Fat oxidation rate and the exercise intensity that elicits maximal fat oxidation decreases with pubertal status in young male subjects

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THE TWO MAIN ENERGY SOURCES oxidized during aerobic exercise are fat and carbohydrate, and their relative proportions depend primarily on the intensity of exercise (45). As the work rate increases, there is a progressive increase in the ratio of carbohydrate to fat oxidation (17). Although the relative rate of fat oxidation decreases with increasing exercise intensity, the absolute rate (e.g., mg/min) increases from low to moderate intensity and then decreases as the exercise becomes more intense (1, 45, 55). Using a graded exercise protocol that allows for the estimation of fat oxidation rates over a wide range of intensities, Achten et al. (1) found that fat oxidation in endurance trained men peaks at 64% of peak aerobic power (\(\dot{V}O_2\) peak), with maximal rates of \(-0.60\) g/min observed. High rates of fat oxidation in these trained cyclists were observed over a wide range of exercise intensities (i.e., between 55 and 72% \(\dot{V}O_2\) peak) with rates decreasing rapidly toward zero by \(-90\%\) \(\dot{V}O_2\) peak. In addition to the exercise intensity, other variables such as the duration of exercise (45), sex (51), nutrient intake (43), fitness level (13), mode of exercise (5), and the level of body adiposity (29) also alter the mix of substrates oxidized during prolonged exercise. The exercise intensity that elicits maximal fat oxidation (Fatmax) is influenced by sex, the amount of fat-free mass and fitness level, according to a large cross-sectional study, although these variables only explain \(-12\%\) of the variance (55). Our laboratory (52–54, 54) and others (9, 24, 25, 37, 39, 40, 44, 48) have shown that the effects of biological age and/or maturational status also impact the mix of fuel utilization during exercise. Indeed, for nearly 70 yr, it has been known that children have a lower respiratory exchange ratio (RER) than adults during submaximal exercise performed at the same relative intensity (i.e., percentage of \(\dot{V}O_2\) peak) (41). The lower RER in children, compared with adults, suggests that children oxidize more fat and less carbohydrate at a given intensity of exercise. Although important, these studies are limited because they frequently compare substrate oxidation at only one exercise intensity (i.e., moderate) and do not attempt to quantify peak fat oxidation rates or the exercise intensity that corresponds to that peak rate (i.e., Fatmax). Recently, Stephens et al. (48) found that early and midpubertal boys have higher relative rates of fat oxidation than either late pubertal or adult male subjects during cycling at 30, 40, 50, 60, and 70% \(\dot{V}O_2\) peak. Although important, this study did not determine Fatmax precisely, nor did it assess whether maturational status or age influences Fatmax in youth. Further study of pubertal effects on energy metabolism during exercise is important for a number of reasons. First, puberty is associated with an increase in insulin resistance (6) and alterations in substrate partitioning in response to lipid (7) and glucose (8) infusions. These studies suggest that pubertal development is associated with a conservation of carbohydrate stores, most likely for the energy requirements of growth. Second, knowledge of substrate metabolism in relation to pubertal status in healthy children is likely to be of clinical relevance in a variety of metabolic-related conditions. For example, our laboratory has assessed substrate utilization in children with Type 1 diabetes mel-
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litus and found that, despite elevated circulating insulin levels, both endogenous and exogenous carbohydrate oxidation during exercise is lower than in children without diabetes (42). Finally, a better understanding of how puberty influences substrate utilization during exercise in healthy-weight children will also help clarify the potential consequences of excess adiposity on fuel selection during exercise in overweight children. To date, all studies of the influence of pubertal status and age on fuel metabolism during exercise have been conducted using cross-sectional designs, thereby limiting the ability to account for between subject variability in any “genetic” variation in substrate metabolism.

The primary purpose of this longitudinal study was to profile carbohydrate and fat oxidation rates over a wide range of relative exercise intensities in a small cohort of growing, healthy, normally active prepubertal boys and compare these values with that of young adult male subjects. We hypothesized that, when expressed in relative terms (mg·kg lean body mass⁻¹·min⁻¹), fat oxidation rates would be higher in prepubertal boys compared with men throughout a wide range of exercise intensities and that peak fat oxidation would occur at a higher percentage of their \( V_{O2 \text{ peak}} \). We also hypothesize that as these boys matured, fat oxidation rates would decrease to values similar to those observed in young men.

