Twelve weeks of moderate aerobic exercise without dietary intervention or weight loss does not affect 24-h energy expenditure in lean and obese adolescents\textsuperscript{1–4}

Gert-Jan van der Heijden, Pieter JJ Sauer, and Agneta L Sunehag

ABSTRACT

Background: Exercise might have a persistent effect on energy expenditure and fat oxidation, resulting in increased fat loss. However, even without weight loss, exercise results in positive metabolic effects. The effect of an aerobic exercise program on 24-h total energy expenditure (TEE) and its components—basal (BEE), sleep (SEE), and awake sedentary (SEDEE)—energy expenditure and substrate oxidation—has not been studied in lean and obese adolescents.

Objective: The objective was to test the hypothesis that 24-h energy expenditure and fat oxidation increase in lean and obese adolescents after 12 wk of moderate aerobic exercise without dietary intervention and weight loss.

Design: Twenty-eight postpubertal Hispanic adolescents [mean ± SE: 13 lean (age: 15.3 ± 0.3 y; body mass index (BMI; in kg/m\textsuperscript{2}): 20.2 ± 0.7; body fat: 18.7 ± 1.6%) and 15 obese (age: 15.6 ± 0.3 y; BMI: 33.1 ± 0.9; body fat: 38.1 ± 1.4%)] completed a 12-wk aerobic exercise program (4 × 30 min/wk at ≥70% of VO\textsubscript{2peak}) without weight loss. Energy expenditure and substrate oxidation were quantified by 24-h room calorimetry at baseline and post-exercise.

Results: This aerobic exercise program did not affect 24-h TEE, BEE, SEE, or SEDEE in lean or obese participants. In obese adolescents, 24-h RQ and RQ during SEE decreased (both \(P < 0.01\)) and fat oxidation increased (\(P < 0.01\)).

Conclusions: A 12-wk aerobic exercise program did not increase TEE, BEE, SEE, or SEDEE in either lean or obese sedentary adolescents. Furthermore, 24-h fat oxidation did not change in the obese adolescents, whereas it increased in the lean adolescents. Am J Clin Nutr doi: 10.3945/ajcn.2009.28686.

INTRODUCTION

Efficient and well-accepted strategies to prevent or delay obesity-related morbidity in children and adolescents are much needed, particularly in minority groups (1–3). Most studies on the outcome of interventions include both exercise and weight loss. Because weight loss is difficult to achieve and maintain in adolescents, we wanted to determine whether an exercise program alone (ie, without weight loss) would be well accepted by sedentary lean and obese Hispanic adolescents and whether it would result in reduced insulin resistance and fat deposits associated with obesity-related disorders (eg, visceral and hepatic fat content). We recently reported that a moderate 12-wk aerobic exercise program without dietary intervention and weight loss increased hepatic and peripheral insulin sensitivity in both lean and obese participants (4). In addition, the program decreased visceral and hepatic fat content in the obese adolescents (5). An important part of the study was to determine whether any of the components of 24-h total energy expenditure (TEE) [basal (BEE), sleep (SEE), and awake sedentary (SEDEE)] and/or substrate oxidation rates are affected by exercise itself (ie, without weight loss) and, if so, whether these changes are related to any specific improvements in metabolic and/or fat distribution.

Numerous studies have addressed energy expenditure and substrate oxidation (fat oxidation in particular) during and after individual exercise bouts and in relation to various exercise programs. Some studies have shown increased resting energy expenditure (REE) in response to exercise (6–9), whereas others have not found any effects (10). These differences might be explained by type, duration, and intensity of the exercise and whether lean body mass increased or not. It has been shown that energy expenditure can remain increased for 36 to 48 h after an exercise bout (11). Thus, the timing of measurements in relation to the exercise session might also be of importance (11). Similarly, the effect of exercise on fat oxidation has been shown to depend on intensity, duration, and age and body composition of the participants. In this regard, it has been reported that low-to-moderate intensity exercise increases fat oxidation more than does high-intensity aerobic training (12, 13); the maximal rate of

