Lipid oxidation in fit young adults during postexercise recovery

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Kuo, Calvin C., Jill A. Fattor, Gregory C. Henderson, and George A. Brooks. Lipid oxidation in fit young adults during postexercise recovery. J Appl Physiol 99: 349–356, 2005. First published December 10, 2004; doi:10.1152/japplphysiol.00997.2004.—To evaluate the hypothesis that lipid oxidation predominates in postexercise recovery, we examined healthy men (n = 6; age = 21.2 ± 0.6 yr) and women (n = 6; age = 22.8 ± 2.1 yr) during and after two exercise tasks [89 min at 45% and 60 min at 65% of peak rate of oxygen consumption (V\:\O_2\:\text{peak})] as well as a time-matched resting control trial (Con). Exercise bouts were matched for energy expenditure. Respiratory exchange ratios (RER) during exercise at 65% V\:\O_2\:\text{peak} for both men and women (0.95 ± 0.01 and 0.93 ± 0.02) were significantly higher than 45% V\:\O_2\:\text{peak} (0.89 ± 0.01 and 0.86 ± 0.02) and Con trials (0.86 ± 0.01 and 0.86 ± 0.02, respectively). During recovery, for men RER values were 0.78 ± 0.01 and 0.76 ± 0.01 after 45% and 65% exercise, respectively. For women, values were 0.79 ± 0.01 and 0.78 ± 0.01. These were significantly lower than during both the preexercise resting period and the corresponding no-exercise Con period (0.82 ± 0.01 and 0.83 ± 0.01, mean RER for men and women, respectively). Hence, the contribution of lipid oxidation to energy supply increased significantly during recovery compared with preexercise levels, and it was greater after exercise than during the time-matched, no-exercise Con period. It is concluded that, although carbohydrate is the major fuel source during moderate- to high-intensity exercise, 1) there is substantial postexercise lipid oxidation; and 2) lipid oxidation is the same during postexercise recovery whether the relative power output is 45% or 65% of V\:\O_2\:\text{peak} when energy expenditure of exercise is matched.

excess postexercise oxygen consumption; substrate utilization; crosstraining; energy expenditure

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HIGHLIGHTED TOPIC | Role of Exercise in Reducing the Risk of Diabetes and Obesity

Obesity has risen to epidemic proportions (29), and in adults, it is associated with development of Type 2 diabetes and other chronic diseases such as coronary heart disease (CHD) and some forms of cancer (22). As well, the incidence of childhood-onset obesity is rising (30, 36), with an associated rise in prevalence of Type 2 diabetes and CHD risk factors. Although public health officials and health care providers recommend exercise as a component of the treatment for obesity, and exercise training and physical fitness have been shown to benefit overweight individuals (25), lipid oxidation during exercise, especially hard exercise (9), may be insufficient to affect body lipid depots. Thus the effect of exercise on energy expenditure as well as the relative contribution of lipids to energy expenditure during and after exercise is of concern to individuals who desire to maintain a body mass index (BMI) in the desirable range of 19.9–24.9 kg/m² (22) or to lose excess body fat (15).

Whereas extensive studies have focused on the relative partitioning of lipids and carbohydrates (CHO) as fuels during exercise (2, 9, 34), less effort has been aimed at uncovering the metabolic events during postexercise recovery. Results show that the relative contribution of lipids decreases as exercise intensity increases [>60% peak oxygen consumption (V\:\O_2\:\text{peak})] (18). For instance, in healthy young men exercising at 65% V\:\O_2\:\text{peak} for 60 min on a leg cycle ergometer, working muscle respiratory quotient (= carbon dioxide production (V\:\CO_2)/oxygen consumption (V\:\O_2)) approximated unity (i.e., 1.0), net free fatty acid uptake was small, glycerol release approximated zero, and there was no utilization of intramuscular triglyceride content (3). Still, even though participants in that study were encouraged to increase dietary energy intake to maintain body mass, exercise training resulted in body fat loss and accretion of lean body mass. Thus, notwithstanding the preferential use of CHO during moderate- to high-intensity exercise, it is possible to hypothesize that lipid oxidation predominates during recovery, especially after physical exercise leading to glycogen depletion.

