Impact of high- and low-intensity targeted exercise training on the type of substrate utilization in obese boys submitted to a hypocaloric diet

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SUMMARY

Background: We assessed the effect of two programs combining a hypocaloric diet with low-intensity (LI) or high-intensity (HI) exercise training, during two months, on substrate utilization at exercise in obese children.

Methods: Fifteen obese boys participated in a combined program of exercise and caloric restriction-induced weight loss (diet starting two weeks before the training program). The maximal fat oxidation point (Lipox max) was determined to individualize exercise training. Training consisted of cycling at either LI (Lipox max) for seven children or HI (Lipoxmax+40% Lipox max) for eight children.

Results: All children exhibited a decrease in weight (LI: -5.2 kg ∂ 0.7 ($P < 0.01$), HI: -7 kg ∂ 0.7 ($P < 0.01$)). While in the LI group, both fat and CHO oxidation were unchanged after training, HI group oxidize less fat and more CHO after training when exercising at 20% and 30% Wmax th ($P = 0.02$).

Discussion: While a LI exercise training program maintains (but does not improve) the ability to oxidize fat at exercise, HI training actually shifts towards CHO the balance of substrate oxidation during exercise. Thus, a low intensity training protocol seems to counteract to some extent the decline in lipid oxidation at exercise that occurs after a hypocaloric diet, and is thus likely to be synergistic to diet in the weight lowering strategy.

Key-words: Obesity · Children · Low-intensity training · High-intensity training · Lipox max · Diet.

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RÉSUMÉ

Effet de deux types d'entraînement (basse et haute intensité) sur le type d'oxydation des substrats énergétiques, chez des enfants obèses soumis à un régime hypocalorique

Contexte : Le but de notre travail était d'évaluer l'effet de deux types d'entraînements d'une durée de deux mois fondés sur des intensités de travail différentes (basse et haute) associés à un régime hypocalorique sur l'utilisation des substrats énergétiques au cours de l'exercice chez l'enfant obèse.

Méthodes : Quinze enfants obèses ont participé à un programme combinant exercice et régime (celui-ci a débuté deux semaines avant l'entraînement). Le point d'utilisation maximal des lipides (Lipox max) a été déterminé pour individualiser l'entraînement à basse intensité (Lipox max) pour sept enfants et haute intensité (Lipox max + 40 % Lipox max) pour huit autres enfants.

Résultats : Tous les enfants ont perdus du poids (basse intensité : -5,2 ∂ 0,7 kg, $P < 0,01$; haute intensité : -7 ∂ 0,7 kg, $P < 0,01$). Alors que pour le groupe à basse intensité l'oxydation des lipides et des glucides reste inchangée après l'entraînement, l'oxydation des lipides est diminuée et celle des glucides augmentée pour le groupe haute intensité à 20 % et 30 % Pmaxth.

Discussion : L'oxydation des substrats est maintenue pour le groupe basse intensité et modifiée (diminution des lipides, augmentation des glucides) pour le groupe haute intensité. L'entraînement à basse intensité semble agir sur le déclin de l'oxydation des lipides provoqué par un régime hypocalorique et doit être ainsi effectué en même temps que le régime pour une meilleure stratégie de perte de poids.

Mots-clés : Obésité · Enfants · Entraînement à basse intensité · Entraînement à haute intensité · Lipox max · Régime.

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Introduction

Several muscular abnormalities, in obese subjects, leading to alterations in muscle substrate metabolism, have been described. Obese subjects exhibited an alteration of the balance of substrate oxidation during exercise, towards an impairment of their fat oxidation ability and an early CHO dependency during exercise compared to controls subjects [1]. In contrast, exercise training induces muscular metabolic adaptations, which can restore these abnormalities. In a previous study [2], we assessed the impact of low-intensity exercise training associated to diet program in obese children during two months. Low intensity was chosen as an intensity at which lipid oxidation was maximal (Lipox max). The diet program consisted on a hypocaloric restriction setting at - 300 kcal/day below the actual energy requirement. We reported that obese children exhibit an increase in their ability to oxidize lipids at exercise after such a low-intensity training. Similar protocols at low intensity have been commonly used in literature [3-7], because the proportion of fat oxidized is higher than that observed during a vigorous effort. Beyond the fact that this is a safe exercise prescription, low-intensity exercise seems also recommended. Actually, data are sometimes conflicting, regarding the training intensity. However, exercise intensity is one of the main factors that influence substrate utilization during exercise. Some studies have focused on benefits of resistance training or high-intensity of exercise [8, 9], because high-intensity training may be more effective in increasing post-exercise fat oxidation [10].