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fat oxidation occurs at a lower percentage of peak oxygen volume (VO$_2$ peak) in postpubertal than in pubertal and prepubertal children (14, 15); training increased resting fat oxidation in lean adults (16–18), whereas its influence on resting fat oxidation in overweight and obese adults was inconclusive (6, 7, 19–21). In general, the referenced studies indicate the potential of exercise to increase energy expenditure and fat oxidation. Therefore, we hypothesized that our 12-wk moderate exercise program would increase TEE, BEE, SEE, and SEDEE and increase fat oxidation.

To our knowledge, there are no published reports on 24-h TEE and its components, BEE, SEE, SEDEE, and substrate oxidation in adolescents in response to an exercise program. In fact, most studies conducted in adults measured energy expenditure and/or substrate oxidation using indirect calorimetry over shorter periods of time (7, 8, 10, 12, 21–24). Thus, the present study has the potential to address several of these gaps in our knowledge.

SUBJECTS AND METHODS
Participants
After approval of the protocol by the Baylor College of Medicine Institutional Review Board for Human Subject Research and the General Clinical Research Center Advisory Board, obese and lean adolescents were recruited by local advertisement. Adolescents were screened and enrolled in the study after written assent from the participant and consent from the legal guardian were obtained.

The calorimeter measurements were part of an extensive study on the metabolic effects of aerobic exercise (4, 5). Twenty-eight postpubertal Hispanic adolescents (Tanner stages IV–V), 13 lean and 15 obese, were studied (Table 1). The lean participants had a body mass index (BMI) less than the 85th and the obese had a BMI greater than the 95th percentile for age according to Centers for Disease Control and Prevention growth charts (25). However, BMI might not be an optimal marker of leanness/obesity (26). Thus, to ensure that our lean participants were indeed lean, not only with regard to BMI criteria but also with regard to fatness, they must have <27% body fat as measured by dual-energy X-ray absorptiometry (Table 1). The participants had been lean or obese for ≥5 y and reported stable body weight for ≥6 mo. Only sedentary adolescents were included, i.e., they did not participate in any school or after school organized athletic activities and performed <45 min light-to-moderate physical activity weekly.

All participants were Hispanic (parents and grandparents of Hispanic descent by self-report) and in good health as determined by a medical history, a physical examination, and a standard blood chemistry analysis including blood lipid measurement, liver and kidney function tests, and measurements of hemoglobin, hematocrit, hemoglobin A$_1c$, and fasting and 2-h postprandial glucose response. Participants were not taking medications including birth control pills and had no first-degree relatives with diabetes. Adolescents with morbid obesity (body fat >50%, sleep apnea, Pickwick syndrome, or cor pulmonale) were excluded.

Study design
Each participant was studied on 2 separate occasions: 1) the weekend before the start of the exercise program (baseline), and 2) the weekend after the final exercise session of the 12-wk program (postexercise). All procedures were identical on both study occasions.

The participants were admitted to the Metabolic Research Unit (MRU) at the Children’s Nutrition Research Center, Houston, TX, where 24-h calorimeter measurements were performed to assess energy expenditure and substrate oxidation rates (27–29).

To exclude effects of dietary intake on measurements obtained at baseline and postexercise, each participant received, before both studies, an identical low-carbohydrate, high-fat diet (30% carbohydrate, 55% fat, and 15% protein; 20% of the total carbohydrate content as fructose) for 7 d (27–29). Total energy

