Impact of high-intensity exercise on energy expenditure, lipid oxidation and body fatness

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OBJECTIVE: Two studies were conducted to assess the potential of an increase in exercise intensity to alter energy and lipid metabolism and body fatness under conditions mimicking real life.

METHODS: Study 1 was based on the comparison of adiposity markers obtained in 352 male healthy adults who participated in the Québec Family Study who either regularly participated in high-intensity physical activities or did not. Study 2 was designed to determine the effects of high-intensity exercise on post-exercise post-prandial energy and lipid metabolism as well as the contribution of β-adrenergic stimulation to such differences under a real-life setting.

RESULTS: Results from Study 1 showed that men who regularly take part in intense physical activities display lower fat percentage and subcutaneous adiposity than men who never perform such activities, and this was true even if the latter group reported a lower energy intake (917 kJ/day, P < 0.05). In Study 2, the high-intensity exercise stimulus produced a greater post-exercise post-prandial oxygen consumption as well as fat oxidation than the resting session, an effect which disappeared with the addition of propranolol. In addition, the increase in post-prandial oxygen consumption observed after the high-intensity exercise session was also significantly greater than that promoted by the low-intensity exercise session.

CONCLUSION: These results suggest that high-intensity exercise favors a lesser body fat deposition which might be related to an increase in post-exercise energy metabolism that is mediated by β-adrenergic stimulation.


Keywords: high-intensity exercise; energy and lipid balance; propranolol

Introduction

Nowadays, many health professionals are reluctant to include physical activity as a therapeutic approach to help obese individuals cope with the difficulties of achieving an energy deficit or even energy balance, because it is believed to impact on energy balance negligibly. In this context, it should be noted that some conditions have to be met for exercise to influence energy balance. Accordingly, it is of importance to note that physical activity when coupled to a healthy diet can lead to a difference in energy balance of as much as 7 MJ per day when compared to a day during which individuals are sedentary and have access to a mixed diet.¹

These data are in agreement with the fact that when individuals perform physical activity in a context where high-fat foods are given to them in the post-exercise period, the outcome is a positive energy balance while under a low-fat diet, a substantial energy deficit can be reached with exercise participation.²,³ Thus, in a context where dietary intake is not manipulated properly, a very probable scenario for the physically active individual is weight stability or even weight gain.

In the same order of idea, the modalities of the exercise prescription also have to be optimized for it to exert its full potential on energy and lipid balance. Increasing exercise intensity might be relevant because such an approach would seem to lead to a better control of energy balance. Indeed, it would seem that in the post-exercise period, high-intensity exercise seems to inhibit energy intake to a greater extent than a low-intensity exercise session of the same caloric cost.⁴ Moreover, post-exercise oxygen consumption as well
as energy expenditure are greater after high- than low-intensity exercise. Although high-intensity exercise does not seem to acutely generate a greater post-exercise fat oxidation in vivo, the chronic influence of such an approach might lead to a different outcome. In this context, it is important to note that individuals who engage in higher intensity activities also demonstrate lower subcutaneous adiposity than individuals who never take part in such activities. It has also been demonstrated that a high-intensity intermittent training program has a greater potential to mobilize lipid than a lower intensity continuous program of approximately twice the energy cost, an effect which might be mediated by the increased potential of skeletal muscle to utilize lipids under such conditions. Also of interest is the possible implication of high-intensity exercise in the maintenance of a reduced body weight after a weight-reducing program. It would seem that increasing exercise intensity is an effective strategy for maintaining weight stability in the reduced-obese state.

The aim of the present study was thus to confirm earlier findings which support the notion that individuals who take part in higher-intensity exercise are generally leaner than those who never do (Study 1) and to also further investigate, under conditions mimicking real life conditions, how high-intensity exercise has the potential to acutely alter energy expenditure and lipid utilization (Study 2).

Methods
Study 1

Subjects. This study is based on the analysis of data obtained in 352 male healthy adults who participated in the Québec Family Study from 1978 to 1982. Their descriptive characteristics are presented in Table 1. Before the study, each subject was fully informed about all experimental procedures, which received the approval of the Laval University Medical Ethics Committee. The written consent of each subject was obtained prior to the admission in the study.