Historically, studies of metabolism during postexercise recovery have focused on excess postexercise V\:\O_2 (EPOC, alternatively “oxygen debt”) in terms of explaining lactate-glycogen interconversions (20). From a monumental body of literature dating to the beginnings of work physiology, the magnitude of EPOC is known to be related to the intensity and duration of a physical activity bout (6, 7). And, although it was observed that the respiratory exchange ratio (RER) becomes very low during recovery from prolonged exercise (14), energy partitioning after exercise was largely ignored. More recently, investigators have utilized exercise and EPOC measurements to assess the effects of physical activity on daily total energy expenditure (27, 30). Still, fewer have investigated substrate partitioning during postexercise recovery (1, 4). Moreover, limited data exist on energy substrate partitioning in women during the recovery period (13, 32, 34). Although Kiens and Richter (26) have provided data showing intramuscular triglyceride mobilization in the hours after high-intensity exercise, and Horton et al. (21) showed that lipid oxidation is promoted in men, but not women, during recovery from 2 h of mild- to moderate-intensity (40% V\:\O_2\:\text{peak}) exercise, others (28) have concluded that physical activity bouts employing intensities and durations typically used in physical fitness and body

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weight management programs do not significantly impact lipid oxidation or daily total energy expenditure.

In terms of exercise tolerance and compliance when making physical activity recommendations for fitness or weight loss, issues regarding exercise intensity and duration need to be considered. Previously, Bergman and Brooks (2) showed that trained men can oxidize a slightly higher percentage of lipids than untrained men during mild- to moderate-intensity exercise, but only in the fasted state. Relative differences in substrate partitioning disappeared at high (>60% VO2 peak) compared with low relative exercise intensities or when men were recently fed a CHO-containing meal. As well, in assessing insulin sensitivity in women with Type 2 diabetes after low- (50%) or high- (75% VO2 peak) intensity exercise bouts expending 125 kcal, Braun et al. (5) found low- and high-intensity exercises to yield similar improvements in insulin sensitivity. Hence, on the basis of these previous findings, it was possible to suspect that exercise energy expenditure, rather than exercise intensity or duration per se, was important to consider in assessing the metabolic consequences of an exercise bout.

To evaluate the hypotheses that, in young men and women 1) lipid oxidation predominates during recovery from prolonged submaximal exercises of the type typically employed in fitness and weight loss programs, and 2) RER (VCO2/VO2) values and substrate partitioning during recovery from exercise are similar during moderate- and high-intensity exercises of similar energy expenditures [i.e., >90 min at 45% vs. 60 min at 65% of VO2 peak], we determined pulmonary VO2 and RER in healthy young men and women during and after moderate- and high-intensity exercise bouts. Importantly, a time-matched control (Con) trial involving no exercise was employed, because time of day and eating may have effects that could mask the subtle effects of exercise intensity and duration on energy substrate partitioning during the EPOC period. Results confirm that moderate-intensity, but more prolonged physical activity is more effective than shorter, but more intense exercise bouts in terms of promoting lipid oxidation during activity, but both forms of exercise are equally effective in promoting lipid oxidation if the postexercise period is considered.

**METHODS**

**Subjects.** Twelve healthy, moderately active volunteers (6 men and 6 women), between the ages of 19 and 32 yr, were recruited from the University of California, Berkeley campus and community by posted notices and E-mail. Subjects were considered for the study if they were nonsmokers, were not taking medications, were disease free by physical examination, exercised between 4 and 8 h/wk, were weight stable for the last 6 mo, had under 25% body fat, and had normal lung function as determined by a 1-s forced expiratory volume of 70% or greater. Subjects gave informed consent, and the study protocol was approved by the University of California Berkeley Committee for the Protection of Human Subjects (CPHS 2002-5-54). In this investigation, we utilized healthy young and relatively fit subjects to obtain baseline data for comparison with data to be obtained on obese, diabetic, and older individuals.

**Screening tests.** After medical history and physical examination, subjects performed two progressive exercise tests to reliably assess VO2 peak. Exercise was performed on a leg-cycle ergometer (Ergometric 818E, Monark, Vansbro, Sweden). A continual, progressive protocol was used to determine VO2 peak; subjects pedaled at a self-selected cadence beginning at 1 kp and increased 0.25 or 0.5 kp every 3 min until voluntary cessation. Respiratory gases were continuously monitored via Vista Mini-CPX O2/CO2 analyzer (Vaccum Med, Ventura, CA) and recorded every 30 s by a personal computer. In each trial, the system was calibrated twice before rest and exercise using room air and a certified calibration gas tank (16% O2 and 4% CO2). Heart rate was continuously monitored by a Quinton Q4500 electrocardiograph (Quinton, Bothell, WA) and blood pressure by auscultation using a stethoscope and sphygmomanometer. Values obtained during VO2 peak tests were accepted as maximal if heart rate reached within 10% of predicted maximal value and RER exceeded 1.1. Body composition was determined by skinfold measurements (23, 24).