### TABLE 1
Participant characteristics

<table>
<thead>
<tr>
<th></th>
<th>Lean participants</th>
<th>Obese participants</th>
<th>P for interaction$^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Postexercise</td>
<td>Baseline</td>
</tr>
<tr>
<td>No. of subjects</td>
<td>9/4</td>
<td>9/4</td>
<td>7/8</td>
</tr>
<tr>
<td>(male/female)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (y)</td>
<td>15.2 ± 0.3</td>
<td>15.5 ± 0.3</td>
<td>15.6 ± 0.3</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>56.2 ± 2.7</td>
<td>56.8 ± 2.8</td>
<td>89.8 ± 3.2$^3$</td>
</tr>
<tr>
<td>BMI (kg/m$^2$)</td>
<td>20.7 ± 0.7</td>
<td>20.3 ± 0.7</td>
<td>33.1 ± 0.9$^3$</td>
</tr>
<tr>
<td>Lean body mass (kg)</td>
<td>44.0 ± 2.3</td>
<td>45.2 ± 2.5$^4$</td>
<td>53.6 ± 2.8$^4$</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>10.5 ± 1.0</td>
<td>10.4 ± 1.0</td>
<td>34.0 ± 1.4$^4$</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>18.7 ± 1.6</td>
<td>18.2 ± 1.6</td>
<td>38.1 ± 1.4$^4$</td>
</tr>
<tr>
<td>VO$_2$ peak (L/min)</td>
<td>2.15 ± 0.15</td>
<td>2.48 ± 0.12$^4$</td>
<td>2.49 ± 0.15</td>
</tr>
</tbody>
</table>

$^1$ All values are means ± SEs. VO$_2$ peak = peak oxygen consumed.

$^2$ General estimating equations were used to determine potential interactions between the lean and obese participants with regard to the effect of exercise. When a significant interaction was found between the lean and obese groups, post hoc procedures provided by general estimating equation were used to compare groups at baseline and postexercise and to assess the effect of the exercise program within each group. When there was no interaction (ie, the response to exercise did not differ between the groups) but both groups showed a decrease or increase in the measured variable, combined statistics were used to determine the significance level.

$^3, 5$ Significantly different from lean participants: $^3P < 0.01$. $^5P < 0.05$.

$^4, 6$ Significantly different from baseline within each group: $^4P < 0.01$. $^6P < 0.05$. 

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**Subjects and Methods**

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intake was calculated to correspond to each individual’s requirement according to the Institute of Medicine Dietary Reference Intakes (30). The food was delivered to the participants’ homes by the metabolic research kitchen. Nonconsumed food was returned and examined for constituents, and the energy and macronutrient contents of the consumed food were calculated by difference (27–29). All meals served during the calorimeter study had a composition identical to that of the 7-d standardized diet consumed at home before the baseline and postexercise studies, except that total energy intake was reduced by \( \approx 5\% \) to adjust for a slightly lower activity level in the calorimeter. To determine the effect of exercise alone, participants were told not to make lifestyle changes and to maintain their habitual diet, except adherence to the controlled diet provided the week before both study occasions.

**Exercise program**

For the duration of 12 wk, participants came to the physical therapy unit at Texas Children’s Hospital twice a week for a 30-min aerobic exercise session on a treadmill, elliptical, or bicycle (dependent on the preference of the participant). Each exercise session was preceded and followed by 10 min of warm up/cool down and stretching. The exercise intensity level was designed to result in a heart rate corresponding to \( \geq 70\% \) of that obtained at VO\(_2\) peak at baseline (see Cardiovascular fitness below), which corresponded to heart rates >140 beats/min in all participants. This rate had to be maintained for the entire 30-min session.

Experienced exercise physiologists were responsible for the training sessions together with the principal investigator. Participants were instructed to perform a similar program (same duration and intensity) twice a week at home, ie, a total of 4 exercise sessions per week. To ensure that the desired heart rate (exercise intensity) was achieved and maintained for 30 min, each participant wore a heart rate monitor (Polar S-710; Health Check Systems, Brooklyn, NY) during all home and hospital exercise sessions. Information from the monitors was downloaded and discussed with the participants weekly. To measure the effect of the exercise program alone, participants were asked not to perform any exercise outside the program.