These controversies indicate that a clear consensus on exercise prescription for optimally improving substrate oxidation is still lacking. Because fat oxidation is higher at low-intensity during acute exercise [1, 11], we hypothesize that low-intensity exercise training associated to diet, leads to a better use of fat oxidation during exercise in obese children, in comparison to high-intensity training. Therefore, the aim of this study was to determine how a program which includes training (at either low- or high-intensity) and diet (caloric restriction), modifies the ability to oxidize fat during exercise.

In an attempt to answer this question, we tested the effect of two programs combining a hypocaloric diet with low-intensity (LI) or high-intensity (HI) exercise training, during two months, on substrate utilization at exercise in obese children. We employed indirect calorimetry, a non invasive method, to measure substrate utilization during exercise.

Materials and methods

Patients

Fifteen obese boys (5 pre pubertal, 2 pubertal and 8 post-pubertal) were recruited. A child was defined as obese when his Body Mass Index (BMI) > 97th percentile defined by the French curves [12]. None of these adolescents had chronic disease, endocrine disorders or diabetes mellitus. Subjects were matched for age and pubertal stage. The subjects were randomly assigned to one of two program groups: low-intensity (LI) and high-intensity (HI) exercise training group. LI group comprised seven subjects and HI group eight.

The study was made after written informed consent by complying ethics rules usually applied in this type of study.

Experimental design

This protocol consists of a comparative study between two programs intensities of exercise training (low- and high-intensity) and diet, before and after two months, of anthropometric and metabolic parameters. More precisely, subjects participated in a combined program of exercise and caloric restriction-induced weight loss. However, the diet program, which consisted to reduce their caloric intake, began two weeks before the training program.

Measurements before and after exercise training

Initial Instructions

All subjects were asked to fast for 12 hours before exercise testing. The conditions and requirements of the exercise testing were explained to each subject.

Evaluation of the pubertal stage

Pubertal stage was evaluated according to the Tanner classification [13, 14], regarding testicular volume and pubic hair, by a trained paediatrician: pre pubertal children comprised children who were in Tanner stage I, pubertal children in Tanner stage II-III, post pubertal children in Tanner stage IV-V.

Anthropometry

Weigh, height waist and hip measurements were performed and BMI was calculated as weight in kilograms divided by height in meter squared (kg/m^2). Body composition as fat mass and fat free mass (FFM), was assessed with a multifrequency bioelectrical impedance (Dietosystem Human IM Scan) using following frequencies: 1, 5, 10, 50, and 100 kHz. Analysis was performed with the software Master 1.0. The Z-score for BMI was calculated according to Cole [15] and Rolland-Cachera [12], with the following formula:

$Z = [(Q/M)^L - 1]/LS$

With: Q = BMI, M = median, L = power, S = coefficient of variation. Individual's values of M, L and S were determined according to Rolland-Cachera [12].

Exercise testing

The subjects performed an exercise testing on an electromagnetically braked cycle ergometer (Ergoline Bosh 500) connected to a breath by breath device (ZAN 600) for gas exchange measurements (VO_2 and VCO_2). Before exercise, we calculated the theoretical maximal power (Wmax th) using the Tanner (1991) equation for boys: 3.5 Watts.FFM^{-1} (Fat Free Mass). As generally used to individualize the increment of exercise testing [16, 17], the workload of each step was calculated from the Wmax th, i.e., power corresponding to the theoretical VO_2max. In consequence, the subjects underwent a test with the same relative incremental workload and were compared at the same percentage of their Wmax th. Individual incremental workload determined by Wmax th was the same before and after the training.