METHODS

Subjects. Initially, six healthy, normally active boys, all prepubertal (i.e., Tanner stage 1, ages 11 or 12 yr) were recruited to participate in a 3-yr longitudinal study that began in late 2003. Because of a non-life-threatening injury, one boy dropped out of the study between year 1 and year 2, leaving \( n = 5 \) for the analysis shown. The reason for the small sample size was to pilot the effects of maturational status on substrate utilization using a previously established protocol. Substrate oxidation rates were assessed on an annual basis (see below) as they developed through puberty over a period of 3.5 yr. As a comparator, nine healthy, normally active, adult male subjects (ages 22–26 yr) also participated, but they were studied only on one occasion (5 men were measured in 2003, and 4 men were measured in 2006). This study was approved by the York University Human Participants Ethics Review Committee. The pubertal status of the boys was established annually according to pubic hair development by the criteria of Tanner (50), assessed by a parent and the child. Tanner self-staging has been validated as a reasonable tool for the assessment of pubertal development (38) and correlates with serum testosterone levels in maturing boys (unpublished observations). Our laboratory has used this self-assessment method previously (53, 54), which we consider appropriate for use in a research setting because it minimizes feelings of embarrassment for the subjects. All of the boys progressed to Tanner stage 4 at a similar rate by the end of the study, according to their own self-reported staging. All participants were healthy, of normal weight, not taking any medication, and recreationally active but not endurance-trained athletes. After the purpose, procedures, and risks of the study were explained to each subject, the men signed an informed consent, while the boys assented verbally to participate. Each boy’s parent then signed an informed consent on his or her son’s behalf.

Subjects reported to the laboratory in a fasted state (~0800) on each occasion for substrate oxidation measures using a standardized protocol (see below). Also during each visit, the following measurements were conducted: height (stadiometer), body mass (Seca digital scale), percent body fat [boys: sum of 2 skinfolds (46); men: sum of 3 skinfolds (31)].

Exercise protocol. Subjects arrived at the laboratory in the morning of the experimental trial in a fasted state. They were asked to avoid strenuous exercise the day before the trial. Before beginning the graded exercise test, subjects would practice breathing into the metabolic mouthpiece and cycle at a low wattage to become accustomed with the cycle ergometer. After sitting quietly for 20 min, subjects performed a graded exercise test to volitional fatigue on an electromagnetically braked cycle ergometer with continuous gas collection and heart rate monitoring, according to a modified protocol developed by Achten et al. (1). For this, boys (Tanner stage 1 and 2/3) started cycling at 12.5 W, and the work rate was increased 12.5 W every 3 min. The men started at 25 W, and the work rate was increased 25 W every 3 min. To help keep the exercise task duration to ~30 min, the watt increment was changed to 20 W when the boys were tested at Tanner stage 4. In all subjects, when the RER was ≥ 1.00, indicating zero fat oxidation, the work rate was increased by the same increments at 1-min intervals until volitional fatigue. The purpose of this modified protocol was to profile both substrate oxidation, at least to the anaerobic threshold, and to determine \( V_{O2 \text{ peak}} \), as has been described by Venables et al. (55) for use in adults. \( V_{O2 \text{ peak}} \) was considered to have been reached when the RER was >1.05 and the subject achieved his age-predicted maximal heart rate [heart rate maximum(220–age)]. It is important to note that this method may underestimate the true aerobic capacity (i.e., \( V_{O2 \text{ max}} \)) of the subjects because of the exercise modality (i.e., cycling compared with treadmill running) and because of the somewhat prolonged test duration. Peak work rate during the incremental test was calculated from the last completed work rate plus the fraction of time spent in the final noncompleted work rate multiplied by the work rate increment. Single-lead heart rate ECG and breath-by-breath volume and gas analysis were recorded continuously using a calibrated Sensor-Medics metabolic cart (Vmax 29). The volume sensor and gas analyzers of the system were calibrated using a 4-liter calibration syringe and commercially prepared gas mixtures of known concentration certified to within 0.02% (cylinder 1: 25.9% \( O_2 \),-4.0% \( CO_2 \),-balance \( N_2 \); cylinder 2: 16.00% \( O_2 \), 4.00% \( CO_2 \),-balance \( N_2 \)). \( O_2 \) consumption (\( V_{O2} \)) and \( CO_2 \) production (\( V_{CO2} \)) were averaged over the final 30 s of each work rate, and the results of this test were used to calculate fat oxidation over a wide range of exercise intensities for each subject (1). Indirect calorimetry, as was used in this study, is the standard method to quantify substrate utilization during strenuous exercise (26), although it does have its limitations during high-intensity exercise (34) (see DISCUSSION).