The last exercise session took place \( \geq 24\) h before the start of the calorimeter study and \( \approx 38\) h before the measurement of BEE. The compliance with the program was very good, as evidenced by the attendance (88 \( \pm \) 2\% and 91 \( \pm \) 2\%) of the lean and obese participants, respectively, at heart rates corresponding to 85 \( \pm \) 1\% (lean) and 85 \( \pm \) 2\% (obese) of that obtained at VO\(_2\) peak.

**Cardiovascular fitness**

VO\(_2\) peak was measured at baseline and postexercise by using a modified Bruce treadmill protocol. The treadmill test started at a speed of 1.7 mph. Subsequently, the speed and incline were gradually increased every 3 min until maximal exercise capacity of the participant was reached. VO\(_2\) was measured with a Vmax-229 metabolic cart (SensorMedics, Anaheim, CA). VO\(_2\) peak was determined by using standard criteria, specifically a heart rate \( >195\) beats/min or a respiratory quotient (RQ) \( >1.0 \) at peak exercise (29).

**Room respiration calorimetry**

VO\(_2\), carbon dioxide production (VCO\(_2\)), and RQ, defined as VCO\(_2\)/VO\(_2\), were measured continuously in 17-m\(^3\) room calorimeters for 24 h at baseline and postexercise. The design and performance of the respiration calorimeters were described in detail previously (31). Mean (\( \pm \)SD) errors from 24-h infusions of nitrogen and carbon dioxide were \(-0.34 \pm 1.24\% \) for VO\(_2\) and 0.11 \( \pm \) 0.98\% for VCO\(_2\) (31). The rooms were equipped with a bed, desk, chair, lamp, toilet, sink, television, videocassette recorder, video games, and telephone. Heart rate was recorded by telemetry (DS-3000; Fukuda Denshi, Tokyo), and physical activity, expressed as activity counts/min, was monitored with a Doppler microwave sensor (D9/50; Microwave Sensors, Ann Arbor, MI). All urine was collected and pooled during the 24-h calorimeter test. For data analysis, we assumed that urinary nitrogen excretion was constant over the 24-h period. Urine samples were digested by using sulfuric acid (modified Kjeldahl), and the nitrogen concentration was determined spectrophotometrically by using a Technicon AAII colorimeter (Technicon Industrial Systems, Tarrytown, NY). From the 24-h VO\(_2\), VCO\(_2\), and urinary nitrogen excretion, TEE and its components were calculated by using the Weir equation (32) and non-protein RQ (NPRQ) and substrate oxidation according to Frayn (33). Energy balance was computed as total energy intake (TEI; kcal/d) minus TEE (kcal/d). The calorimeter test began at 1600. While in the calorimeter, the participants were allowed free choice of sedentary activities (television, videocassette recorder, video games, arts and crafts, reading, etc). Four meals were consumed in the calorimeter. On day 1, dinner was consumed at 1730 and a snack at 1830 (no food was allowed after 1900); on day 2, breakfast was consumed at 0830 and lunch at 1200. Bedtime was between 2100 and 2200.

**Components of 24-h TEE**

Twenty-four-hour TEE consisted of BEE, SEE, and SEDEE. BEE was measured after a 12-h overnight fast. The participants were awakened at 0630 and asked to void, after which they returned to sleep. The participants were again awakened \( \approx 30\) min later, and after being confirmed that they were awake, their BEE was measured for 40 min, beginning at 0720. Participants were monitored both visually and by an activity sensor to confirm that they were lying still (<50 activity counts/min) for the entire measurement. SEE was measured throughout the night while the participants were sleeping, as verified by a heart rate monitor and motion sensor. The measurement of SEDEE began as the BEE measurement ended (0800) and continued until the end of the calorimeter study (1600). Only sedentary activity (eg, television watching) was allowed during this measurement.

