Substrate oxidation and energy expenditure in athletes and nonathletes consuming isoenergetic high- and low-fat diets

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ABSTRACT Changes in substrate oxidation with isoenergetic high-carbohydrate (HC) and high-fat (HF) diets in male nonathletic subjects, aerobically trained athletes, and weight-trained athletes were examined in a crossover study. A whole-room respiration chamber was used to measure 24-h energy expenditure (EE) and substrate oxidation with control, HC, or HF diets for 7 d. The nonathletic group had higher 24-h EE (P < 0.05), exercise EE (P < 0.03), and resting metabolic rate (P < 0.04) than did the aerobically trained athletes when these measurements were corrected for lean body mass. Fat oxidation was significantly correlated with lean body mass and diet. However, athletic status had no effect on substrate oxidation. Carbohydrate oxidation across groups increased acutely by 23% after 24 h of the HC diet (P < 0.0001). Carbohydrate balance increased significantly over time with the HC diet (P < 0.002) and decreased acutely after return to the control diet (P < 0.0001). With the HF diet, carbohydrate balance increased and was significantly different from balance with the control diet by day 7 (P < 0.03). Fat balance decreased significantly with both the HF (P < 0.04) and HC (P = 0.0075) diets by day 7. Carbohydrate oxidation correlated with carbohydrate intake with both the control (r = 0.61, P < 0.01) and HC diets (r = 0.59, P < 0.02), but not the HF diet. Fat oxidation was not correlated with fat intake. In conclusion, substrate oxidation in a respiration chamber is significantly affected by diet, but not by prior athletic training. Am J Clin Nutr 1998;67:405–11.

KEY WORDS Substrate oxidation, respiratory quotient, whole-room indirect calorimetry, 24-h energy expenditure, men, high-carbohydrate diet, high-fat diet, lean body mass, athletes

INTRODUCTION Maintenance of body fat stores is related to energy and substrate balances (1, 2). According to this hypothesis, if energy expenditure (EE) equals energy intake, then substrate balances are zero. When energy balance occurs, balance of substrate oxidation will follow. If energy balance is not met, changes in body weight and body composition will occur.

An increased carbohydrate content in the diet leads to increased carbohydrate oxidation (3). The tight control of carbohydrate oxidation in relation to carbohydrate intake is probably due to the limited carbohydrate storage capacity of liver and muscle, and the exclusive role of carbohydrates as a source of energy for the brain. Carbohydrate oxidation appears to be better regulated than fat oxidation (4) and is dependent on carbohydrate intake (4–6). A similar increase in fat oxidation with increased fat in the diet is supported in some studies (7) but not in others (4, 8–10). This raises the possibility that whereas immediate corrective responses to changing carbohydrate intake exist, the inability to correct fat oxidation in response to an increase in dietary fat may be one key to the development of obesity.

Physical activity increases total substrate flux through the body (1). With prolonged aerobic activity, there is an increase in the proportion of fat used as fuel, whereas the proportion of carbohydrate used is reduced (11, 12). Conversely, weight training is thought to use mainly carbohydrates as fuel because the bouts of activity are usually short and intense. However, regardless of the type of exercise, total substrate flux increases with exercise (2).

The purpose of this study was to examine changes in substrate oxidation in response to experimental manipulations of dietary fat and carbohydrate content in three groups of male subjects: nonathletic (NA) subjects, aerobically trained (AT) athletes, and weight-trained (WT) athletes. We hypothesized that 1) changes in carbohydrate oxidation would be more responsive to changes in carbohydrate intake than changes in fat oxidation would be to changes in fat intake and 2) substrate oxidation in athletes would differ from that in nonathletes.

SUBJECTS AND METHODS Subjects Seventeen healthy, young, nonsmoking men were recruited for the study. Six were in the NA group, six were in the AT group, and five were in the WT group. The subjects had normal blood lipid concentrations and blood pressure and were not taking any medications. They were required to pass criteria established for each group before being accepted into the study. The

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NA subjects were not involved in any type of regular exercise program before or during the course of the study. The AT athletes were engaged in a minimum of 1.5 h of aerobic exercise three times per week, whereas the WT athletes did an equal amount of weight training each week. Additionally, the WT athletes were required to pass a grip strength test with a hand dynamometer established by the Canadian Public Health Association. The subjects were asked to maintain their normal level of activity during the study but to refrain from strenuous activity for 24 h before insulin sensitivity assessment and for 36 h before the stay in the whole-room respiration chamber. During the chamber stay, all subjects walked on a treadmill at 3.2 km/h for 1.5 h. The athletes were asked to limit their activity to the stated protocol. All subjects signed an informed consent form before participating in the study and the study was approved by the Louisiana State University Institutional Review Board.