Indirect calorimetry and calculations. Fat oxidation was calculated using the following equation (26) with the assumption that the urinary nitrogen excretion rate was negligible:

\[
\text{Fat (g/min) = 1.67} \cdot V_{O2} (l/min) - 1.67 \cdot V_{CO2} (l/min)
\]

For each individual, a best-fit polynomial curve was constructed of fat oxidation rate (expressed as mg·kg lean body mass⁻¹·min⁻¹) vs. exercise intensity (expressed as %\( V_{O2 \text{ peak}} \)), as illustrated in the example in Fig. 1. Because of the likelihood that indirect calorimetry underestimates fat oxidation at exercise intensities above the “anaerobic threshold” because of a reduction in the functional size of the bicarbonate pool (34), we profiled the curves only up to 75% of the subjects \( V_{O2 \text{ peak}} \). Each individual curve was then used to determine the peak fat oxidation rate and the exercise intensity that was associated with maximal fat oxidation rate (\( \text{Fatmax} \)), according to the protocol of Achten et al. (1). The average \( R^2 \) for the fitted curves in the boys were: Tanner 1 = 0.75 ± 0.12, Tanner 2/3 = 0.85 ± 0.16 and Tanner 4 = 0.79 ± 0.13. The average \( R^2 \) for the fitted curves in the men was 0.81 ± 0.10. Individual results were then used to compose average fat oxidation curves for the men and for the boys at each stage of maturation. To do this, the \( \text{Fatmax} \) and the exercise intensities that elicited fat oxidation rates at 95, 90, and 80% of peak fat oxidation to the left side of \( \text{Fatmax} \) were determined for each individual (1). In addition, the exercise intensity that 95% of fat oxidation rates above \( \text{Fatmax} \) was also determined (1) (Fig. 1). These specific points were averaged separately for both the boys (at each Tanner stage) and for the men and then plotted against the average fat oxidation rates (expressed as mg·kg body mass⁻¹·min⁻¹). Similar graded exercise
tests in children (16, 48, 59) and in adults (1, 4, 5, 55) have been used to quantify substrate oxidation with the following limitations: 1) substrate oxidation rates may not be valid because of bicarbonate buffering at high intensities (i.e., >70% \( \dot{V}O_2 \) peak), 2) the duration of the exercise may have been too long to have allowed for subjects to reach their true cycling \( \dot{V}O_2 \) peak, and 3) the progressive nature of the exercise may not allow for an accurate measure of substrate oxidation at steady state, because time and duration are known to influence substrate utilization rate.

**Statistical analysis.** Experimental data are expressed as means ± SD. Significant differences in anthropometric data, functional data, \( F_{\text{max}} \), and the calculated fat oxidation rates between the boys at different stages of maturation were determined using a one-way repeated-measures ANOVA with three levels of maturation (Tanner 1 vs. Tanner 2/3 vs. Tanner 4). For comparisons between the boys at different maturation stages and the men, a one-way between-factor ANOVA (4 levels of group) was used in a separate analysis. This two-step analysis allowed us to accommodate our mixed-methods design (repeated measures in the boys and between measures in the boys vs men). Differences between mean values were determined if a significant \( F \)-ratio was found by using Tukey’s multiple comparison test. All statistical procedures were computed using GraphPad Prism software (GraphPad Software 7, San Diego, CA).