**Dietary controls.** Participants were instructed to maintain constant diet and physical activity habits throughout the experimental period. Three-day dietary records were completed before each experimental trial to monitor the habitual dietary composition of each subject. During the morning of the first experimental trial, subjects were asked to eat the same breakfast for the remaining two trials. Each trial day, the subjects ate breakfast 2 h before reporting to the laboratory. Analysis of food records was performed using the Nutritionist III program (N-Squared Computing, Salem, OR).

**Experimental trials.** The order of the experimental trials was randomly assigned. Each subject was tested under three conditions: exercise at 65% of VO2 peak (65%) for 1 h; 45% of VO2 peak (45%) for 86–89 min, to obtain an isocaloric expenditure between the two intensities; and a control (Con) trial, where a 60-min resting period was substituted for exercise. Duration of the 45% VO2 peak test was determined through a prediction of caloric expenditure on the basis of corresponding RER values at 45% obtained during the preliminary VO2 peak assessments. Exercise power output was continually adjusted to maintain the desired relative exercise intensity. Each trial consisted of a preexercise resting period of 30 min, the exercise period of 60–89 min, and a 3-h recovery period. During rest and recovery periods, subjects were seated at a table where they were allowed to read quietly. Subjects were transported in a wheelchair to the restroom in between preexercise rest and exercise as well as 120 min into recovery. Furthermore, heart rate and blood pressure were monitored throughout the experimental trial. Subjects were instructed to abstain from strenuous activity 24 h before each experimental trial, and trials were separated by at least 3 days.

In selecting exercise intensities and durations for investigations, we were cognizant of findings in the most recent Institute of Medicine Macronutrient Report (22) in which a minimum of 60 min of exercise most days was recommended for healthy adults to maintain body weights in the healthy body mass index (19.9–24.9 kg/m2) range. However, because the physical activity estimates were based on results of studies using doubly labeled water technology, the results were nonspecific with regard to the intensity or duration of exercise necessary to oxidize body lipid stores. Hence, we decided on a 60-min activity duration as a baseline condition, but we sought to extend that period 50% while maintaining exercise energy expenditure constant.

**Pulmonary gas determinations.** For determination of gas-exchange parameters, expired gas samples were taken at 15-min intervals during preexercise rest, exercise, and recovery. Pulmonary VO2, VCO2, and RER were determined using Vista Mini-CPX O2/CO2 analyzer as during assessments of VO2 peak. During exercise, expired air was constantly monitored for the first 17 min to ensure achievement of the target VO2. Subsequently, breath was sampled for 8 of every 15 min until the end of exercise. At the end of each exercise bout, subjects were instructed to stop cycling and to remain seated on the cycle ergometer for the first 15 min of recovery while expired air was constantly monitored. Afterward, the subject moved to a seat at a nearby table to rest, and again breath was sampled for 8 of every 15 min for time periods 30–180 min of recovery. Respiratory values were averaged over the last 5 min of each time point. Throughout the study period when subjects were not wearing the mouthpiece, they were allowed to drink water ad libitum.

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Table 1. Physical characteristics and dietary data

<table>
<thead>
<tr>
<th></th>
<th>Men (n = 6)</th>
<th>Women (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>21.2±0.6</td>
<td>22.8±2.1</td>
</tr>
<tr>
<td>Height, cm</td>
<td>180±2.6*</td>
<td>160±1.2</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>71.0±4.8*</td>
<td>51.1±1.4</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>8.0±1.1*</td>
<td>16.8±1.5</td>
</tr>
<tr>
<td>Fat-free mass, kg</td>
<td>65.2±4.2*</td>
<td>42.5±1.4</td>
</tr>
<tr>
<td>VO2peak, ml·kg⁻¹·min⁻¹</td>
<td>48.2±4.2</td>
<td>50.5±1.9</td>
</tr>
<tr>
<td>Exercise, h/wk</td>
<td>7.1±1.8</td>
<td>7.3±0.8</td>
</tr>
<tr>
<td>Maximal power output, W</td>
<td>224±11.5</td>
<td>170±14.3</td>
</tr>
<tr>
<td>Maximal heart rate, beats/min</td>
<td>190±3.8</td>
<td>188±3.4</td>
</tr>
</tbody>
</table>

