Comparison of the Type of Substrate Oxidation During Exercise Between Pre and Post Pubertal Markedly Obese Boys

Abstract

The aim of this study was to investigate, in markedly obese children, the effect of puberty on substrate oxidation during an acute bout of exercise. Two groups of markedly obese boys (7 pre pubertal, 8 post pubertal, matched for adiposity) performed an exercise-test designed for measuring carbohydrate and fat oxidation with indirect calorimetry, and consisting of five six-minute steady-state workloads at 20, 30, 40, 50, and 60% of the theoretical maximal aerobic power. Fat oxidation (mg·min⁻¹) is correlated to fat free mass (FFM) (r = 0.7, p = 0.02). When expressed in crude flow rate units, fat oxidation is slightly higher in PostP than PreP children (p < 0.05). However, when expressed per unit of FFM or as a percentage of total fuel oxidation, fat oxidation is lower in PostP than PreP children (p < 0.05). Multivariable analysis shows that the influence of age on the ability to oxidize fat at exercise is explained by the pubertal increase in FFM. In markedly obese children during puberty, the ability of each kg of FFM to oxidize fat at exercise decreases (~ 28% at 20%Wmax th), but the pubertal increase in FFM overcomes this effect, resulting in an increase in whole body ability to oxidize fat at exercise (+ 17.3% at 20%Wmax th).

Key words
Obesity · children · fat oxidation · indirect calorimetry

Abbreviations

BMI · body mass index
CHO · carbohydrate
FFA · free fatty acids
FFM · fat free mass
PreP · pre pubertal
PostP · post pubertal
RER · respiratory exchange ratio
WHR · waist to hip ratio
Wmax th · theoretical maximal aerobic power

Introduction

The lack of physical activity appears to be one of the most significant factors which explains the increasing prevalence of obesity in children. Indeed, children today are far more sedentary than ever before [20], as sedentary activities such as watching television and playing video games, both risk factors for obesity [40], have greatly supplanted the active physical games of the past.

Skeletal muscle is largely involved in the development of obesity [31]. More precisely, muscular abnormalities could alter the balance of substrate utilization, thus facilitating fat accumulation in

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Bibliography

adipose tissue. In contrast, regular exercise training, generally recommended in obese people, induces muscular metabolic changes, which can reverse these defects [11].

We have recently reported in obese children that two months of exercise training increase fat oxidation during exercise [7]. Although the subjects served as their own controls in this study, it was difficult to differentiate between the effects of training and those of puberty, because the trained group consisted of both pre pubertal (PreP) and post pubertal (PostP) children.

While some authors have investigated the changes in skeletal muscle metabolism that occur during puberty [23,33], there is a paucity of data concerning this issue in obese children [6,42]. However several authors have used skeletal muscle biopsies to investigate skeletal muscle composition and metabolism [13–15]. These studies have shown a possible relationship between anaerobic enzyme activity and maturation, initiating the concept of limited anaerobic capacity in children. Taylor et al. [39] evaluated metabolic responses in the calf muscle of children, younger adults and older adults during exercise. They showed that, at the same relative intensity of exercise, children relied less on glycolysis and more on oxidative metabolism to meet the energy demand. However, others [18] showed no significant difference in adults versus adolescents in citrate synthase activity.

Studies using calorimetry at rest generally indicate that the rate of lipid oxidation is higher in obese children [24,27], and that, in normal weight children, it increases during puberty [3]. However, the influence of both puberty and childhood obesity on the balance of substrates during exercise remains poorly known, although exercise is more and more proposed as an important component of the management of obesity. Recently, exercise calorimetry has been developed by several teams in order to target more closely training protocols in both adults [11,31] and children [7] suffering from obesity. Thus, it becomes important to know to what extent puberty, on its own, modifies the balance of substrates as assessed with this technique in obese children.