**RESULTS**

Lean body mass increased in the boys from Tanner stage 1 (33.5 ± 5.6 kg) to Tanner stage 4 (48.3 ± 8.6 kg) \((P < 0.05)\). As expected, the boys during Tanner stage 1 and Tanner stage 2/3 had a lower lean body mass than the men (65.7 ± 9.0 kg) (both \( P < 0.05)\). Other physical and functional characteristics of the subjects are summarized in Table 1. During exercise, boys at Tanner stage 1 had a lower absolute peak work rate \((P < 0.001)\) but were similar in fat mass, relative \( \dot{V}O_2 \) peak, and percentage of age-predicted heart rate maximum to the men. As expected, over the study period, the boys grew in height (\( P < 0.001)\), weight (\( P < 0.001)\), and lean body mass (\( P < 0.001)\) but still remained shorter (\( P < 0.05)\) and lighter (\( P < 0.05)\) than the male subjects by the time they reached Tanner 4. The exercise duration was 27.5 min (range 24.5–35.3 min) for the boys at Tanner stage 1. The exercise duration was 28.4 min (range 22–32 min) in the men (not significantly different). The duration was similar at 34.5 min (range 27.5–39 min) by the time the boys reached Tanner stage 4 \((P > 0.05)\). Peak power output was higher in the men compared with the boys in Tanner 4 \((P < 0.05)\). Peak heart rate during exercise and percentage of age-predicted heart rate maximum was similar between the boys at all stages of puberty and the men.

Figure 2 illustrates the relationship between relative fat oxidation rate (expressed in mg·kg lean body mass\(^{-1} \cdot \text{min}^{-1}\)) and exercise intensity (expressed as a percentage of \( \dot{V}O_2 \) peak) as determined by the polynomial curve-fitting method described above. The peak fat oxidation rate was approximately twofold higher \((P < 0.05)\) in the boys in Tanner stage 1 (8.6 ± 1.5 mg·kg lean body mass\(^{-1} \cdot \text{min}^{-1}\)) compared with the men (4.2 ± 1.1 mg·kg lean body mass\(^{-1} \cdot \text{min}^{-1}\)). Peak fat oxidation rate was similar between Tanner 1 and Tanner 2/3 (7.6 ± 0.6 mg·kg lean body mass\(^{-1} \cdot \text{min}^{-1}\)), but it decreased significantly \((P < 0.05)\) by Tanner 4 (5.4 ± 1.8 mg·kg lean body mass\(^{-1} \cdot \text{min}^{-1}\)) to values similar to those observed in the men. The exercise intensity that elicited peak fat oxidation rate (i.e., \( F_{\text{max}} \)) was higher in the boys at Tanner stage 1 (56 ± 6% \( \dot{V}O_2 \) peak), stage 2/3 (55 ± 2 \( \dot{V}O_2 \) peak), and stage 4 (45 ± 10% \( \dot{V}O_2 \) peak), compared with the men (31 ± 4% \( \dot{V}O_2 \) peak) (main effect of group \( P < 0.0001)\). Repeated-measures ANOVA on the effect of Tanner stage revealed a trend for a decrease in \( F_{\text{max}} \) with maturation \((P = 0.058)\). High rates of fat oxidation (i.e., within 5% of peak) were observed between 47 ± 6 to 65 ± 7% \( \dot{V}O_2 \) peak in the boys in Tanner 1 compared with 27 ± 4% to 37% ± 5% \( \dot{V}O_2 \) peak in the men (both \( P < 0.05)\). The

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**Table 1. Physical and functional characteristics of the boys at each stage of maturation and the men**

<table>
<thead>
<tr>
<th>Age, yr</th>
<th>Height, cm</th>
<th>Weight, kg</th>
<th>Body Fat, %</th>
<th>( \dot{V}O_2 ) peak, ml·kg(^{-1} \cdot \text{min}^{-1} )</th>
<th>Peak Work Rate, W</th>
<th>Peak RER</th>
<th>Peak Heart Rate, beats/min</th>
<th>Percentage of Age-Predicted Heart Rate Maximum, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tanner I</td>
<td>12.0 ± 0.4(^a)</td>
<td>150.4 ± 5.1(^a)</td>
<td>40.8 ± 10.9(^a)</td>
<td>16.3 ± 10.4</td>
<td>45.6 ± 6.5</td>
<td>118 ± 21(^a)</td>
<td>1.05 ± 0.06</td>
<td>192 ± 9</td>
</tr>
<tr>
<td>Tanner 2/3</td>
<td>13.2 ± 0.5(^b)</td>
<td>159.4 ± 7.3(^b)</td>
<td>47.9 ± 11.7(^b)</td>
<td>13.2 ± 6.6</td>
<td>48.3 ± 9.2</td>
<td>170 ± 52(^b)</td>
<td>1.01 ± 0.06</td>
<td>193 ± 3</td>
</tr>
<tr>
<td>Tanner 4</td>
<td>14.7 ± 0.4(^c)</td>
<td>168.5 ± 5.6(^c)</td>
<td>56.6 ± 12.2(^c)</td>
<td>14.1 ± 5.8</td>
<td>46.6 ± 3.3</td>
<td>214 ± 48(^c)</td>
<td>1.04 ± 0.05</td>
<td>194 ± 3</td>
</tr>
<tr>
<td>Men</td>
<td>23.8 ± 1.2</td>
<td>177.3 ± 5.4</td>
<td>75.3 ± 11.7</td>
<td>12.3 ± 6.3</td>
<td>44.6 ± 9.0</td>
<td>279 ± 59</td>
<td>1.07 ± 1.08</td>
<td>187 ± 6</td>
</tr>
</tbody>
</table>