Measurements. Habitual energy and macronutrient intakes were determined using a 3 day dietary record as previously described. Before reporting food intake, subjects received detailed instructions on how to record the nature and quantity of ingested foods. Following the completion of the diary, the subjects were measured in the laboratory for anthropometry and body composition. Data were subsequently coded and energy and macronutrient contents of food were calculated with Canadian Nutrient File. The reliability of the food intake assessment procedure has been previously tested in our laboratory and was found to be satisfactory. The dietary journal was completed during two week days and one weekend day. Subjects were requested to report their food intake on the same days as those during which they recorded their habitual physical activities.

A physical activity record was used to evaluate physical activity participation. Briefly, this diary required reporting the main activity performed during each of the 96 periods of 15 min on a testing day. Activities were coded from 1 to 9 depending on their intensity, corresponding to a range of 1–7.8 METs (1–7.8 times estimated basal energy expenditure) and more.

Skinfold thicknesses were measured with a Harpenden caliper at six sites (biceps, triceps, medial calf, subscapular, abdominal, and suprailiac) following the procedures described by the International Biological Program. We also derived the three following additional indicators of subcutaneous adiposity: the sum of the six above-referenced skinfolds, the sum of trunk skinfolds (sum of subscapular, abdominal, and suprailiac), and the sum of extremity skinfolds (sum of biceps, triceps, and medial calf).

Body density was also measured in a subgroup of men by using the hydrostatic weighing technique. Percentage body fat was estimated from body density after correction for residual lung volume by using the helium dilution technique previously described by Meneely and Kaltreider.

Statistical analysis. A Student’s t-test was used to compare values of subjects reporting at least one 15 min period of activity coded as 9 and referred to as high-intensity exercise participants (HIEP) to those of reference subjects (RS) who did not report this level of activity. The Pearson correlation coefficient was calculated to quantify the associations between exercise intensity (number of 9 scores in the subgroup reporting this level of activity) and nutritional variables or morphological indicators. All statistical analyses for Study 1 were performed using the SAS statistical package. Differences were considered to be statistically significant at P < 0.05.

Study 2

Subjects. Eight moderately trained male subjects volunteered to participate in this study. All subjects were nonsmokers and healthy, as indicated by a medical history and physical examination. Participants were asked to maintain their regular dietary habits and to consume no alcohol and no caffeine the day before the investigation. Strenuous physical activity was not allowed for 2 days before each experimental session. The written consent of each subject was obtained prior to admission in the study. This study received the approval of the Laval University medical ethics committee. Subjects’ characteristics are presented in Table 3.

Pre-experiment testing. Maximal aerobic power (O_{max}) was assessed using a standardized progressive treadmill protocol (treadmill, Q-65 Quinton, Seattle, WA; electrocardiograph, Q4000 Quinton, Seattle, WA) previously established in our laboratory. An open-circuit gas analysis system was used to measure oxygen uptake (O_2, S-3A Applied Electro Chemistry, Sunnyvale, CA), CO_2 production (ANARAD, International Journal of Obesity
Sunnyvale, CA), respiratory quotient (RQ) and ventilation (Spirometric Module KL Engineering, CA). The maximal oxygen uptake was defined as the highest \(O_2\) recorded during the test for 1 min. We followed the criteria that are generally used in the laboratory to determine whether a subject had reached \(VO_{2max}\). These criteria are: (1) a final respiratory quotient (RQ) > 1.15; and (2) \(O_2\) consumption increased by < 2 ml/kg with an increase in exercise intensity.

Body density was determined by the underwater weighing technique\(^{21}\) and percentage body fat was derived from body density.\(^{22}\) Pulmonary residual volume was measured using the helium dilution method (Spiroflow MORGAN, Chatham Kent, UK).\(^{23}\) Fat mass was derived from percentage body fat while fat-free mass was calculated as total body weight minus fat mass.