Therefore, this study was undertaken in order to investigate the effect of puberty on the ability to oxidize lipids during exercise. We hypothesize that puberty may alter the balance of CHO and fat oxidation during exercise in obese children.

In an attempt to verify this hypothesis, we compared CHO and fat oxidation during exercise between pre and post pubertal obese children. CHO and fat oxidation rates were assessed using calorimetric measurements.

Subjects and Methods

Subjects

Fifteen markedly obese adolescent boys (7 Pre pubertal, 8 Post pubertal) participated in this study. A child was defined as obese when his Body Mass Index (BMI) > 97th percentile defined by the French curves [35]. None of these adolescents had chronic disease, endocrine disorders or diabetes mellitus.

Initial instructions

All subjects were asked to fast for 12 h before the exercise testing. The conditions and requirements of the exercise testing (hospital routine measurements) were explained to each subject.

Evaluation of pubertal stage

Pubertal stage was evaluated according to the Tanner classification [37,38], regarding testicular volume and pubic hair, by a trained physician. The subjects were distributed according to stage of puberty: pre pubertal group (PreP group) who were in Tanner stage 1 and post pubertal group (PostP group) who were in Tanner stages 4 and 5.

Anthropometry

Weigh, height, waist and hip measurements were performed and BMI was calculated as weight in kilograms divided by the square of height in meter (kg/m²). Body composition, as fat mass and fat free mass (FFM), was assessed with a multifrequency bioelectrical impedance (Dietosystem Human IM Scan) using the following frequencies: 1, 5, 10, 50, and 100 kHz. Analysis was performed with the software Master 1.0. In this study, we used Houtkooper’s equation which has been cross-validated in a very rigorous study [19] and which is accurate for subjects aged between 10 and 19 years, so that it was valid for all the subjects of the study. Moreover, hydration conditions were standardized for measuring body composition, and all children were examined in the morning in a fasting state.

The Z-score for BMI was calculated according to Cole [9] and Rolland-Cachera [35], with the following formula:

\[ Z = \left[ (Q/M)^2 - 1 \right] / LS \]

\[ \text{With: } Q = \text{BMI, } M = \text{median, } L = \text{power, } S = \text{coefficient of variation.} \]

Individual’s values of M, L and S were found in the tables of Rolland-Cachera [35].

The BMI z-scores were 4.2 ± 0.3 in the two groups showing that we studied markedly obese children.

Exercise testing

The subjects performed an exercise test on an electromagnetically braked cycle ergometer (Ergoline Bosh 500) connected to a breath by breath device (ZAN 600) for gas exchange measurements (\(\text{VO}_2\) and \(\text{VCO}_2\)).

Before exercise, we calculated the theoretical maximal aerobic power (\(\text{Wmax th}\)) using the Tanner (1991) equation: 3.5 Watts/Fat Free Mass (FFM). As generally used to individualize the increment of exercise testing [36,41], the workload of each step was calculated from the theoretical maximal aerobic power (\(\text{Wmax th}\)), i.e., power corresponding to the theoretical \(\text{VO}_2\text{max}\). In consequence, the subjects underwent a test with the same relative incremental workload and were compared at the same percentage of their Wmax th.

The test consisted of five six-minute steady-state workloads corresponding to 20, 30, 40, 50, 60% of \(\text{Wmax th}\). Heart rate was monitored continuously throughout the test. Ventilatory parameters (\(\text{VO}_2\), \(\text{VCO}_2\)) were recorded during the last three-minutes of
each workload. Pedal frequency was maintained between 60 and 70 rpm throughout the test.

Fat and CHO oxidation

Whole body substrate oxidation was calculated from the measurement of the respiratory exchange ratio (RER = VCO₂/VO₂ in expired gases) in order to determine whole body substrate oxidation. RER is the simplest and most widely used method for determination of fuel utilization. VO₂ and VCO₂ were determined as the means of measurements during the last three-minutes of each workload, according to Mac Rae [7,11,26,31].