Values are means ± SD. \( \dot{V}O_2 \) peak, peak aerobic capacity. Different letters within a column (i.e., a, b, c) denote difference between maturational stages as determine by a repeated measures ANOVA. *Values differs from men at \( P < 0.05)\ as determined by post hoc analysis following a significant main effect of group using a one-way ANOVA.
have been reported previously using a variety of steady-state (9, 24, 37, 54) and non-steady-state (39, 40, 44) exercise protocols. Previously, our laboratory found that boys use ~70% more fat and ~23% less carbohydrate compared with men during prolonged moderate-intensity exercise, performed after a small standardized meal (54). The higher relative rate of fat oxidation in children, compared with adults, occurs even when exogenous carbohydrates are consumed during exercise (53, 54). In this study, we extend these observations by showing that throughout a wide range of relative exercise intensities, fat oxidation rate, expressed relative to lean body mass, is approximately twofold higher in prepubertal boys compared with men (Fig. 2). This dramatically higher fat oxidation rate drops as the boys develop through puberty to values similar to what is observed in the men. In addition to the higher relative rate of fat oxidation in prepubertal boys, we also found that Fatmax was considerably higher in the boys at the early stages of puberty compared with the men (Fig. 2). Duncan and Howley (20) also reported fat oxidation rates in prepubertal children during five, 6-min steady-state exercise tasks and estimated that the peak fat oxidation rate occurs somewhere between 50 and 65% \( \dot{V}O_2\text{peak} \), although the nature of their protocol did not allow them to determine the Fatmax in their subjects. Stephens et al. (48) recently showed that fat oxidation rate was higher in prepubertal boys compared with pubertal boys at the same relative exercise intensity, and based on their RER values collected, it appeared that maximal fat oxidation occurred at somewhere between 40 and 70% \( \dot{V}O_2\text{peak} \) in prepubertal boys. Our observation that peak fat oxidation rate occurs at 56% \( \dot{V}O_2\text{peak} \) in normally active prepubertal boys, based on our curve-fitting technique, is also comparable to the Fatmax of 64% of \( \dot{V}O_2\text{peak} \) reported for elite male cyclists using a similar protocol (1). Moreover, like the elite male athletes studied previously, the untrained boys in our study had high rates of fat oxidation (i.e., within 5% of the highest fat oxidation rate observed) up to at least 65% \( \dot{V}O_2\text{peak} \). Together, these data suggest that normally active prepubertal boys possess the capacity for high fat oxidation rates even at high work rates similar to that observed in endurance-trained men.

The average peak fat oxidation rate in the normally active boys (Tanner stages 1–4, 6.1 mg·kg\(^{-1}\)·min\(^{-1}\)) in the present study and in the trained men (~8.0 mg·kg\(^{-1}\)·min\(^{-1}\) in a previous study (4) contrasts with the considerably lower Fatmax (~4.0 mg·kg\(^{-1}\)·min\(^{-1}\)) observed in the untrained men in the present study. Compared with untrained individuals, trained individuals have higher absolute rates of fatty acid oxidation during exercise performed at the same relative work rate, in men (33) and in women (49). This greater reliance on fat as fuel occurs because of a higher absolute work rate performed in the trained vs. untrained individuals and possibly because of training-induced adaptations in the skeletal muscle of trained individuals (30, 32). We also found that the Fatmax is lower in the untrained men in our study (31% \( \dot{V}O_2\text{peak} \)) compared with the trained subjects used by Achten et al. (1) (64% \( \dot{V}O_2\text{peak} \)). In a large cross-sectional study conducted by Venables et al. (55), Fatmax during treadmill running occurred at 45 and 52% \( \dot{V}O_2\text{max} \) in men and women, respectively. Taken together, these findings suggest that either the level of habitual physical activity and/or the aerobic capacity influences Fatmax. It is unclear whether these variables would influence the fat oxida-