**Calculations.** Energy derived from CHO and lipid oxidation was calculated (6):

\[
\text{Energy from CHO oxidation (kcal/min)} = \left(\frac{\%\text{CHO}}{100}\times\text{VO}_2\right) (5.05 \text{ kcal/l O}_2)
\]

\[
\text{Energy from lipid oxidation (kcal/min)} = \left(1 - \frac{\%\text{CHO}}{100}\right) (\text{VO}_2) (4.7 \text{ kcal/l O}_2)
\]

Energy expenditure (kcal/min) = \left(\frac{\%\text{CHO}}{100}\times\text{VO}_2\right) (5.05 \text{ kcal/l O}_2) + \left(1 - \frac{\%\text{CHO}}{100}\right) (\text{VO}_2) (4.7 \text{ kcal/l O}_2)

where \text{VO}_2 is in liters per minute.

Calculations during the exercise period of the 45% of \text{VO}_2 peak trial were made using the last 60 min of measurements to correspond to the 60-min exercise duration of the 65% of \text{VO}_2 peak and resting control trials.

**Statistics.** Data are presented as means ± SE. Within genders, paired t-tests were used to determine significance of differences between trials for physical characteristics. A two-way ANOVA with repeated measures was used to compare means for RER, absolute CHO, and lipid oxidation values, as well as relative contributions to energy expenditure on SPSS 11.5 Graduate Pack (SPSS, Chicago, IL). Significance of differences over time during exercise and postexercise recovery was determined by using one-way ANOVA with repeated measures. Statistical significance of mean differences was set at \(\alpha = 0.05\).

**RESULTS**

**Subject characteristics.** Physical characteristics of study participants are listed in Table 1. Subjects were weight stable throughout the study period. There were no significant gender differences in age, relative \text{VO}_2 peak, habitual physical activity levels, maximal workload, or maximal heart rate during assessment of \text{VO}_2 peak. However, retrospective analysis showed dietary macronutrient composition differed between genders; women were on a lower fat diet (24.7 ± 1.5%) than men (33.5 ± 2.2% of energy). Nonetheless, study participant dietary energy intake and macronutrient composition did not vary significantly among trials.

**RER and relative exercise intensity.** There were no significant differences in RER during the preexercise resting period among trials for men or women (Fig. 1, A and B, respectively). During exercise, the mean RER was 0.89 ± 0.01 and 0.95 ± 0.01 for 45% and 65%, respectively, for men, and 0.86 ± 0.02 and 0.93 ± 0.02, respectively, for women. RER values during the 65% trial for both men and women were significantly higher than in 45% and Con trials (0.86 ± 0.01 and 0.86 ± 0.02 for men and women, respectively). During postexercise recovery, for men and women, RER values after both exercise trials (0.78 ± 0.01 and 0.76 ± 0.01 for 45% and 65%, mean RER, respectively, for men, and 0.79 ± 0.01 and 0.78 ± 0.01, respectively, for women) were significantly lower than during both the preexercise resting period and the corresponding no-exercise Con period (0.82 ± 0.01 and 0.83 ± 0.01, mean RER for men and women, respectively; Fig. 1, A and B). Despite trends apparent in Figs. 1-3, no differences were found between RER values during the recovery from 45% and 65% trials for either men or women (Fig. 1, A and B); as well no significant gender differences were found (see below).

**Relative substrate oxidation.** The relative contributions of CHO and lipid oxidation to energy expenditure during exercise...
and postexercise recovery are given in Table 2. During exercise, a significantly larger contribution of CHO oxidation was found during the 65% trial than during the other conditions (65% trial: 81.9 ± 4.5 and 74.7 ± 5.3%, 45% trial: 62.5 ± 4.5 and 53.1 ± 5.3%, and Con trial: 52.5 ± 4.5 and 52.9 ± 5.3%, for men and women, respectively). In contrast, a greater contribution of lipid to energy expenditure was found during recovery between 45% and 65% exercise trials. As well, no significant gender differences in postexercise substrate oxidation patterns were found.