Study design

We used a randomized, crossover design to compare the effects of high-fat (HF) and high-carbohydrate (HC) diets on substrate utilization in athletes and nonathletes. Both dietary regimens consisted of a 5-d stabilization period during which subjects consumed the control diet (days −5 to −1), a 7-d period during which subjects consumed either the HF or HC diet in random order (days 1–7), and 1 d on which subjects again consumed the control diet (day +1). There was a 2-wk washout between diet regimens. EE and substrate oxidation for 24 h were assessed with use of a whole-room respiration chamber on days 1, 1, 7, and +1 for both regimens. The subjects were free-living and carried out their normal activities except before and during the days spent in the respiration chamber. A detailed and standardized schedule was maintained in the respiration chamber, with specific meal and sleep times and two 45-min walking sessions on the treadmill (M9.4 Electronic Treadmill; Precor Incorporated, Bothell, WA) at 3.2 km/h with no incline.

Diets

All diets were prepared in a metabolic kitchen. The control diet contained 40% fat, 45% carbohydrate, and 15% protein. The experimental diets were either high in fat (50% fat, 35% carbohydrate, and 15% protein; HF diet) or low in fat (20% fat, 65% carbohydrate, and 15% protein; HC diet). Diet composition was determined by computerized diet analysis. Energy intake was calculated initially as 1.5 × resting metabolic rate (measured with a metabolic cart) and was adjusted to maintain weight during the first control diet period. Once a weight-maintaining energy level was determined for each subject, this energy level was maintained regardless of diet. The three diets included foods that were typical of the region and consisted of a 4-d menu rotation. Subjects were told to consume only food provided by the metabolic kitchen during the study and at least two meals per day were eaten on the premises of the research center during the week. Weekend meals and lunches were packed for the subjects to take home.

Energy expenditure and substrate oxidation

Total 24-h EE, respiratory quotient (RQ), and 24-h substrate oxidation were determined from 0800 to 0700 the next day in a whole-room respiration chamber. The metabolic chamber is a 3 × 3.7 × 2.4 m room with a total volume of 27000 L. It is furnished with a folding bed and chair, toilet facilities, television, VCR, telephone, radio, treadmill, and intercom. Subjects were monitored periodically with a television camera connected to the control room and nurses’ station. Food and drink were passed into the chamber via air locks. The accuracy and precision of chamber measurements were determined with weekly propane tests. During the period of the study, oxygen recovery averages ranged from 95.6% to 99.3% with an SD of 2.4%; carbon dioxide recovery averages ranged from 96.1% to 99.1% with an SD of 2.5%. Therefore, oxygen error ranged from 0.7% to 4.4%, whereas carbon dioxide error ranged from 0.9% to 3.9%. The raw data for the subjects were corrected by using the propane test recovery rate temporally closest to the subject’s test day.

Sleeping EE was defined as the lowest sustained metabolic rate between 0200 and 0500 and exercise EE was calculated during the two 45-min treadmill sessions. The air flow rate was maintained at 60 L/h. Gas exchange was integrated over 1-h periods and values for 23 h were interpolated to 24 h. The oxygen concentration of the outgoing air sample was measured with a Magnos 4G magnetopneumatic oxygen analyzer (Applied Automation Inc, Bartlesville, OK). The carbon dioxide concentration of the sample was measured with an Uras 3G NDR industrial photometer (Applied Automation Inc). Substrate oxidation was calculated from oxygen and carbon dioxide concentrations of the air samples.

Corrections were made for changes in urinary nitrogen before calculation of the nonprotein RQ. So that this correction could be made, subjects collected all urine while in the chamber. Urinary nitrogen was then analyzed with an Antek 703C chemiluminescent nitrogen system (Antek Instruments, Houston). Metabolic rate and substrate utilization were computed as described by Acheson et al (5).