The percentages of CHO and fat oxidation were calculated by using the following equations [30]:

\[
\% \text{CHO} = \left(\frac{\left|RER - 0.71\right|}{0.29}\right) \times 100
\]

\[
\% \text{Fat} = \left(\frac{1 - RER}{0.29}\right) \times 100
\]

Fat and CHO oxidation rates were calculated from the gas exchange measurements according to the non-protein respiratory quotient technique [32]:

\[
\text{CHO (mg·min}^{-1}\text{)} = 4.585 \text{VCO}_{2} - 3.2255 \text{VO}_{2}
\]

Fat (mg·min\(^{-1}\)) = \(-1.7012 \text{VCO}_{2} + 1.6946 \text{VO}_{2}\) with gas volume expressed in milliters per minute. CHO and fat oxidation rate were normalized by FFM.

We were thus able to determine the proportion of CHO and fat, which is a function of the balance of substrates oxidized by the body.

After smoothing the curves, we calculated a parameter representative of the balance between fat and CHO utilization induced by increasing exercise intensity: the maximal fat oxidation point [31]. This point (Lipox max) is the point where the increase in fat oxidation induced by increasing workload reaches a maximum, which will then be followed by a decrease as CHO becomes the predominant fuel. It is calculated from equations, determined in previous papers on children [7] and adults [31].

Data analysis

Unpaired Student's t-tests were used to compare subject characteristics. Significant differences between absolute oxidation rates of fat and CHO were determined by using factorial ANOVA. Significance of differences among groups and changes for RER with the exercise intensity were determined by using repeated-measures ANOVA. Post-hoc comparisons were made using Sheffe's test for significant difference. Finally, we used partial correlations analysis to delineate the respective influence of age and FFM on the ability to oxidize fat at exercise. For all statistical analyses, values were expressed as mean (±SEM) and significance was accepted at p < 0.05.

Table 1 Anthropometric characteristics in Pre pubertal (PreP) and Post pubertal (PostP) groups.

<table>
<thead>
<tr>
<th></th>
<th>PreP n = 7</th>
<th>PostP n = 8</th>
<th>p</th>
</tr>
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<tbody>
<tr>
<td>Tanner's stage</td>
<td>I</td>
<td>IV–V</td>
<td></td>
</tr>
<tr>
<td>Age, yr</td>
<td>10.6 ± 0.5</td>
<td>13.5 ± 1.1</td>
<td>0.001</td>
</tr>
<tr>
<td>Body weight, kg</td>
<td>62.9 ± 3.3</td>
<td>94.2 ± 6.2</td>
<td>0.001</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.5 ± 0.02</td>
<td>1.7 ± 0.02</td>
<td></td>
</tr>
<tr>
<td>BMI, kg·m(^{-2})</td>
<td>28.6 ± 1.6</td>
<td>32.2 ± 1.6</td>
<td>NS</td>
</tr>
<tr>
<td>Z-score</td>
<td>4.2 ± 0.24</td>
<td>4.2 ± 0.32</td>
<td>NS</td>
</tr>
<tr>
<td>Body fat, kg</td>
<td>29.3 ± 5.2</td>
<td>33.2 ± 2.6</td>
<td>NS</td>
</tr>
<tr>
<td>Fat free mass, kg</td>
<td>36.5 ± 2.7</td>
<td>60.9 ± 4.02</td>
<td>0.001</td>
</tr>
<tr>
<td>Waist, cm</td>
<td>85.7 ± 3.3</td>
<td>95.7 ± 3.0</td>
<td>0.01</td>
</tr>
<tr>
<td>Hip, cm</td>
<td>99.0 ± 4.7</td>
<td>110.5 ± 3.3</td>
<td>0.05</td>
</tr>
<tr>
<td>WHR</td>
<td>0.86 ± 0.01</td>
<td>0.86 ± 0.02</td>
<td>NS</td>
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</tbody>
</table>

BMI = Body mass index; WHR = waist to hip ratio

Results

Anthropometric characteristics

The PostP group (n = 8), although older and taller than the PreP group (n = 7), had a similar amount of body fat and the same degree of adiposity expressed as a Z-score on BMI charts. Moreover, their BMI failed to be different and their WHR is quite the same. The physical characteristics of the two groups are shown in Table 1. Fat free mass (FFM) showed a significant increase from pre puberty to post puberty. No difference was noted among groups in total body fat (kg).