**Absolute substrate oxidation.** The contributions of CHO and lipid to energy expenditure during exercise and postexercise recovery are shown in Fig. 2, A and B, for men, and Fig. 2, C and D, for women. The contribution of CHO during exercise was significantly different among trials (P < 0.001) for both men and women. During recovery, lower CHO oxidation was found in trials after exercise (0.39 ± 0.05 and 0.44 ± 0.06 kcal/min for 65% and 45%, respectively, in men; 0.37 ± 0.04 and 0.40 ± 0.03 kcal/min for 65% and 45%, respectively, in women) than in Con (0.72 ± 0.05 and 0.53 ± 0.03 kcal/min for men and women, respectively). Again, no significant differences between exercise conditions were detected in postexercise energy expenditures.

Expressed in either relative (Table 2), or absolute terms (Table 3), men exhibited a greater contribution of lipids to energy expenditure during recovery from exercise than during the resting control period (P < 0.02), but no difference was established between exercise intensities (1.51 ± 0.09 kcal/min for 65% and 1.41 ± 0.10 kcal/min for 45%). In relative terms (Table 2), women exhibited a greater contribution of lipids to energy expenditure during the second and third hours of recovery from exercise than they did during the resting control period (P < 0.05), but we observed no significant differences

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**Table 2. Relative substrate oxidation of energy expenditure**

<table>
<thead>
<tr>
<th>Time</th>
<th>Condition</th>
<th>Men (n = 6)</th>
<th>Women (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CHO</td>
<td>Lipid</td>
<td>CHO</td>
</tr>
<tr>
<td>Pre-Ex</td>
<td>45%</td>
<td>54.0±5.2</td>
<td>46.0±8.1</td>
</tr>
<tr>
<td></td>
<td>65%</td>
<td>52.4±5.2</td>
<td>47.6±3.9</td>
</tr>
<tr>
<td></td>
<td>Con</td>
<td>61.0±5.2</td>
<td>39.0±4.5</td>
</tr>
<tr>
<td>Ex</td>
<td>45%</td>
<td>62.5±4.5</td>
<td>37.3±6.1</td>
</tr>
<tr>
<td></td>
<td>65%</td>
<td>81.9±4.5†</td>
<td>21.6±2.5</td>
</tr>
<tr>
<td></td>
<td>Con</td>
<td>52.5±4.5</td>
<td>52.0±6.1</td>
</tr>
<tr>
<td>Post-Ex 1 h</td>
<td>45%</td>
<td>24.6±4.0*</td>
<td>75.4±4.0*</td>
</tr>
<tr>
<td></td>
<td>65%</td>
<td>21.8±3.7†</td>
<td>78.2±3.7†</td>
</tr>
<tr>
<td></td>
<td>Con</td>
<td>44.2±3.7</td>
<td>55.8±3.7</td>
</tr>
<tr>
<td>Post-Ex 2 h</td>
<td>45%</td>
<td>25.2±4.3</td>
<td>74.8±4.3</td>
</tr>
<tr>
<td></td>
<td>65%</td>
<td>19.0±4.3*</td>
<td>81.0±5.1*</td>
</tr>
<tr>
<td></td>
<td>Con</td>
<td>35.7±4.3</td>
<td>64.3±4.3</td>
</tr>
<tr>
<td>Post-Ex 3 h</td>
<td>45%</td>
<td>20.7±3.2*</td>
<td>79.3±3.2*</td>
</tr>
<tr>
<td></td>
<td>65%</td>
<td>17.1±3.2*</td>
<td>82.9±3.2*</td>
</tr>
<tr>
<td></td>
<td>Con</td>
<td>35.8±3.2</td>
<td>64.2±3.2</td>
</tr>
</tbody>
</table>

Values are means ± SE given in percent; n, no. of subjects. Pre-Ex, preexercise resting period; time (t) = -30 min to t = 0 min; Ex, exercise; t = 15 min to t = 60 min; Post-Ex 1 h, first hour of postexercise recovery, t = 75 min to t = 120 min; Post-Ex 2 h, t = 135 min to t = 180 min; Post-Ex 3 h, t = 195 min to t = 240 min; Con, control trial; 45%, exercise for 90 min. 45% VO2peak. 65%, exercise for 60 min 65% VO2peak. *Significantly different from corresponding Con, P < 0.05. †Significantly different between exercise intensities, P < 0.05.
between experiments in absolute lipid oxidation for women during recovery time periods (Table 3).