Resting metabolic rate and body-composition assessment

Resting metabolic rate was measured using a ventilated hood indirect calorimetry system (Sensormedics 2900Z metabolic cart, SensorMedics Corp, Anaheim, CA). A fraction of the air flowing through the system was analyzed for oxygen and carbon dioxide concentrations by zirconium oxide and infrared analyzers, respectively. The instruments were calibrated before each measurement of resting metabolic rate as follows: room air was first flushed through the system for 30 s. Span gases (span 1: 26% O2, 0% CO2; span 2: 16% O2, 4% CO2) were then passed through the analyzer for 20 s and calibration tables were created based on the barometric pressure, temperature, and humidity present during the calibration. Room air was then reanalyzed by using the corrected tables and span gases were verified. The flow meter was calibrated with a 3-L calibration syringe, correcting for temperature and pressure.

Before resting metabolic rate was measured, subjects were prohibited from exercising for 24 h. The subjects rested on a bed for 30 min in the morning after a 12-h fast. A clear ventilated hood was placed over the subject’s head for sampling respiratory gases at 1-min intervals for 30 min. Room air was drawn through the hood at a flow rate of ≈40 L/min. The 1-min REE measurements were averaged from all steady state measurements (minimum of 20 min) and used in the analyses. Lean body mass (LBM) and fat mass in the three groups were assessed by a dual-energy X-ray absorptiometry (QDR2000; Hologic Inc, Waltham, MA) (13).
Data analysis
Statistical analyses were done on a personal computer with SAS for WINDOWS, version 6.10 (SAS Institute Inc, Cary, NC). Repeated-measures analysis of variance using the maximum likelihood method to estimate differences between means (proc mixed) was used to assess effects of diet (control, HC, and HF) and group (AT, NA, and WT) on the outcome variables. In the repeated-measures analysis there was one independent variable (group) and the repeated variable was day (diet). Multiple comparisons were performed with Tukey’s test and appropriate adjustments to significance level were made. All results are reported after LBM as a covariate was adjusted for (14). Diet-order effects were also examined to determine whether assignment to an HF or HC diet first influenced the data. Because analyses showed there were no effects of diet order, which was randomized, results shown do not include diet order as a covariate. An alpha < 0.05 was considered significant.

RESULTS
Subject characteristics at baseline are shown in Table 1. Age was not significantly different among groups, but weight (P < 0.04), body mass index (BMI; P < 0.04), and percentage body fat (P < 0.03) were. WT athletes had the highest BMIs and LBM, whereas AT men were the leanest, having significantly less body fat than the other two groups (P < 0.03). Body weight did not change during the study in any group (data not shown).

Energy expenditure
All EE measurements are reported both with and without adjustment for LBM. Because LBM correlated significantly with 24-h EE (r = 0.89, P < 0.0001) as expected, WT athletes had higher unadjusted 24-h EE (Figure 1; P < 0.01) and exercise EE (P < 0.05) than did AT or NA subjects. The amount of energy expended in all activities (eg, exercise, fidgeting, eating, and dressing) during the day was not significantly different among groups for either unadjusted (AT: 2.87 ± 0.28 MJ/d; NA: 2.69 ± 0.40 MJ/d; and WT: 3.40 ± 0.45 MJ/d) or adjusted data (not shown). The proportion of energy expended in activity did not differ among groups or among days (AT: 13.5%; NA: 13.4%; and WT: 13.9%). There were also no significant differences in sleep EE among groups (AT: 7.07 ± 0.13; NA: 7.33 ± 0.17; and WT: 6.97 ± 0.20 MJ).

After LBM was corrected for, several measures were unexpectedly higher in the NA men than in the AT men. (There were no significant differences between NA and WT subjects for the adjusted data.) NA men had significantly higher 24-h EE and exercise EE than the AT athletes (11.24 ± 0.34 compared with 10.36 ± 0.19 MJ, P < 0.05, and 17.5 ± 0.5 compared with 15.4 ± 0.4 kJ/min, P < 0.03, respectively; Figure 1). Subtraction of exercise EE from total 24-h EE resulted in a significantly higher value in NA than in AT subjects (9.70 ± 0.22 compared with 9.04 ± 0.19 MJ, respectively, P < 0.05). Resting metabolic rate measured with the metabolic cart was significantly higher in the NA men than in the AT men (8.40 ± 0.34 compared with 7.37 ± 0.24 MJ, respectively, P < 0.04).

