori hypothesis, our data would suggest that gender differences are dependent on exercise intensity, and this should be an area of future research.

**Table 3. Correlations between maximal enzyme activity (\(\mu\text{mol} \cdot \text{g}^{-1} \cdot \text{min}^{-1}\)) and 24-h carbohydrate and fat oxidation (mg/kg FFM) on the CON, LI, and HI days**

<table>
<thead>
<tr>
<th></th>
<th>24-h Carbohydrate Oxidation</th>
<th>24-h Fat Oxidation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Con</td>
<td>LI</td>
</tr>
<tr>
<td>PFK</td>
<td>0.10</td>
<td>-0.25</td>
</tr>
<tr>
<td>HADH</td>
<td>-0.07</td>
<td>0.18</td>
</tr>
<tr>
<td>CS</td>
<td>0.10</td>
<td>0.02</td>
</tr>
<tr>
<td>PFK/HADH</td>
<td>0.30</td>
<td>-0.14</td>
</tr>
<tr>
<td>PFK/CS</td>
<td>0.04</td>
<td>0.06</td>
</tr>
<tr>
<td>HADH/CS</td>
<td>-0.21</td>
<td>0.14</td>
</tr>
</tbody>
</table>

PFK, phosphofructokinase; HADH, \(\beta\)-hydroxyacyl-CoA dehydrogenase; CS, citrate synthase. *\(P < 0.05\).
An unexpected finding was that rates of fat oxidation (mg·kg FFM$^{-1}$.min$^{-1}$) were higher in women on the Con day (during the exercise period, during the period from lunch to bedtime, and over 24 h) as well as the exercise days. Subjects adhered to the same prestudy controls, and the timing of meals and percentage of total daily kilocalories were the same for men and women. Thus we cannot speculate on why these sex differences occurred on the Con day. It could be argued that these differences on the Con day make our conclusions of greater rates of fat oxidation in women than men after HI exercise tenuous. However, the differences in fat oxidation between men and women appeared to be greatest on the HI day, although none of the interaction terms achieved statistical significance. We were underpowered to detect gender differences in nutrient oxidation rates, and this remains an area for future investigation.

There has been much speculation regarding the mechanisms contributing to sex differences in nutrient utilization during exercise. Possible mechanisms include differences in nutrient mobilization (7–9), role of the sex steroid hormones (10, 22, 30, 31), or differences in muscle fiber-type and enzymatic profiles (17, 32, 34). With regard to enzymatic capacity, men appear to have a greater glycolytic capacity (17), whereas women have a greater oxidative capacity (32, 34). Consistent with these data, we also found a greater glycolytic capacity (absolute PFK activity, PFK/HADH ratio) in men. However, we observed no sex differences in oxidative capacity (HADH/CS). Our data suggest that the sex differences in fuel metabolism are driven in part by the higher glycolytic capacity of men.

In conclusion, we have found that exercise intensity has no effect on 24-h EE measured by using whole room indirect calorimetry. Furthermore, low- and high-intensity aerobic exercise, matched for energy expended during exercise, have similar effects on 24-h nutrient oxidation. We therefore conclude that low-intensity exercise does not promote greater “fat burning,” as has been popularized among the lay press. Women appear, however, to have slightly greater rates of fat oxidation during and after exercise, and this gender difference may be greatest after high-intensity exercise. This suggests that, as a result of high-intensity exercise, women preferentially rely on fat oxidation, thereby sparing carbohydrate reserves during waking and active periods.

We thank the nurses, laboratory technicians, and kitchen personnel of the GCRC for professional assistance, and the staff of the Clinical Nutrition Research Unit Energy Balance Core Laboratory who managed the whole room indirect calorimeter and monitored the exercise sessions. We are also grateful to Michael Pagliassotti and Yuren Wei for assistance in performing the enzymatic analyses. This work was supported by National Institutes of Health Grants M01 RR-00051, P30 DK-48520, R01 DK-42549, and HL-049331.

REFERENCES