**RER and metabolic power output.** Exercise energy expenditures were similar during 45% and 65% exercise trials in both men and women (Table 3). CHO oxidation was significantly greater during exercise in 65% than during 45% in both men and women (Table 3). During the postexercise recovery period in both exercise trials, energy expenditure was the same as during Con (Table 3, Fig. 2, A–D, and Fig. 3, A and B).

**DISCUSSION**

People who are physically active tend to be leaner than sedentary individuals (6, 16, 17), but CHO energy sources (glycogen, lactate, and glucose) are the major fuels during exercise of moderate and greater exercise intensities (2). Physical activity raises total energy expenditure (8) and daily lipid oxidation (4), even though little lipid is used during physical activity (2, 3). Hence, our working hypothesis is that the energy deficit of physical activity results in glycogen depletion and sets into motion signals that permit lipid oxidation to predominate during postexercise recovery. Results of the present investigation are supportive of our hypothesis. Major findings of this investigation are the following: 1) during postexercise recovery, the body shifts to oxidize predominantly lipid for energy, even though CHO energy sources predominated during moderate- to high-intensity physical activity; 2) exercise intensity has no significant effect on substrate oxidation in postexercise recovery when the total energy cost is matched during activities of moderate and greater exercise intensity; and 3) in a resting, postabsorptive person, the relative contribution of lipid to substrate oxidation shifts progressively toward lipid throughout the day. These findings are discussed sequentially.

**RER data indicate predominance of lipid oxidation during postexercise recovery.** Our data corroborate results of previous studies showing that exercise raises metabolic rate and shifts the pattern of whole body metabolism toward lipid oxidation...
during the postexercise recovery period (4, 26, 28, 32). Although we did not sample tissue glycogen contents during our experiments, on the basis of extensive previous work, our data (Table 3) can be interpreted to suggest that muscle and liver glycogen stores are mobilized to support a majority of energy expenditure during exercise. It has been shown that the body prioritizes the resynthesis of glycogen during postexercise recovery (7, 19). The increase in relative lipid contribution during recovery from prolonged exercise in our study is consistent with the idea that the body will increase its lipid oxidation for energy, while sparing CHO to minimize further challenge to glycemia and to facilitate subsequent restoration of glucose homeostasis and glycogen repletion.

A few trends were notable throughout the postexercise recovery period. Although 

\( V_O^2 \) fell rapidly after exercise in both men and women, RER did not drop rapidly until 15–30 min into recovery. However, we observed a postexercise nadir in RER, which stabilized ~30 min into recovery. Also, there were upward spikes during recovery at 210 min, 30 min after subjects were wheelchair transported to and from the restroom. A similar upward spike is noticeable 150 min in the Con trial. These minor spikes associated with physical activity did not significantly alter the relative contribution of lipid and CHO oxidation in recovery because subsequent values decreased to baseline.

After exercise, RER was substantially lower compared with preexercise levels in both intensities (\( P < 0.05 \)); similarities in substrate partitioning during exercise recovery may be attributed to the isocaloric nature of the exercise tasks used. In this regard, results of our investigation are consistent with those of Al Mulla et al. (1), who matched exercise energy expenditure values for 40% and 60% of 

\( V_O^2 \) peak activity bouts for energy cost. Indeed, the present study design is most similar to that of Al Mulla and colleagues, however, they fasted study participants overnight, or possibly longer, making exercise RERs very low (0.79 ± 0.02 and 0.82 ± 0.04 for 40 and 60% exercises, respectively). Likely, Al Mulla and colleagues did not find a shift to lipid oxidation during recovery because the shift had occurred during activity. Additionally, their study lacked a time of day control. As a result, our data can be interpreted to support the assertion that RER decreases after prolonged exercise at low (1, 4) and high intensities (26, 33).

Substrate oxidation rates during exercise and recovery. During exercise, CHO oxidation was significantly greater at 65% than 45%, which is consistent with results of previous studies (2, 34). At the same time, lipids were oxidized to a lesser extent during exercise at the higher (65%) power output. During the 3-h recovery period after 65% exercise, however, lipid oxidation was promoted such that total lipid oxidation during exercise and recovery for the two exercise tasks was not different (Table 3).