**Experimental protocol.** The experimental protocol was designed to determine the effects of high-intensity exercise on post-exercise post-prandial energy and lipid metabolism as well as the contribution of \(\beta\)-adrenergic stimulation to such differences under a real-life setting. Figure 1 presents a diagram of the experimental protocol of Study 2. Each session began at about 7:30 am, ie when subjects took a standardized breakfast at home by following a predetermined menu of foods containing 700 kcal (18, 34 and 48% energy from protein, lipid and carbohydrate, respectively) as previously described.\(^4\) Upon arrival at the laboratory at about 8:30 am, electrodes were placed on the chest to obtain an electrocardiogram and heart rate (HR) during exercise. A catheter was inserted into a forearm vein for blood sampling. Subjects rested for at least 30 min on a reclining chair and, at 9:30 am, RMR was measured for 15 min with an open-circuit gas analysis system (Hartmann & Braun Uras 10E, Bartlesville; KL Engineering Volume Ventilation measuring system S-430 with a turbine flow transducer K520-C521, California) through a mouthpiece while a clip was fixed on the nose. A blood sample was obtained at 9:50 am, and the subject then began one of the following four randomly assigned conditions: (1) resting session; (2) exercise session at 38% \(VO_{2max}\) (low-intensity exercise session (LIES)); (3) exercise session at 77% \(VO_{2max}\) (high-intensity exercise session (HIES)); or (4) exercise session at 77% \(VO_{2max}\) followed by the oral administration of propranolol. Under the propranolol condition, subjects were administered a nonselective beta-adrenoceptor blocker (propranolol, 80 mg) immediately after the exercise session. The workload during all three exercise sessions was monitored through heart rate, which was derived from the \(VO_{2max}\) test. Calorimetric and HR measurements were performed at every 15 min period for 5 min during exercise sessions by using the same equipment as for the \(VO_{2max}\) test and at every 15 min period for 10 min during the resting session with the same equipment used for the RMR measurement. The energy equivalent of \(O_2\) at the target exercise intensity during the \(VO_{2max}\) test was used to determine the exercise duration necessary to expend 500 kcal during exercise sessions. The mean values of exercise intensity and duration, energy expenditure and \(RQ\) during the exercise sessions are presented in Table 4. It is also important to note that as a result of the experimental design, the time elapsed between the controlled breakfast and the end of exercise sessions was approximately 30 min longer after the low-intensity exercise session than after the high-intensity exercise sessions. After all four sessions, subjects took a shower and were instructed to keep water temperature and shower duration as constant as possible. Resting indirect calorimetry and HR measurements were sampled at 30 and 60 min after each session. A blood sample was taken 60 min after each session (rest or exercise), prior to the ingestion of a 900 kcal meal (19, 36 and 45% energy from protein, lipid and carbohydrate, respectively). Thereafter, blood samples were taken at 90, 150, 210 and 270 min after each session and calorimetric and HR measurements were performed at the same interval. Blood samples were then centrifuged to obtain plasma samples which were stored at \(-20^\circ\)C until analysis of plasma FFA concentrations and at \(-80^\circ\)C for plasma catecholamine assays.

Substrate oxidation rate was estimated from RMR and \(RQ\) using the tables of Lust\(^{25}\) by assuming that the relative contribution of protein to energy expenditure was 10%. Plasma FFA concentrations were measured using an enzymatic assay (NEFA C, Wako Kit Pure Chemical Industries Ltd, Osaka, Japan). Plasma catecholamine concentrations were determined using high-performance liquid chromatography. Plasma epinephrine and norepinephrine were extracted on alumina and their concentrations determined using an electrochemical detector (ESA Coulonorm II) after a high performance liquid chromatography (HPLC) separation of both catecholamines on a C\(_{18}\) reverse-phase HPLC column.

**Figure 1** Diagram of the protocol. RMR = resting energy expenditure; HR = resting heart rate; and same = same measurements as time 60.
3,4-Dihydroxybenzylamine hydrobromide (DHBA) served as internal standard. A volume of 20 μl alumina eluate samples was injected in a system. The mobile phase which was circulated in the HPLC system included NaH₂PO₄ (0.5 M), EDTA (0.1 mM), sodium octane sulfonic acid and methanol (8%). The separation of the catecholamines was achieved onto a C₁₈ reverse-phase HPLC column.

**Statistics.** A two-way analysis of variance (ANOVA) with repeated measurements was used to determine the effects of beta-adrenoceptor blockade and time as well as of the beta-adrenoceptor blockade-time interaction effect on dependent variables. Contrast analysis was applied to determine which conditions were significantly different from each other when the ANOVA revealed a statistically significant effect. Since the effects of oral propranolol are predominantly observed after 2 h following its administration, the results were analyzed from 150 to 270 min. In order to further document the effects of exercise intensity, a two-way ANOVA was also performed on the whole data set (from 60 to 270 min) while considering values of the resting session, LIIS and HIES-placebo, thus excluding values of the HIES-propranolol. When this ANOVA revealed a significant effect, contrast analyses were performed to determine which conditions were significantly different from each other.