**Statistical methods**

Generalized estimating equations (GEE) (SPSS 17.0; SPSS Inc, Chicago, IL) were used to assess the effects of the exercise program, weight status (lean or obese), and interaction between the effect of the exercise program and weight status on TEE, its components (BEE, SEE, and SEDEE), and substrate oxidation. Post hoc procedures provided by GEE were used to compare groups at baseline and postexercise and to assess the effect of the exercise program within each group. This method accounts
for repeated measurements in individuals at baseline and post-exercise and when appropriate for the incorporation of confounding variables, including baseline measurements and variables that change with time. Confounding variables for the between-group comparison on energy expenditure and substrate oxidation statistics were sex and activity counts. The confounding variable for the within-group comparison was the activity count (34). Post hoc analysis per group is reported only when a significant interaction between the effect of the exercise program and weight status was observed. No interaction indicates that there was no difference in the response to exercise in lean and obese participants, ie, a variable might increase, decrease, or not change in both groups. The $P$ value reflects the combined group analysis.

RESULTS

Participant characteristics

According to the design, the obese participants had a higher body weight, BMI, fat mass, and percentage body fat than did the lean participants (all $P < 0.01$). Also, lean body mass was higher in the obese participants (Table 1; $P < 0.01$). The effect of the exercise program on body composition was not different between the lean and obese groups (no significant interaction).

Fitness and lean body mass increased to the same extent in both groups (both $P < 0.01$).

Energy intake in the calorimeter

In both lean and obese participants, total energy intake (TEI) was not different at baseline and postexercise (Table 2). TEI was higher in obese participants as a result of their higher requirements as calculated by Dietary Reference Intakes (30) ($P < 0.05$). In addition, the macronutrient distribution of the intake corresponded to the design and was not different between groups and study occasions.

Energy expenditure

At baseline, TEE (kcal/d) and its components BEE, SEE, and SEDEE (kcal/min) were significantly higher in the obese group than in the lean group (Table 2 and Table 3; both $P < 0.01$). These differences remained significant ($P < 0.01$) when adjusted for confounding variables (sex and activity counts). TEE expressed as kcal · kg$^{-1} · d^{-1}$ was higher in the obese than in the lean adolescents, whereas TEE expressed per kg lean body mass did not differ between groups. The effect of the exercise program on energy expenditure was not different between lean and obese participants. No significant

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>Energy intake and energy expenditure over 24 h in the calorimeter$^1$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lean participants ($n = 13$)</td>
</tr>
<tr>
<td></td>
<td>Baseline</td>
</tr>
<tr>
<td>TEI (kcal/d)</td>
<td>2134 ± 85</td>
</tr>
<tr>
<td>TEE (kcal/d)</td>
<td>1918 ± 86</td>
</tr>
<tr>
<td>Energy balance</td>
<td>215 ± 50</td>
</tr>
<tr>
<td>TEE (kcal · kg$^{-1} · d^{-1}$)</td>
<td>34 ± 1</td>
</tr>
<tr>
<td>Energy expenditure</td>
<td>44 ± 1</td>
</tr>
<tr>
<td>Activity counts (counts/min)</td>
<td>104 ± 7</td>
</tr>
<tr>
<td>24 h RQ</td>
<td>0.84 ± 0.01</td>
</tr>
<tr>
<td>24 h NPRQ</td>
<td>0.84 ± 0.01</td>
</tr>
<tr>
<td>24-h Substrate intake</td>
<td></td>
</tr>
<tr>
<td>Carbohydrate (kcal/d)</td>
<td>652 ± 26</td>
</tr>
<tr>
<td>Carbohydrate (% of TEI)</td>
<td>31 ± 1</td>
</tr>
<tr>
<td>Fat (kcal/d)</td>
<td>1151 ± 49</td>
</tr>
<tr>
<td>Fat (% of TEI)</td>
<td>54 ± 1</td>
</tr>
<tr>
<td>Protein (kcal/d)</td>
<td>331 ± 14</td>
</tr>
<tr>
<td>Protein (% of TEI)</td>
<td>15 ± 1</td>
</tr>
<tr>
<td>24-h Substrate oxidation</td>
<td></td>
</tr>
<tr>
<td>Carbohydrate (kcal/d)</td>
<td>717 ± 40</td>
</tr>
<tr>
<td>Carbohydrate (% of TEE)</td>
<td>37 ± 1</td>
</tr>
<tr>
<td>Fat (kcal/d)</td>
<td>886 ± 50</td>
</tr>
<tr>
<td>Fat (% of TEE)</td>
<td>46 ± 1</td>
</tr>
<tr>
<td>Protein (kcal/d)</td>
<td>312 ± 26</td>
</tr>
<tr>
<td>Protein (% of TEE)</td>
<td>16 ± 1</td>
</tr>
</tbody>
</table>