Considering exercise plus recovery, lipid oxidation was higher during postexercise recovery than during Con for men (Fig. 2, A and B, and Fig. 3A), but it did not reach significance for women. Results for men were in agreement with previous investigations (1, 4, 26). Similarly, CHO oxidation was significantly lower during postexercise recovery than in Con for men and women, which is consistent with the idea that the body shifts toward lipid oxidation when glycogen preservation or resynthesis becomes a priority.

Lipid oxidation for men during recovery accounted for ~54% of total lipid oxidized throughout the 45% trial and 70% of total lipid oxidation throughout the 65% trial. For women, lipid oxidation in recovery was 46% of total lipid oxidized during the 45% trial and ~60% of total lipid oxidized throughout the 65% trial. Although lipid oxidation occurred at generally higher rates after exercise, total caloric expenditure during exercise activity was about double that of recovery in both men and women. With regard to the harder (65%) exercise trial, it is noteworthy that, whereas CHO oxidation was favored during exercise, total lipid oxidation during the 3-h recovery period was greater than during the exercise itself.

Our study produced an unexpected result in that absolute lipid oxidation in the recovery period for women was not different between 45%, 65%, or Con trials (65% and Con, \( P = 0.07 \); Fig. 2, C and D, and Fig. 3B). Previously, our laboratory has observed relatively greater lipid oxidation in women than men during exercise (16, 17). However, others have also shown that women do not oxidize more lipid during postexercise recovery than during a time-matched resting control trial (21). Participants were asked to sit quietly during resting and recovery periods; however, nonexercise physical activity such as reading was allowed. Fidgeting may have limited contrasts of substrate oxidation during recovery periods between exercise and Con trials (21). However, retrospective analysis of dietary intake data indicated that women were on a relatively low-fat diet (~25% energy from fat), so it is possible that the unexpected difference in dietary macronutrient composition affected energy substrate partitioning during postexercise recovery. Regrettably, also, we did not control for menstrual cycle phase or oral contraceptive use, but these factors have, at best, small effects on lipid metabolism (11, 37, 38).

Irrespective of the two conditions of exercise intensity and duration we studied, results indicate that isoenergetic exercise tasks result in similar elevations in postexercise lipid oxidation. Such knowledge may be useful to practitioners and participants alike in weight management programs. In terms of exercise compliance, harder exercises result in higher levels of perceived exertion, and some participants may be more compliant with exercise prescriptions if easier, but more prolonged, exercise training tasks are practiced, especially when a training regimen is commenced.

Changes in pattern of substrate utilization over time. Data in Fig. 3 show that stable metabolic rates were achieved by men and women after 30 min of rest. However, although metabolic rates were stable during the control trial, relative lipid contribution to energy expenditure increased continuously over the 4.5-h period. Although there was a decrease in absolute CHO oxidation and increase in absolute lipid oxidation, energy expenditure remained constant. It is important to note that during rest, just as during exercise, in fasting persons the body will increase its lipid oxidation over a period of time as endogenous CHO energy sources are reduced and glucose homeostasis becomes increasingly dependent on gluconeogenesis (38). The results obtained were those expected on the basis of extensive previous work. However, the control resting trials were necessary for purposes of comparison with results of exercise trials.

Dietary influences. Application of dietary control is difficult and can influence the results and conclusions. For instance, in our study using two levels of isoenergetic exercise and stan-
standard techniques of indirect calorimetry, we observed exercise to result in increased lipid oxidation during postexercise recovery. In contrast, using two levels of isoinertial exercise (40 and 70% VO₂ peak) and room indirect calorimetry, Melanson et al. (28) did not observe an effect of exercise intensity on either 24-h energy expenditure, lipid oxidation, or CHO oxidation. However, in their trials, subjects were fed an extra 500 kcal to cover the energy costs during (400 kcal) and after (100 kcal) activity. In contrast, we did not allow study participants to eat during the observation period. Overall, we view results of Melanson et al. to be similar to ours but with differences in emphasis and interpretation. Physical exercise results in an EPOC, the magnitude of which depends on the intensity and duration of activity (7, 8). By definition, EPOC implies an increase in energy expenditure over rest. Both Melanson et al. and we report that physical activity results in increments in energy expenditure over sedentary, nonexercise control conditions, but we observed a preferential use of CHO during activity and preferential use of lipid during recovery. However, Melanson et al. conducted a different experiment; the additional nutrition provided to participants by Melanson et al. on activity days (500 kcal, 55% CHO, 30% fat, 15% protein) likely would have influenced the subtle effects of exercise on energy substrate partitioning we observed during the EPOC period.