**Results**

Table 1 presents descriptive characteristics of the subjects in Study 1. HIEP were significantly younger than RS. Body weight and height were not statistically different between groups. As expected, FFM in the HIEP group was significantly greater than that observed in the RS. Although fat mass tended to be greater in the RS group, this difference did not reach statistical significance. Other indicators of adiposity, ie percentage body fat, the sum of three, six as well as extremity subcutaneous skinfolds, were all significantly greater in the RS than in the HIEP group.

Depicted in Table 2 are values related to estimated energy and macronutrient intake of subjects who were included into Study 1. These results demonstrate that the RS values for these variables, be it for energy intake (kcal/day), protein (g/day) or carbohydrate (g/day), were all significantly lower in this group. Daily lipid intake also tended to be lower in the RS group than in the HIEP one, but this difference failed to reach statistical significance.

The characteristics of subjects who participated in Study 2 are presented in Table 3 while parameters of exercise testing are shown in Table 4. As reported in this table, the exercise intensity of the two different sessions was 38 vs 77% of VO₂max and the energetic equivalent of these sessions was 500 and 499 kcal, respectively. As expected, the duration of the low-intensity exercise session was greater than the high-intensity exercise session due to the fact that the same amount of calories had to be dissipated under both experimental conditions.

**Table 2 Estimated energy and macronutrient intake of subjects in Study 1**

<table>
<thead>
<tr>
<th>Variables</th>
<th>High-intensity exercise participants (n = 71)</th>
<th>Reference subjects (n = 281)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy intake (kJ/day)</td>
<td>11482 ± 2256</td>
<td>10565 ± 2415*</td>
</tr>
<tr>
<td>Protein intake (g/day)</td>
<td>101.0 ± 22.5</td>
<td>93.3 ± 23.9*</td>
</tr>
<tr>
<td>Lipid intake (g/day)</td>
<td>108.8 ± 32.8</td>
<td>101.4 ± 32.2</td>
</tr>
<tr>
<td>Carbohydrate intake (g/day)</td>
<td>306.5 ± 67.1</td>
<td>275.4 ± 71.8*</td>
</tr>
</tbody>
</table>

Values are means ± s.d. *Significant difference between high-intensity participants reporting at least one period of activity coded as 9 and those not reporting such activities (P < 0.05).

**Table 3 Characteristics of subjects in Study 2**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean ± s.d.</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>26.6 ± 3.1</td>
<td>23 - 31</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>73.3 ± 9.9</td>
<td>59.5 - 88.8</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>176.2 ± 5.9</td>
<td>166.9 - 183.6</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>10.6 ± 5.8</td>
<td>4.18 - 15.23</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>65.7 ± 8.9</td>
<td>55.3 - 84.0</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>13.7 ± 6.7</td>
<td>5.4 - 25.0</td>
</tr>
<tr>
<td>VO₂max (ml kg⁻¹ min⁻¹)</td>
<td>54.3 ± 3.7</td>
<td>46.4 - 57.4</td>
</tr>
</tbody>
</table>

Values are means ± s.d.

**Table 1 Descriptive characteristics of subjects in Study 1**

<table>
<thead>
<tr>
<th>Variables</th>
<th>High-intensity exercise participants</th>
<th>Reference subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>43.1 ± 4.3 (n = 71)</td>
<td>44.5 ± 5.3* (n = 281)</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>74.6 ± 10.4 (n = 71)</td>
<td>75.5 ± 11.3 (n = 281)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>25.2 ± 2.9 (n = 71)</td>
<td>25.5 ± 3.3 (n = 281)</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>15.5 ± 8.1 (n = 43)</td>
<td>17.8 ± 7.0 (n = 191)</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>59.9 ± 6.7 (n = 43)</td>
<td>57.5 ± 6.3* (n = 191)</td>
</tr>
<tr>
<td>Percentage body fat (%)</td>
<td>19.8 ± 7.4 (n = 43)</td>
<td>23.1 ± 6.7* (n = 191)</td>
</tr>
<tr>
<td>Sum of six skinfolds (mm)</td>
<td>66.3 ± 22.8 (n = 71)</td>
<td>75.8 ± 28.9* (n = 281)</td>
</tr>
<tr>
<td>Sum of three trunk skinfolds (mm)</td>
<td>45.4 ± 17.1 (n = 71)</td>
<td>52.3 ± 21.8* (n = 281)</td>
</tr>
<tr>
<td>Sum of three extremity skinfolds (mm)</td>
<td>21.0 ± 7.4 (n = 71)</td>
<td>23.5 ± 8.7* (n = 281)</td>
</tr>
</tbody>
</table>