The test consisted of a progressive five six-minute steady-state workloads corresponding to 20, 30, 40, 50, 60% of Wmax th. Heart rate was monitored continuously throughout the test. Ventilatory parameters (VO_2, VCO_2) were recorded during the last three-minutes of each workload, according to Mac Rae [18], for calculations of the substrate oxidation flow rates.

Fat and CHO oxidation

Whole body substrate oxidation was calculated from the measurement of the respiratory exchange ratio (RER = VCO_2/VO_2 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. VO_2 and VCO_2 were determined as the means of measurements during the last three-minutes of each workload, as previously described [1, 2, 18, 19].

The percentage of CHO and fat oxidation were calculated by using the following equations [20]:

% CHO = ((RER-0.71)/0.29) Δ 100

% Fat = ((1-RER)/0.29) Δ 100

Fat and CHO oxidation rates were calculated from the gas exchange measurements according to the non-protein respiratory quotient technique [21]:

CHO ($mg.min^{-1}$) = 4.585 VCO_2 – 3.2255 VO_2

Fat ($mg.min^{-1}$) = -1.7012 VCO_2 + 1.6946 VO_2

with gas volume expressed in milliters per minute. CHO and fat oxidation were normalized by FFM. We are thus able to determine the proportion of CHO and fat used from the RER, 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 [1]. 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 as previously described in children [2], and in adults [19]. We used this point for individualize training.

Exercise training

The exercise training was offered 2d.wk^{-1} for 2 months. An exercise prescription was developed for each subject based on data from baseline exercise testing. First, the determination of Lipox max for each child was performed, for determining the exercise intensity program. The exercise training consisted of cycling on an ergometer (Ergoline Bosh 500) at either LI (Lipox max) or HI (Lipox max + 40% du Lipox max). Seven subjects participated in the LI and eight subjects in the HI training program. We decided to hold estimated energy expenditure constant at 10 Kcal.kg FFM^{-1} regardless of group assignment. Indeed, LI exercise requires a longer duration to expend a similar number of calories compared to HI exercise. Therefore, the LI training group were scheduled to exercise 35 min and the HI group to 20 min. So the time was 35 min (∂ 1.7) for low-intensity group and 20 min (∂ 1.1) for high-intensity group. During every exercise session, heart rate (HR) was monitored continuously. Each subject worked at the heart rate corresponding to LI and HI for the training sessions. All training sessions took place at the laboratory under the supervision of a sport physician.

Diet

A balanced and personalized hypocaloric diet was established by a dietician after an initial dietary assessment in order to define the total amount of calories consumed per day. The diet is set at - 300 Kcal/day below the actual energy requirement. It is composed of 17% Proteins, 30% Lipids, 53% CHO. Children had already started the diet two weeks before the onset of the training sessions.

Data analysis

Unpaired Student's t-tests were used to compare subjects' characteristics. Significant differences between absolute oxidation rates of fat and CHO were determined by the use of one way factorial ANOVA. Significance of differences among groups at various levels of exercise intensity the exercise intensity was determined by using repeated-measures ANOVA. Post-hoc comparisons were made using Sheffe's procedure for significant differences. For all statistical analyses, values were expressed as mean (∂ SEM) and significance was accepted at $p < 0.05$.

Results

Anthropometric characteristics

Data show that the two groups are correctly matched for age and pubertal stage. They have a similar amount of

body fat and the same degree of adiposity expressed with a Z-score on BMI charts *(Tab I)*. Training parameters are summarized in *Table II*.

Anthropometric parameters that measure adiposity show a significant reduction in the two groups. For the LI group, weight loss was -5.2 kg ∂ 0.7 ($P < 0.01$), fat mass loss was -5.07 kg ∂ 1.7 ($P < 0.03$). Fat free mass was unchanged. For HI group, weight loss was -7 kg ∂ 0.7 ($P < 0.01$), fat mass loss was -5.8 kg ∂ 0.9 ($P < 0.01$). There was a non significant tendency towards a reduction in fat free mass of -1.2 kg ∂ 0.9 in HI group ($P = 0.08$).