$^1$ All values are means ± SEs. The data were not corrected for activity counts and sex. TEI, total energy intake; TEE, total energy expenditure; RQ, respiratory quotient; NPRQ, nonprotein respiratory quotient; LBM, lean body mass.

$^2$ General estimating equations were used to determine potential interactions between the lean and obese participants with regard to the effect of exercise. When a significant interaction was found between the lean and obese groups, post hoc procedures provided by general estimating equation were used to compare groups at baseline and postexercise and to assess the effect of the exercise program within each group. When there was no interaction (ie, the response to exercise did not differ between the groups), but both groups showed a decrease or increase in the measured variable, combined statistics were used to determine the significance level.

$^{1,4,5}$ Significantly different from lean participants: $^{1}P < 0.05$, $^{4}P < 0.01$.

$^{5,6}$ Significantly different from baseline within each group: $^{5}P < 0.01$, $^{6}P < 0.05$. 
change in TEE (kcal/d and kcal · kg lean body mass⁻¹ · d⁻¹), BEE, SEE, or SEDEE was observed after the exercise program in either group. The change in TEE (kcal · kg⁻¹ · d⁻¹) in lean participants was no longer significant after adjustment for activity counts.

Energy expenditure values obtained at 1-h intervals for the duration of the calorimeter measurements (baseline and postexercise) showed higher energy expenditure (kcal/min) in the obese than in the lean participants at all time points, with no effect of the exercise program (Figure 1). Energy balance was positive in both groups on both study occasions. We did not measure fecal losses. These losses would most likely have canceled out the slightly positive energy balance.

**Substrate oxidation**

At baseline, 24-h total RQ and NPRQ and basal, sleep, and sedentary awake RQ were significantly lower in the obese than in the lean participants (Tables 2 and 3; \( P < 0.01, P < 0.01, P < 0.01, P < 0.01, P < 0.05, \) respectively). Fat oxidation expressed per kcal/d and as % of TEE was higher in the obese than in the lean participants (both \( P < 0.01 \)). Total carbohydrate oxidation (kcal/d) was not significantly different between groups, whereas carbohydrate oxidation (% of TEE) was significantly lower in the obese participants (\( P < 0.01 \)). After adjustment for confounding variables (sex and activity counts), all differences remained significant. Protein oxidation (kcal/d and % of TEE) was not significantly different between groups.

In the obese participants, the exercise program did not affect 24-h RQ and NPRQ or basal, sleep, and sedentary awake RQ. In addition, 24-h total carbohydrate, fat, and protein oxidation (kcal/d and % of TEE) did not change as a result of the exercise program. In contrast, in lean participants, the exercise program resulted in a significant decrease in 24-h total RQ and NPRQ and sleep RQ (all \( P < 0.01 \)). Fat oxidation (% of TEE) increased (\( P < 0.01 \)) after the exercise program, whereas carbohydrate oxidation (kcal/d and % of TEE) decreased (both \( P < 0.01 \)). Thus, carbohydrate and fat oxidation rates postexercise were no longer different between the obese and lean participants. Protein oxidation (kcal/d and % of TEE) remained unchanged. All differences remained significant after correction for activity counts. RQ values at 1-h intervals throughout the calorimeter study are shown for both groups in Figure 2. The figure shows an overall pattern of lower RQ in the obese than in the lean participants at baseline and a decrease in RQ postexercise in the lean participants.