Values are means ± s.d. *Significant difference between high-intensity participants reporting at least one period of activity coded as 9 and those not reporting such activities (P < 0.05).
Table 4 Characteristics of each exercise session in Study 2

<table>
<thead>
<tr>
<th>Variables</th>
<th>Rest</th>
<th>Low-intensity</th>
<th>High-intensity</th>
<th>High-intensity (with propranolol)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intensity (% VO₂(max))</td>
<td>8 ± 1</td>
<td>38 ± 2</td>
<td>77 ± 6</td>
<td>77 ± 6</td>
</tr>
<tr>
<td>Duration (min)</td>
<td>67 ± 11</td>
<td>65 ± 9</td>
<td>33 ± 6</td>
<td>33 ± 6</td>
</tr>
<tr>
<td>Energy expenditure (kcal)</td>
<td>109 ± 13</td>
<td>500 ± 2</td>
<td>499 ± 4</td>
<td>502 ± 4</td>
</tr>
<tr>
<td>Respiratory quotient</td>
<td>0.82 ± 0.01</td>
<td>0.87 ± 0.04</td>
<td>0.96 ± 0.02</td>
<td>0.94 ± 0.04</td>
</tr>
</tbody>
</table>

Values are means ± s.d.

Figure 2 presents the impact of this experiment on heart rate, O₂ consumption and energy expenditure. Following the high-intensity exercise session (HIES), heart rate was significantly greater than under the low-intensity exercise session (LIES), the propranolol session or the control session. Heart rate was also significantly greater after the LIES than after the propranolol session. No significant differences in energy expenditure were observed between any of the conditions, despite the fact that this variable tended to be higher following the HIES condition when compared to all other experimental conditions. In contrast, oxygen consumption was significantly greater in the HIES when compared to the LIES, or to the resting condition.

As shown in the first panel of Figure 3, RQ was the highest following the resting condition and this value was significantly different from the values observed under the HIES. However, no significant differences in RQ were observed between the HIES, the LIES and the propranolol session, although for the latter two RQ was slightly more elevated than under the control session when the values obtained 150–270 min after each session were considered. Fat oxidation tended to be greater after the HIES condition, but differed only statistically from the resting condition. Carbohydrate oxidation was greater under the HIES, LIES and propranolol sessions than under the control session (results not shown).

No significant difference was observed for all experimental conditions regarding epinephrine concentrations (results not shown). On the other hand, FFA concentrations were significantly greater after the HIES than under the LIES or than the control session. Circulating plasma FFA were also more elevated under the HIES than under the propranolol session, but this difference failed to reach statistical significance.

The comparison of dependent variables for the whole data set (60–270 min) was performed between values of the resting session, the LIES and the HIES-placebo exercise session, thus not including the HIES-propranolol condition. These analyses revealed a significant effect of exercise for heart rate (P < 0.05), O₂ consumption (P = 0.05), RQ (P = 0.07), fat oxidation (P = 0.05), carbohydrate oxidation (P = 0.07) as well as for circulating plasma free-fatty acids (P < 0.05). When using a contrast analysis to perform a posteriori comparison of two specific sessions, these differences persisted between the resting and the two exercise sessions. In addition, a significant difference was also observed between the HIES and LIES for postexercise resting O₂ consumption and heart rate.

Discussion

These studies were performed to assess the potential of vigorous physical exercise to promote lipid utilization under free-living conditions. In this sense, our results support our hypothesis since in a first study we demonstrated that individuals who perform vigorous physical activities on a regular basis are leaner than individuals who never take...
part in such activities despite the fact that energy intake of the RS was lower than the HIEP. Moreover, we also demonstrated that in a real-life setting, after having taken a shower and a meal, a high-intensity exercise stimulus tends to generate a greater oxygen consumption and lipid oxidation than a low-intensity stimulus in the post-exercise-postprandial state, an effect which might be mediated by β-adrenergic stimulation.