Respiratory exchange ratio (RER)

RER increased proportionally to the intensity of exercise expressed as relative power output in both groups (p < 0.001). RER values tended to be higher in the PostP group than in the PreP group, but the difference did not reach significance (Fig. 1).

Substrate oxidation

Fig. 2a shows the changes in fat oxidation (expressed in mg·min\(^{-1}\)) during submaximal exercise between the two groups.
Fat oxidation (mg·min⁻¹)

Fig. 2a Fat oxidation (mg·min⁻¹) in PreP (closed circle) and Post (closed squares) groups, with the exercise intensity expressed as relative power output (% Wmax th). * p < 0.05 significantly different between the two groups.

The PostP group relied more on fat during exercise than the PreP group (p < 0.05). Fat oxidation is higher in the PostP than in the PreP group during exercise in obese children (for example, + 17.3% at 20% Wmax th). However, since FFM increases during puberty, we normalized fat oxidation for FFM in order to neutralize a possible effect. Accordingly, Fig. 2b shows the same pubertal changes in fat oxidation during sub maximal exercise, expressed as mg·min⁻¹·FFM⁻¹. In this case, the PostP group relied less on fat during exercise than the PreP group (p < 0.05). Therefore, when fat oxidation is expressed per kg of FFM, puberty seems to decrease the use of fat during exercise in obese children (for example, – 28% at 20% Wmax th).

The power intensity at which the Lipox max occurs (expressed in % Wmax th), is higher in PostP compared to PreP children (Fig. 3). This result indicates that during puberty, the level of exercise intensity at which fat oxidation is maximal is increased.

Figs. 4a and b show the relationships among fat oxidation at Lipox max, FFM and age. Expressed in mg·min⁻¹, the rate of fat oxidation at the level of the Lipox max increases proportionally with both age (r = 0.66; p = 0.03) and FFM (r = 0.7; p = 0.002). When this fat oxidation is expressed per kg of FFM, there is a non significant tendency to decrease when age increases (data not shown), but a significant decrease when FFM increases (r = –0.66; p = 0.006) (Fig. 5). In order to delineate the respective influence of age and FFM on the ability to oxidize fat at exercise, we performed partial correlations analysis among age, fat oxidation in mg·min⁻¹ and FFM. If the variable “FFM” is kept constant by the statistical procedure, the correlation between age and fat oxidation disappears (r = 0.417, ns), indicating that the variable “fat oxidation” is correlated to age only because age is correlated to FFM. FFM is thus the explanatory variable of the increase in whole body lipid oxidation associated with the increase in age during the pubertal period. On the whole, this partial correlation analysis indicated that in PostP children, there is an increase in the ability to oxidize fat at exercise only because FFM increases during puberty, while the ability to oxidize fat expressed per kg of FFM rather decreases.

Fig. 6 shows the percentage of fat and CHO oxidation at each workload of exercise in PreP and PostP groups. During rest and mild to moderate exercise, fat remains the predominant source of energy for oxidation, while a shift towards CHO oxidation occurs at exercise above 50% Wmax th (p < 0.01). This figure shows that the PostP group has a greater dependence on CHO than the PreP group. At 20% Wmax th, PostP children oxidize a significantly lower percentage of fat and a higher percentage of CHO compared to the PreP group (64.6%±2.03 vs. 74.4%±4.9 for fat and 35.4%±2.03 vs. 25.6%±4.9 for CHO, p < 0.05).