A training effect was observed in the NA men even with the small amount of activity required during the chamber stays. EE during exercise decreased in the NA group from 16 ± 1.4 kJ/min on the first day of the study to 15 ± 1.2 kJ/min on the last day of the study (P < 0.05). Diet had no effect on any component of EE in any of the groups.

Respiratory quotient
There were no significant differences among the three groups in any aspect of substrate balance; therefore, the results are shown collapsed across groups for RQ, substrate oxidation, and substrate balance. There was a highly significant overall difference in RQ between the HF and HC diets (P < 0.0001), with RQ being significantly higher with the HC diet (Table 2). RQ increased acutely when subjects switched from the control diet to the HC diet (P < 0.0001) and also decreased significantly when subjects returned back to the control diet (day +1, P < 0.002). RQ did not change significantly between the control and HF diets.

Fat and carbohydrate oxidation
As a covariate, LBM had a significant effect on fat oxidation, with more fat being oxidized in individuals with more LBM (P = 0.0001). There were no significant differences in fat oxidation by athletic status. Diet, however, continued to have a significant effect on fat oxidation (P < 0.0001), even after LBM was corrected for.

There was a significant difference among diets in carbohydrate oxidation (Figure 2). Significantly more carbohydrate was oxidized by all groups with the HC diet than with the HF or control diets (P < 0.0001) as expected, WT athletes had the highest BMIs and LBM, whereas AT men were the leanest, having significantly less body fat than the other two groups (P < 0.03). Body weight did not change during the study in any group (data not shown).

### Table 1

<table>
<thead>
<tr>
<th>Group</th>
<th>Age</th>
<th>Weight</th>
<th>BMI</th>
<th>LBM</th>
<th>Body fat</th>
<th>Energy intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aerobically trained men</td>
<td>22.8 ± 1.6</td>
<td>70.8 ± 2.2</td>
<td>22.2 ± 0.9</td>
<td>61.7 ± 1.6</td>
<td>11.2 ± 0.8</td>
<td>12.46 ± 0.33 (2966 ± 80)</td>
</tr>
<tr>
<td>Nonathletic men</td>
<td>23.8 ± 1.5</td>
<td>72.8 ± 2.5</td>
<td>23.1 ± 0.7</td>
<td>56.5 ± 2.0</td>
<td>20.6 ± 1.9</td>
<td>11.20 ± 0.67 (2666 ± 160)</td>
</tr>
<tr>
<td>Weight-trained men</td>
<td>26.2 ± 3.3</td>
<td>86.8 ± 6.8</td>
<td>25.9 ± 1.0</td>
<td>69.2 ± 3.0</td>
<td>18.5 ± 3.9</td>
<td>14.28 ± 0.75 (3400 ± 179)</td>
</tr>
<tr>
<td>Overall</td>
<td>24.1 ± 1.2</td>
<td>76.3 ± 2.7</td>
<td>23.6 ± 0.6</td>
<td>62.1 ± 1.7</td>
<td>16.6 ± 1.6</td>
<td>12.55 ± 1.83 (2988 ± 438)</td>
</tr>
</tbody>
</table>

1 x ± SE. Means in a column with different superscript letters are significantly different, P < 0.05.
had significantly higher 24-h EE than AT or NA men (the top panel is shown uncorrected data for each chamber day. WT men fat and high-carbohydrate diets. Diet periods are shown connected. In nonathletic (NA) subjects, and weight-trained (WT) athletes eating high-carbohydrate intake with the control (HC) diet (day 1, 0.33, NS). When the diet was switched back to the control diet back to the control diet, balance decreased significantly compared with day 1 when subjects switched back to the control diet (day +1: 88.10 ± 12.74 g/d, P < 0.05). During the HC diet period, there was a stepwise increase in carbohydrate balance from the control diet to the last day of the HC diet (39.05 ± 16.12 g/d on day 1, P < 0.0005). During the HC diet period, there was a stepwise increase in carbohydrate balance from the control diet to the last day of the HC diet (39.05 ± 16.12 g/d on day 1, P < 0.0005) and was significantly increased compared with day 1 when subjects switched back to the control diet (day +1: 88.10 ± 12.74 g/d, P < 0.03). During the HC diet period, there was a stepwise increase in carbohydrate balance from the control diet to the last day of the HC diet (39.05 ± 16.12 g/d on day 1 to 111.45 ± 18.53 g/d on day 7, P < 0.002). When subjects switched from the HC diet back to the control diet, balance decreased significantly (day +1: 44.61 ± 22.05 g/d, P < 0.0001). Adjusting for LBM did not influence these results.