In a first paper documenting the effects of high-intensity exercise on body fatness,12 we have demonstrated that individuals who engage in vigorous physical activities are leaner than individuals who never take part in such activities. These earlier findings are also in accordance with other recent results from our group which demonstrated that preadolescent males who exercised vigorously were leaner than those who did not perform such activities.26 Hence, results from Study 1 confirm our earlier findings. Furthermore, the observations from the present study are reinforced by the fact that energy and macronutrient intakes were lower in the RS group. Hence, it would seem that under a real-life setting, high-intensity exercise has the potential to facilitate the control of lipid balance and may contribute to reduced accumulation of body fatness over time. These observations are also in accordance with earlier results from our group which demonstrated that a 15-week high-intensity intermittent training protocol induced a significantly greater weight loss than a 20-week moderate intensity continuous exercise program of almost twice the energy cost.13 They are also consistent with results from our most recent clinical trial that demonstrated that reduced-obese individuals who adhered to a rather high-intensity prescription were able to maintain their body weight and even accentuate fat loss.14 This is also in agreement with recent results from McGuire et al,15 who reported that one of the characteristics of individuals who maintained their weight after a very substantial weight loss is that they engaged in regular vigorous physical activities.

It has been reported that exercise-trained individuals display an increased resting energy expenditure27,28 and resting fat oxidation.29,30 Although some studies have also reported carryover effects of an exercise intervention on resting31 and basal metabolic rate,32 others failed to confirm these findings when measuring sleeping metabolic rate.33,34 Beyond these observations, it would seem that an exercise challenge has the potential to acutely impact on energy metabolism in the post-exercise period. In fact, it is a well-known fact that exercise generates an excess in post-exercise oxygen consumption,8,35 an effect which might be substantiated by an increase in exercise intensity.3,7 Our results add to the body of existing knowledge on the influence of high-intensity exercise over energy metabolism, that is, how this type of exercise influences energy metabolism related parameters in a real-life setting, ie after a shower and a meal. It can undoubtedly be argued that what we are measuring is confounded by the intake of the meal and the shower which has the potential to promote cold-induced thermogenesis. The reader has to bear in mind that the objective of these studies was mainly to derive clinical applications for these findings which could then be used as tools to enhance the potential of exercise to influence the control of energy and lipid balance.

We also wanted to verify whether the administration of a non-selective β-blocker could abolish the differences observed between HIEP and the control session when propranolol was administered after exercise. For most of the measured variables this strategy brought the levels of the variables measured after the high-intensity exercise session to values quite comparable to the effects observed following
the lower-intensity exercise and resting sessions, even if epinephrine levels were not different when all conditions were compared. One of the possible explanation for the increased fat oxidation under the HIES condition when compared to the resting session might be related to the increase in circulating plasma FFA under these conditions. Indeed, an increased gradient of circulating FFA might promote greater lipid utilization.36,37 In this sense, since glycercol levels (a lipolytic index) during exercise are predicted by plasma noradrenaline46 and that SNS activation has been shown to be related to exercise intensity,39 the greater fat oxidation following the high-intensity session might be indirectly related to the greater SNS activation during this type of exercise, which ultimately produces increased levels of circulating FFA in the postexercise-postprandial condition. Another possibility for the increased contribution of fat to the production of energy in response to exercise might be related to the status of glycogen stores following exercise. In this regard, it has been shown that exercise can favor an increase in post-exercise fat utilization when there is some carbohydrate store depletion.40,41 We have also demonstrated that when carbohydrate depletion during a moderate-intensity exercise session is totally compensated in the post-exercise period, differences in post-exercise substrate oxidation are abolished.42 Since HIES promotes a greater carbohydrate utilization, one might thus realistically infer that the increased fat oxidation observed after HIES is simply the consequence of a greater impact of this type of exercise on glycogen stores.

In summary, Study 1 suggests that high-intensity exercise has the potential to chronically influence lipid balance by favoring a lesser body fatness. Moreover, exercise also acutely increases postexercise energy metabolism-related variables, an effect which seems to be substantiated by increased exercise intensity under a real-life setting.

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References