Fig. 7 shows substrate oxidation rates (expressed as mg·min⁻¹·FFM⁻¹) at each workload in PreP and PostP groups. Fat oxidation rates were lower in the PostP group compared to the PreP group (p < 0.01) at 20, 30, 40% Wmax th (F = 7.92, F = 7.14, F = 7.81, respectively). CHO oxidation (mg·min⁻¹·FFM⁻¹) increased with exercise intensity in both groups (p < 0.001) without any significant difference between PreP and PostP groups.

The increase of exercise intensity, in Fig. 8, results in a significant difference in the contribution of the relative rate of substrate oxidation between PreP and PostP groups. Actually, the rate of fat

Fat oxidation at Lipox max (mg·min⁻¹)

![Graph](image)

Fig. 4a Relationship between fat oxidation at Lipox max (mg·min⁻¹) and age (years) \( r = 0.66, p = 0.03 \).

Fat oxidation at Lipox max (mg·min⁻¹·FFM⁻¹)

![Graph](image)

Fig. 5 Relationship between fat oxidation at Lipox max (mg·min⁻¹·FFM⁻¹) and FFM \( r = -0.66, p = 0.006 \).

Fat oxidation at Lipox max (mg·min⁻¹·FFM⁻¹) & FFM

![Graph](image)

Fig. 6 Percentage of fat and CHO oxidation rates, in relation to the exercise intensity expressed as relative power output (% Wmax th). PreP: Closed circle = fat %, closed triangle = CHO %; PostP: Open circle = fat %, open triangle = CHO %. * \( p < 0.01 \) significantly different between fat and CHO oxidation in both groups; ‡ \( p < 0.05 \) significantly different between PreP and Post in fat oxidation; ? \( p < 0.05 \) significantly different between PreP and Post in CHO oxidation.

Substrate oxidation (kcal·min⁻¹·FFM⁻¹) & % Wmax th

![Graph](image)

Fig. 7 Mean absolute substrate oxidation rates (kcal·min⁻¹·FFM⁻¹) in PreP and PostP groups with the exercise intensity (% Wmax th). $p < 0.01$ significant difference in fat oxidation between the two groups.

oxidation was significantly lower in the PostP group than in the PreP group but this difference was only significant at 20 and 30% Wmax th \( p < 0.05, F = 5.56; p < 0.03, F = 5.22 \), respectively.

**Discussion**

Our results show that in markedly obese children, puberty is associated with significant alterations in the balance of substrate oxidation during exercise. The ability to oxidize fat, which is pro-

portional to fat free mass, actually decreases when it is expressed per unit of fat free mass or as a percentage of total fuel oxidation. However, when expressed in crude flow rate units, it increases slightly, but this effect is only due to the gain in fat free mass during this period which prides, to some extent, the diminishing effect of puberty.

Gender, age and adiposity may all be confounding factors, and both the body mass index and the percentage of fat increase during puberty. For this reason, we have tried to very carefully match our subjects. Comparison of BMI measurements, expressed as Z-scores, shows that our subjects exhibit the same degree of overweight. The Z-score indicates the degree to which an individual’s measurement deviates from what is expected from the height charts. Thus, a Z-score of 0 is equivalent to the 50th percentile and a Z-score of +2.0 is equivalent to the 97th percentile. In our study, a similar value of 4.2 was found for the Z-score of the BMI in both groups. Such a Z-score confirms morbid obesity and shows that PreP and PostP children were correctly matched in terms of adiposity.