With the HF diet, there was a significant difference in fat intake from the first day of the HF diet (day 1, P < 0.0001). When they switched from the HC diet back to the control diet (day +1), fat oxidation increased significantly (P < 0.005), reaching 96% of the baseline value within the 24-h period. There was no correlation between dietary fat intake and fat oxidation with the control (r = −0.32, NS), the HF (r = 0.15, NS), or the HC diet (r = 0.19, NS).

**Substrate balances**

Carbohydrate balance (intake minus oxidation) decreased significantly after the control diet on the first day of the HF diet (123.77 ± 21.99 g/d on day −1 compared with 32.12 ± 11.03 g/d on day 1, P < 0.05; Figure 2). Carbohydrate balance increased during the HF diet to 73.40 ± 16 g/d on day 7 (NS) and was significantly increased compared with day 1 when subjects switched back to the control diet (day +1: 88.10 ± 12.74 g/d, P < 0.05). During the HC diet period, there was a sharp change in fat balance when subjects switched from the control diet to the HC diet. Fat balance decreased both acutely (from 38.26 ± 7.35 g/d on day −1 to 12.94 ± 8.34 g/d on day 1, P < 0.005) and chronically (to 2.09 ± 8.43 g/d on day 7, P < 0.005). When the diet was switched back to the control diet on the last study day, a similar acute change in fat balance was seen but in the opposite direction: fat balance increased significantly to 37.69 ± 13.08 g/d (P < 0.0009).

**TABLE 2**

<table>
<thead>
<tr>
<th>Table: Effect of diet on respiratory quotient (RQ)</th>
<th>RQ</th>
<th>FQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>High-fat diet</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C, day −1</td>
<td>0.822 ± 0.006&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>0.85</td>
</tr>
<tr>
<td>HF, day 1</td>
<td>0.847 ± 0.005&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.82</td>
</tr>
<tr>
<td>HF, day 7</td>
<td>0.823 ± 0.007&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.82</td>
</tr>
<tr>
<td>C, day +1</td>
<td>0.843 ± 0.007&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>0.85</td>
</tr>
<tr>
<td>High-carbohydrate diet</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C, day −1</td>
<td>0.866 ± 0.007&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.85</td>
</tr>
<tr>
<td>HC, day 1</td>
<td>0.909 ± 0.008&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.91</td>
</tr>
<tr>
<td>HC, day 7</td>
<td>0.895 ± 0.009&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.91</td>
</tr>
<tr>
<td>C, day +1</td>
<td>0.866 ± 0.012&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.85</td>
</tr>
</tbody>
</table>

<sup>1</sup> x ± SE. Means in a column with different superscript letters are significantly different, P < 0.01. FQ, food quotient; C, control diet; HF, high-fat diet; HC, high-carbohydrate diet.
substrate balances during days of the HF diet. A trend for increased fat oxidation (from day 1 to day 7) with the HC diet also supports the idea that energy balance to zero, counterbalancing the small positive balances during free-living periods also showed positive energy balances during calorimetry days.

The Flatt (2) hypothesis predicts that, during exercise, differences in RQ may occur at different levels and types of exercise training. However, we did not observe any group differences in RQ in this study. Physically active individuals were predicted to have greater fat oxidation and hence lower RQs than nonathletes. During long aerobic activity, RQ is reduced as a result of relative depletion of carbohydrate and increased use of fatty acids for fuel (11, 12). In contrast, during weight training, which involves short bouts of activity, more carbohydrate is used, leading to higher RQs. Although prior athletic training had no significant effect on substrate oxidation or substrate balance in this study, there was a trend \( P = 0.09 \) toward a group effect on fat oxidation.

One explanation for the lack of group difference in RQ may be that the subjects were basically sedentary when the measurements were taken, an artificial situation for the athletes. Perhaps if the athletic subjects had continued their normal amount of physical activity in the chamber we would have detected differences between their substrate oxidation rates and those of the nonathletes. Unfortunately, we could not perform calculations of RQ during the exercise bouts because of the large chamber volume and reduced sensitivity of this measure at short intervals (ie, minutes).