**DISCUSSION**

To our knowledge, this is the first study to examine the effects of maturation on peak fat oxidation rate and the exercise intensity that elicits Fatmax during exercise. We found that, compared with men, prepubertal boys had considerably higher relative rates of fat oxidation (mg·kg lean body mass\(^{-1}\)·min\(^{-1}\)) throughout a wide range of exercise intensities (Fig. 2). Furthermore, we found that the peak fat oxidation rate in prepubertal boys occurs at a significantly higher relative metabolic rate than in men. Finally, we demonstrated that in these same healthy growing adolescent boys, both the peak fat oxidation rate and Fatmax decrease with advancing pubertal stage, with the greatest decreases occurring during the final stages of puberty.

It has long been known that children have a higher rate of fat oxidation during exercise compared with adults. Higher relative rates of fat oxidation in children, compared with adults,
tion rates in children who are already achieving high rates of fat oxidation during high-intensity exercise.

Similar to other studies measuring fat oxidation rates over a wide range of exercise intensities, we found considerable interindividual differences in the range of exercise intensities at which high rates of fat oxidation occur (Fig. 2). Differences in $V_{O2max}$, daily physical activity levels, nutritional status, and perhaps a large genetic component appear to contribute to the variance in $Fat_{max}$ and overall fat oxidation rates during exercise (55). It is important to note, however, that we attempted to match the men in our study to the boys measured in Tanner 1 in percent body fat and relative $V_{O2peak}$ (Table 1) in an attempt to limit the influence that these variables may have on fat oxidation rates. Even as the boys progressed through puberty, $V_{O2peak}$ values remained relatively constant (Table 1), yet both the $Fat_{max}$ and the relative fat oxidation rate dropped significantly. It is currently unclear whether changes in spontaneous physical activity patterns, dietary factors, growth-related changes, and/or changes in skeletal muscle gene expression caused by pubertal hormones might influence whole body fat oxidation during physical activity in adolescents (41).

Several hypotheses may explain the higher rate of fat oxidation commonly observed during exercise in young children compared with adults. First, based on limited biopsy data collected from 6-yr-old children, prepubertal children may have an enhanced ability to oxidize fat because they have higher intramuscular triglyceride (IMTG) stores (11). This hypothesis is supported by a recent study of adult women in whom a higher resting concentration of IMTG resulted in a greater use of this fuel during subsequent submaximal exercise (47). Muscle biopsy samples may not be reflective of whole muscle IMTG stores, however, and the relative contribution of this fuel source to whole body RER during exercise is unclear. Second, higher rates of fat oxidation in children may result from a higher free fatty acid turnover during exercise (18), although elevated plasma lipids in youth compared with adults are generally not reported (14, 37). Finally, higher rates of fat oxidation in children may be a consequence of an underdeveloped glycogenolytic and/or glycolytic system (12, 21, 22, 28). In support of the later point, children have consistently been reported to have lower lactate levels during exercise (23, 36, 57), and, as pointed out by Astrand (10), the utilization of fatty acids is thought to be reciprocally related to the rate of anaerobic glycolysis and lactate production. Interestingly, plasma lactate levels correlate negatively with fatty acid oxidation rates during exercise (3, 15), suggesting that the lower lactate production in children helps facilitate higher rates of fat oxidation. Finally, it is important to note that a lower RER during exercise might not be explained entirely by a greater oxidation of fatty acids in lower limb exercising muscle per se as measurements of lipid oxidation by muscle only explain ~25% of the total lipid utilization as measured by RER (27). Future studies are needed to determine the mechanism by which the high rate of fat oxidation during exercise is diminished with physical maturation.