**DISCUSSION**

The results of the present 24-h calorimeter measurements show that a 12-wk moderate aerobic exercise program without weight loss did not affect TEE or its components: BEE, SEE, and SEDEE in either lean or obese adolescents. No changes in substrate oxidation were observed in obese adolescents. In lean

**FIGURE 1.** Hourly energy expenditure (EE) in lean and obese participants during the course of the calorimeter study at baseline and postexercise. \( n = 13 \) (lean); \( n = 15 \) (obese).
adolescents, carbohydrate oxidation decreased and fat oxidation increased in response to the exercise program. Obese adolescents had a significantly higher energy expenditure than did their lean counterparts. It has been suggested that metabolically active lean body mass is the main predictor of energy expenditure (34–36). Thus, the higher TEE observed in the obese participants in our study was explained by their higher lean body mass. This was further supported by the fact that TEE expressed per kg lean body mass was not different between lean and obese participants. TEE expressed per kg body weight was, however, lower in obese participants, which indicated that the large fat mass had less of an effect on energy metabolism than lean body mass. We found no effect of our exercise program on 24-h TEE. Reports from other investigators are divergent. Some studies have shown increased REE in response to exercise (6–9), whereas others have found no effects (10). Several factors might explain these inconsistencies. Most studies measured REE over a short 30-min period (7, 8, 10). Extrapolating these data to a 24-h period might provide erroneous estimates of 24-h TEE. The use of 24-h room calorimetry allowed us to accurately measure TEE taking into account the variation resulting from basal, sleep, and sedentary activity (31). Another potential confounder was the timing of the measurements in relation to the previous exercise session (6–10). Although the acute effect of an exercise bout on energy expenditure was greatest during the first couple of hours, some effects might persist for 36 to 48 h depending on the duration and intensity of the exercise (11). In the present study, postexercise TEE was measured between 24 and 48 h after the last exercise session, and BEE was measured ≥38 h after the final exercise session. In addition, energy expenditure was not different during the first and the last hours of the 24-h measurement period in either lean or obese participants (Figure 2). Therefore, it is highly unlikely that our energy expenditure data were overestimated. Two studies (6, 7) measured energy expenditure at 48 and 72 h, respectively, after the final exercise session of an exercise program (ie, persistent acute exercise effects were unlikely). The investigators reported that resistance training 3 times/wk for 6 mo (6) and 3–5 aerobic exercise sessions (20–45 min/session at 60–75% of heart rate reserve) weekly for 6 mo (7) increased energy expenditure. They concluded that their results were primarily related to increased lean body mass but suggested that exercise-induced changes in other factors known to affect BEE [eg, sympathetic nervous system activity (37) and thyroid hormones (38)] also might have played a role. The small increase in lean body mass (2–3%) in our participants might have been insufficient to affect energy expenditure. The intensity and duration of an exercise program required to modulate hormone and sympathetic nervous system activity in adolescents are not known.

We previously showed that substrate oxidation rates are affected by the macronutrient composition of the diet (29). Therefore, the energy content and macronutrient distribution of the intake were well controlled and did not differ between the 2 study occasions (baseline and postexercise). This allowed us to accurately assess the effect of the exercise program on substrate oxidation and to compare the data between lean and obese adolescents.