Indirect calorimetry is a usual method to quantify substrate oxidation rates at rest and during exercise. Nevertheless, during exercise above the lactate threshold, the accuracy of the technique has been discussed. The main concern is that lactate acidosis increases CO₂ production and is thus likely to increase the RER independent of the balance of substrates. However, the contribution of bicarbonate-derived CO₂ to VCO₂ has been studied and seems to be rather negligible, so that the RER remains a fair mirror of the balance of substrate oxidations during exercise [21]. These methodological aspects of the validity of the RER technique during six-minute workloads have been extensively discussed in some of our previous papers [31]. On the whole, exercise calorimetry appears to be valid for measurements of substrate oxidation during submaximal steady-state exercise bouts. Such measurements have shown that obese people oxidize less lipids at exercise than lean matched controls [31] and that low intensity exercise training markedly reverses this defect in both adult and adolescent obese subjects [7,11,28].

To individualize the increment of exercise testing [36,41], the workload of each step was calculated from the theoretical maximal aerobic power (Wmax th), i.e., power corresponding to the theoretical VO₂max. Moreover, to avoid overestimating Wmax th in obese children in connection with an excess of fat mass, we used the Tanner formulas, taking into account the fat free mass.

The exercise test was designed to make children exercise at the same relative working intensities, expressed as percentage of theoretical Wmax, and substrate oxidation rates were normalized by fat free mass. Thus, our findings are not due to a difference in age resulting in a difference in exercise tolerance for similar work loads. Actually, if it were the case, we would observe a better tolerance of exercise in older children, and thus presumably a higher lipid oxidation at similar work loads. Due to the correct matching of children, we find the opposite and observe that children exhibit a lower ability (per kg of FFM) to oxidize fat at exercise after puberty.

Our finding of alterations in substrate oxidation during sub maximal exercise after puberty in markedly obese children should be interpreted taking into account the fact that obese children exhibit excess of both fat mass and fat free mass (FFM). In addition, during puberty, boys gain lean tissue, especially skeletal muscle. However, during this period of life, skeletal muscle becomes insulin resistant [5,10]. This insulin resistance is likely to impair metabolic flexibility [22], i.e., muscle ability to oxidize CHO at rest and fat at exercise are both impaired. In other terms, the insulin-resistant muscle is unable to adequately shift its balance of substrate oxidations from the “resting pattern” to the “exercise pattern” and thus burns too little CHO at rest and too little fat at exercise [22]. This mechanism is consistent with our finding that the whole capacity for fat oxidation at exercise (mg·min⁻¹) increases with puberty only because FFM increases, but if this influence of FFM is neutralized in order to analyze only the effect of puberty, the ability to oxidize fat (mg·min⁻¹·FFM⁻¹) during exercise decreases in markedly obese children. Several authors have previously reported an increase in fat oxidation with puberty in normal weight children [3], but in those studies, fat oxidation was not measured during exercise, but during a hyperinsulinemic euglycemic clamp. Therefore, this increase in fat oxi-
dation at rest does not rule out the possibility of an impairment of the ability to oxidize fat at exercise due to the loss of metabolic flexibility that characterizes muscular insulin resistance.

Whatever the exact mechanism of this defect of fat oxidation at exercise, it is interesting to note that it occurs at a period where insulin sensitivity is also known to markedly decrease [5,10]. Muscular insulin resistance, which is quite common in obese adults, appears also to be associated with alterations of substrate balance. There is also, in this case, a decrease in fat oxidation ability and an early shift from lipid towards CHO-derived fuel, compared to lean people [31], consistent with the above mentioned concept of impaired metabolic flexibility [22]. However, the physiological mechanism linking low insulin sensitivity to low fat oxidation in muscle is unclear.

Concerning puberty, however, this issue is even more complicated, since the decrease in insulin sensitivity during this period of life is a physiological event and not a disease, and its mechanism is not well understood. This decrease in insulin sensitivity is likely to be associated with other physical and hormonal changes (including changes in growth hormone and testosterone which are involved in the development of FFM). Recent studies that investigated a lot of possible hormonal mechanisms suggested that a possible candidate to explain pubertal insulin resistance is the rise in plasma androstenedione that occurs during this period [17].