This study has some important clinical considerations that need to be acknowledged. In children, high rates of fat oxidation during physical activity may have some beneficial health effects. High rates of fat oxidation during physical activity may limit fat mass gain and appear to be protective against the development of obesity and obesity-related disorders. Indeed, the inability to oxidize fat appears to be an important factor in the etiology of obesity and Type 2 diabetes mellitus (35, 60). Specifically, an elevated 24-h RER, indicative of reduced levels of fat oxidation, is associated with a high rate of weight gain in adult Pima Indians (60) and has been shown to be associated with increased insulin resistance (35). In contrast, a high rate of fat oxidation during exercise is associated with increased insulin sensitivity at rest as well as reduced hypertension and lower levels of plasma low-density lipoproteins (2). Obese children have been shown to have lower rates of fat oxidation compared with lean controls, and this “metabolic defect” could be reversed by 2 mo of endurance exercise training (16). In obese adults, exercise training at $Fat_{max}$ increases peak fat oxidation rate and improves insulin sensitivity more than eucaloric interval training at an exercise intensity corresponding to a lower rate of fat oxidation (56). Future studies should emphasize how pubertal status influences fat oxidation rates during rest and physical activity and whether decreases in fat oxidation with age contribute to increases in fat storage and obesity-related disorders. However, because there is little evidence of age- or maturation-related differences in fat oxidation at rest, our findings highlight the importance of regular physical activity during childhood to maintain the capacity for fat oxidation and reduce the risk of metabolic disease.

This study also has some important limitations that need to be recognized. First, we had a very small sample of maturing boys that may not be representative of that population. A larger sample size with an adequate representation of both sexes is clearly needed. Moreover, we asked these participants to self-report their Tanner stage, which is not as accurate as having a trained pediatric endocrinologist conduct the staging (19). Second, because of our study design that utilized a prolonged and progressive exercise test to determine both $Fat_{max}$ and $V_{O2peak}$ in one visit, we may have overestimated $Fat_{max}$ in our subjects. The average duration of the exercise test was ~30 min in all of the subjects, rather than the 10- to 12-min range that is recommended for measurement of aerobic capacity with a progressive exercise task to exhaustion. As a result, it may be that the rates of fat oxidation were plotted against a more accurate estimate of $V_{O2peak}$, as determined by a more traditional exercise test that we typically use to measure aerobic capacity in adolescents, the $Fat_{max}$ would shift left on the figures shown (Figs. 1 and 2). However, we are confident that all of the subjects achieved at least near $V_{O2peak}$ for cycling, because all subjects had RER values over 1.00 at exhaustion and had heart rates that were close to their age prediction (Table 1). Moreover, as mentioned above, our determination of $Fat_{max}$ appears well in line with what has been estimated previously by Duncan and Howley (20) and by Stephens et al. (48). Third, although indirect calorimetry is used extensively to determine substrate oxidation during exercise in both children and adults, it does have some important limitations. Care must be taken to ensure that subjects attain a steady state at each progressive increase in work rate. To address this issue, we choose to have participants exercise at each stage that profiled fat oxidation rates for 3 min to allow for achievement of steadystate. It is important to note, however, that the achievement in steady state may occur more quickly in children compared with adults because the former tend to store less CO2 during exercise (58). In addition, changes in the size of the bicarbon-
ate pool at higher exercise intensities invalidate the calculations of carbohydrate and fat oxidation, particularly at intensities above the anaerobic threshold. Because of the concern that the use of indirect calorimetry may be inaccurate in assessing fuel utilization above the “anaerobic threshold,” we choose not to profile fat oxidation rates at exercise intensities above 75% of $\dot{V}O_2$ peak in this study, as has been done by others previously (1, 4, 55). Indeed, fat oxidation rates may be underestimated at exercise intensities above the ventilatory threshold because RER increases in response to the bicarbonate buffering of the $H^+$ production and the increased release of $CO_2$ in the expired gas.

In summary, we found that compared with men, young normally active boys have higher relative rates of fat oxidation throughout a wide range of exercise intensities and that the exercise intensity that elicits peak fat oxidation rate is considerably higher in boys than in men. Using these same boys studied in a longitudinal design, we show that the high rate of fat oxidation is diminished during maturation, particularly as boys develop from midpuberty (Tanner stage 2/3) to full maturation (Tanner stage 4). This comparatively higher rate of fat oxidation during exercise in prepubertal and early pubertal boys may have important consequences in storing less fat in adipose tissue and may be protective in the development of obesity and its related disorders.

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