In the effort to quantify magnitude of lipid oxidation after exercise, like Melanson et al. (28), others (40–42) have also added energy to the diet by providing a postexercise meal. Such treatment is appropriate if one considers that most of us live in a postprandial state. However, matching or surpassing exercise energy output with dietary input masks the effect of exercise on total energy expenditure and postexercise lipid oxidation. Nonetheless, results of such studies serve as an important reminder that the economy of human locomotion is great. Historically, it was probably an advantage that the energy expenditure of physical activity, including that for postexercise recovery, could be accommodated by subtle changes in behavior and relatively minor increments of dietary intake. In contemporary society, however, the lesson may be that both physical activity and dietary guidelines are needed to prevent and manage body fat accretion.

Limitations. There are several limitations to our study. First, the experimental trials were performed on moderately active, but not highly trained individuals. Thus the responses observed may not be representative of either very physically fit or sedentary individuals. Similarly, we studied normal-weight individuals, who may respond differently during exercise and recovery than overweight, obese, or Type 2 diabetic individuals. Next, although there is less of a learning curve for riding a leg-cycle ergometer for 60–90 min than running, the use of a cycle ergometer with friction braking may have led to variances in substrate oxidation. Furthermore, length of the recovery period led to extraneous movements from subjects, who were reminded to remain still during the periods of quiet rest. Thus we suspect that fidgeting, especially in the Con trial, minimized opportunity to find significant contrasts between energy expenditures or substrate utilization patterns during recovery periods from exercise and time-matched Con conditions (27).

As well, we did not assess or control for the possible effect of menstrual cycle phase (10, 38) or oral contraceptive use (11) on substrate utilization and energy expenditure (12, 37). Additionally, beyond broad categories of CHO and lipid, the precise identities of fuels oxidized could not be determined. For example, we could not differentiate between intramuscular triglycerides, free fatty acids, and lipoproteins. Similarly, we could not discriminate among CHO sources such as oxidized muscle glycogen, blood or muscle lactate, or blood glucose. Importantly, we did not investigate the potential effects of meal size, composition, and timing on lipid oxidation during postexercise recovery. Like physical activity, as well as by definition, nutrition is known to affect metabolism. And, as already indicated, differences in dietary macronutrient composition between men and women may have affected results. Finally, we acknowledge that, given intersubject variability and the small number of study participants, our study may have lacked power to document small (e.g., gender-specific) effects, whereas more robust (e.g., exercise) effects could be identified as different.

Although simple in design, results of the present investigation serve as a reminder that the energy expenditure of exercise includes components of both exercise and recovery. Although the elevation in energy expenditure during recovery is typically significantly less than that during activity, there occurs a shift in the pattern of endogenous substrate utilization during recovery such that it represents a period of predominant and substantial lipid oxidation. And, whereas catabolic processes predominate during activity, anabolic processes predominate during recovery (6). This knowledge is important not only at a basic level but also it offers possibilities for the design of different postactivity interventions to benefit diverse populations ranging from obese individuals to athletes.

Summary. Although CHO is the major fuel source of exercises requiring moderate and greater exercise intensities (9, 17, 18), 1) there is substantial postexercise lipid oxidation, and 2) lipid oxidation is the same during postexercise recovery whether the relative workload was moderate (45% VO₂ peak) or hard (65% VO₂ peak) when energy cost of exercise is matched. These results are supportive of the hypothesis that physical exercise is useful in the mobilization and utilization of lipid energy deposits in normal weight, healthy young men and women. Some lipid is oxidized during physical activity itself, especially if exercise is of moderate intensity and duration is prolonged (Table 3) (9). However, moderate and greater exercise intensities set into play mechanisms that shift the pattern of substrate utilization away from CHO-derived energy sources toward lipid so that lipid becomes the predominant energy source during recovery (Fig. 2). Hence, on the basis of evidence in this experiment, it is reasonable to conclude that physical activity raises total energy expenditure, resulting in some lipid oxidation during activity, especially if intensity is mild to moderate, and, moreover, that it shifts the pattern of substrate utilization during postexercise recovery when lipids become predominant energy sources.

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