Whatever the hormonal background, at the muscular level, some previous studies show evidence of a possible conversion of fibre phenotype towards fast twitch during puberty in lean children. This mechanism is likely to result in a higher rate of glycolysis. Actually, there is a relative paucity of muscle biopsy studies in children compared to adults, because of an obvious ethical limitation. Some authors have shown that children have a higher proportion of slow twitch fibres in the vastus lateralis muscle as compared with untrained adults [4,15]. The proportion of fast twitch fibres increases significantly with age from the age of 5, caused by a transformation of slow twitch to fast twitch [25]. This difference disappears during late adolescence [16].

Another finding of this study is that, as already known in adults, substrate utilization shifts as a continuum from a greater dependence on fats at low intensities of 20, 30 and 40% Wmax th, which is the zone of lipid oxidation, towards a preferential use of CHO at exercise above 50% Wmax th, when exercise intensity increases. The physiological relevance and the mechanism of this “crossover” of substrates have been extensively investigated by the team of G. Brooks [8]. The shift in substrate selection with exercise intensity is undoubtedly under multiple regulatory controls. In this study, the PostP children exhibited an earlier shift from fat to CHO-derived fuels during exercise than PreP children. Fat oxidation, in percentage, was lower during exercise in PostP group.

A recent study [34] demonstrated that in healthy weighting 11 – 12 year-old boys, maximal fat oxidation occurs around 56% $V\Omega_{\text{max}}$ (8.18 mg·min$^{-1}$·FMM$^{-1}$) during an incremental type exercise protocol derived from that of Achten [1]. In our study, we show that the maximal fat oxidation (Lipox max) occurs around 50% Wmax th (6.94 mg·min$^{-1}$·FMM$^{-1}$) in PreP boys and around 47% Wmax th (5.43 mg·min$^{-1}$·FMM$^{-1}$) in PostP boys. So, in comparison to the team of Riddell [34], maximal fat oxidation seems to be lower in obese PreP boys than healthy PreP boys. Moreover, we observed that PostP boys have a lower maximal fat oxidation, because of the FFM parameter. We did not use the same protocol as Riddell et al. for determining maximal fat oxidation. While they used a protocol derived from that of the team of Achten (12.5 Watts increments, 3 min intervals vs. 35 Watts, 5 min), in our study, we used an individualized protocol with an increment of 10% of Wmax th every 6 min. Despite this difference between protocols, it appears that obese PreP children have a lower maximal fat oxidation (representing a lower capacity to oxidize fat during exercise) than healthy PreP children, and higher maximal fat oxidation than PostP children.

It is, of course, interesting to try to further elucidate mechanisms which lead to a lower fat oxidation in the PostP group than the PreP group. However, this finding of an impairment of the ability to oxidize fat at exercise during puberty in already markedly obese children may be by itself an important issue for clinicians involved in the management of pubertal morbid obesity. It may help to explain the worsening of their overweight during this period, if less fat is burned at exercise. Moreover, since endurance training is known to markedly reverse this shift in the balance of substrates in both adults [12,29] and children [7], such findings emphasize the importance of regular exercise at this particular period of life in the management of obesity. They also demonstrate the importance of exercise training protocols at lower intensities [2] in the late pubertal markedly obese adolescents in order to utilize relatively more fat.

Conclusion

The current study shows that puberty alters the balance of substrate oxidation during exercise in markedly obese children. These modifications depend on fat free mass, which increases during puberty. In order to elucidate the specific effect of puberty, we corrected results for FFM, and so show that PostP obese children rely less on fat oxidation (mg·min$^{-1}$·FMM$^{-1}$) during exercise than PreP obese children. Exercise at the same relative sub maximal intensity elicits a higher CHO utilization in PostP children. We suggest that this process may be involved in the difficulty to manage morbid obesity during puberty, and that such findings strongly emphasize the importance of exercise at this period of life in obese children or children prone to obesity. However, more studies will be required, although ethical and methodological considerations obviously limit invasive research in children.

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