In the present study, the 5 free-living days brought the subjects’ energy balance to zero, counterbalancing the small positive balances during the chamber stays. Zero energy balance during free-living days is supported by the lack of weight gain with the two diet regimens and the significant increase in fat oxidation on the first and last days of the HF diet. A trend for increased fat oxidation (from day 1 to day 7) with the HC diet also supports the idea that energy balance
was zero. At the same time, carbohydrate oxidation was adjusted downward during the week with both diets. This resulted in decreased fat balance and increased carbohydrate balance over the 7 d (Figure 2). This seems to indicate that glycogen stores were not filled to capacity with either diet over the 5 free-living days. However, with overfeeding during the chamber days, glycogen stores probably increased as a result of reduced activity, resulting in increased carbohydrate oxidation and reduced fat oxidation. This result is reflected in higher RQs after the subjects switched from the control diet to the HF diet during the second chamber day (Table 2).

The finding of higher RQs agrees with other chamber studies (17, 22) that have shown increased carbohydrate oxidation and reduced fat oxidation during overfeeding (20). This shift in oxidation occurs even when the proportion of energy from fat is increased during the overfeeding period. The slight overfeeding in the chamber may also have masked any difference in substrate oxidation that might have occurred among groups had the energy balance been zero during the chamber days.

Although we found no significant group differences in fat oxidation, overall fat oxidation was significantly associated with LBM, being higher in men with more LBM. Presumably, individual variability, the relatively small subject number, and overfeeding during the chamber stay may have masked any significant difference that might have occurred among the three groups of men. There was a strong tendency toward a group difference in fat oxidation ($P = 0.09$), consistent with the observed effects of LBM.

Diet, on the other hand, had a major effect on fat oxidation. However, shifts in carbohydrate oxidation in response to diet appeared to be greater than changes in fat oxidation, confirming some previous reports (8). This was supported by the results of correlational analyses, which showed a significant positive relation between carbohydrate intake and oxidation but not between fat intake and oxidation. On the basis of previous chamber studies (4, 6, 9, 10, 19–23), we anticipated differences in substrate oxidation that were due to diet.

With the HF diet, fat balance remained positive on day 7 after 5 free-living days, supporting the observation that HF diets promote obesity (8, 23) and lead to body-composition changes to match fuel intake (2). When the subjects switched back to the control diet, however, the high level of fat oxidation continued, resulting in a near zero fat balance (Figure 2). Thus, the higher rate of fat oxidation with the HF diet continued for 24 h after the diet was switched to one with less fat. The subjects adapted to each diet by reducing carbohydrate oxidation and increasing fat oxidation during the relatively short 7-d diet periods. Adjustment to different compositions of dietary macronutrients occurs in the first 2–3 d, with nearly complete adjustment after 5 d (24). The results from this study show that in healthy young men, fuel oxidation shifts both acutely and chronically to approximate the macronutrient composition of the diet. There were no differences in any aspect of substrate balance in AT athletes, WT athletes, or NA men by group.

One limitation of our study was the small number of subjects in each group. Although this is typical of respiration chamber studies, which are time consuming and resource intensive, data from a limited number of subjects require caution in interpretation. We performed a priori power analysis for 24-h EE and observed significant group effects for this variable. To our knowledge, there have been no previous 24-h metabolic studies using weight-trained athletes or trained athletes as subjects in the chambers. Thus, it was difficult to power the study, particularly for the substrate oxidation variables. Post hoc power analysis indicated that a larger number of subjects would be required to detect significant differences in fat oxidation among groups; however, the differences observed are within the range of biologically significant differences obtained in other studies (ie, 20–30 g) and were of borderline significance ($P = 0.09$).

In conclusion, increased total LBM appeared to have a significant effect on fat oxidation. Additionally, diet had a major effect on substrate oxidation, whereas prior athletic training had no effect on substrate oxidation in the controlled environment of the respiration chamber. Although exercise (ie, the free-living component of this study) is an important regulator of energy balance by allowing greater expenditure of energy, there did not appear to be any effects of chronic exercise training on substrate oxidation in this study.

We are grateful to the research volunteers and to the clinical staff at the Pennington Biomedical Research Center for their assistance during this study. In particular, we thank Jana Ihrig for coordinating the subjects’ clinical assessments, Marlene Afton for preparing the diets, and Mark Klemperer for assisting with the respiration chambers.

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