At baseline, total fat oxidation (kcal/d) was significantly higher in the obese than in the lean participants. In addition, obese participants relied to a greater extent on fat oxidation for TEE than did the lean participants, even though fat oxidation was the principal contributor to energy expenditure in both groups (57% and 46%, respectively). This is in agreement with the reports from Maffeis et al (39). These investigators showed that total fat oxidation was highly related to fat mass and that the rate of exogenous fat oxidation increased with adiposity. They postulated that increased fat oxidation might be a protective measure to avoid a further increase in fat mass in obese children (39). In agreement with the observation by Rueda-Maza et al (40), total carbohydrate oxidation (kcal/d) was not different between our lean and obese participants. However, the proportion of TEE derived from carbohydrates was lower in the obese than in the lean adolescents.

The exercise program did not change substrate oxidation in the obese participants. On the other hand, fat oxidation (% of TEE) increased and carbohydrate oxidation (kcal/d and % of TEE) decreased in the lean participants. Similar findings have been reported in adults (7, 16–18, 20). Physical activity increased resting fat oxidation in lean adults (16–18), whereas no increase was observed in overweight and obese adults (7, 20). A recently published study (6) found increased resting and sleep fat oxidation after 6 mo of low-volume resistance training in overweight adults as compared with a nonexercising control group. However, 24-h fat oxidation remained unchanged (6). The explanation for this discrepancy between lean and obese individuals is not well understood. Blaak et al (19) proposed that obese individuals have an impaired capacity to oxidize fat, possibly prohibiting an increase in fat oxidation in response to exercise. At baseline, fat oxidation was higher in our obese than in our lean participants, which contradicts impaired fat oxidation. However, the lean but not the obese adolescents responded with increased fat oxidation to the exercise program. This might indicate impaired metabolic flexibility in obese adolescents. Rueda-Maza et al (40) suggested that lower carbohydrate oxidation in obese than in lean children would indicate reduced glycogen turnover in the obese. We measured glycogenolysis in our adolescents and found no difference between lean and obese participants at baseline, despite lower carbohydrate oxidation in the obese participants (4). Postexercise, carbohydrate oxidation rates were similar in obese and lean adolescents, despite the decrease in glycogenolysis in the obese adolescents, but remained unchanged in the lean participants (4). These findings contradict a relation between glycogen turnover and carbohydrate oxidation rates.
The calorimeter test is an important part of our extensive study of the metabolic effect of a 12-wk aerobic exercise program. The purpose of this study was to determine the effect of a controlled aerobic exercise program alone (ie, without weight loss) on energy expenditure and its subcomponents, substrate oxidation, body composition, detailed body fat distribution, insulin sensitivity, and glucose and lipid metabolism. We previously reported (in partly the same participants) (4, 5) that this moderate 12-wk aerobic exercise program resulted in increased peripheral insulin sensitivity (59 ± 19% and 35 ± 14%) and hepatic insulin sensitivity (23 ± 4% and 19 ± 7%) in the lean and obese participants, respectively. In addition the program decreased the visceral and hepatic fat content in the obese adolescents. Collectively, our data showed that, in obese adolescents, important reductions in visceral and hepatic fat and significant improvements in both peripheral and hepatic insulin sensitivity occurred without weight loss and without changes in whole-body TEE and its components or in substrate oxidation rates. These findings indicate that aerobic exercise may have its major effects on the cellular level in muscle (41) and liver (42), not reflected by changes in whole-body energy expenditure and substrate oxidation. Because it is unethical to perform muscle and liver biopsies in healthy adolescents, these avenues could not be pursued. We speculate that an exercise program with a greater effect on lean body mass and/or a combined exercise and weight-loss intervention would be required to achieve a significant increase in energy expenditure and fat oxidation.

In conclusion, a 12-wk moderate aerobic exercise program did not increase TEE, BEE, SEE, and SEDEE in either obese or lean adolescents. In lean participants, fat oxidation increased post-exercise, whereas oxidation rates were not affected in the obese. Thus, in obese adolescents, changes in whole-body energy expenditure and substrate oxidation rates are not required to reduce the visceral and hepatic fat content and improve peripheral and hepatic insulin sensitivity.

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