When we compared the anthropometric parameters changes after two months program between the two groups, there were no significant differences *(Fig 1)*.

Substrate oxidation

Figure 2a shows substrate oxidation rates normalized by FFM at each workload of exercise intensity in the LI group before and after training. Fat and CHO oxidation rates do not change after two months of training. By contrast, in the HI group, substrate oxidation rates are different after training at 20 and 30% Wmax th *(Fig 2b)*, with a decrease in fat oxidation and an increase in CHO oxidation at 20 and 30% Wmaxth.

Figure 3a and 3b show the percentage of fat and CHO oxidation at each workload of exercise in LI and HI group between pre and post training. During rest and mild-to moderate exercise, fat predominates as energy source while a shift towards CHO oxidation occurs at higher intensities ($p < 0.001$). In the LI group, both fat and CHO oxidation are unchanged after training *(Fig 3a)*. *Figure 3b* shows that after training, the HI group oxidizes a lower percentage of fat and a higher percentage of CHO during exercise. At 20 and 30% Wmax th, HI group oxidize a significant lower percentage of fat and a higher percentage of CHO after training. (51.4 ∂ 1.6 *vs* 63.8 ∂ 4.4 at 20% Wmax th, 42.8 ∂ 3.2 *vs* 55.4 ∂ 3.9 at 30 % Wmax th for fat, and 48.6 ∂ 1.6 *vs* 36.1 ∂ 4.4 at 20% Wmax th, 57.1 ∂ 3.2 *vs* 44.6 ∂ 3.9 at 30% Wmax th fo CHO, $p < 0.02$).

The comparison of LI and HI group does not show any significant difference in substrate oxidation during exercise before *(Fig 4a)* and after exercise training *(Fig 4b)*.

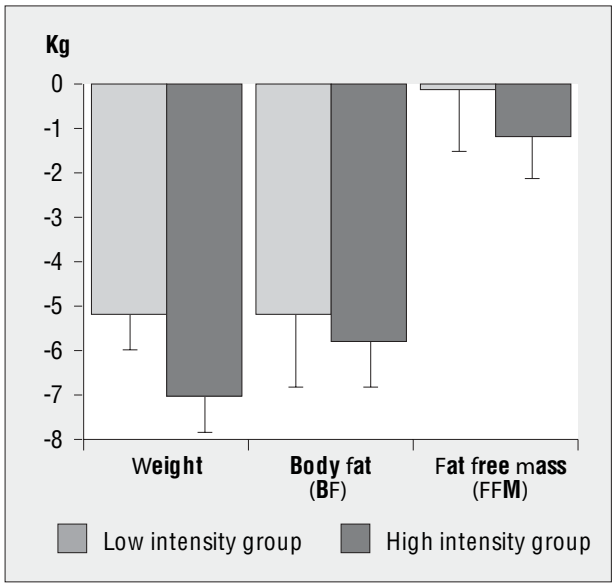

Figure 1
Weight loss (in kg) between pre and post training.LI = low intensity group; HI = high intensity group; BF = body fat; FFM = fat free mass.

Table I
Anthropometric characteristics of children of the low intensity (LI) and the high intensity (HI) groups.

	LI n = 7	HI n = 8	p
Pubertal status male (I / II-V)	2/5	3/5	
Age [range]	11.8 ∂ 0.5 [10-14]	12.8 ∂ 0.5 [11-15]	NS
Weight, kg	77.7 ∂ 5.9	86.7 ∂ 8.3	NS
Height, m	1.61 ∂ 0.03	1.68 ∂ 0.03	NS
BMI, kg/m^2	29.5 ∂ 1.7	30.1 ∂ 1.7	NS
Z-score	3.98 ∂ 0,18	4.13 ∂ 0.31	NS
Fat mass, kg	28.9 ∂ 3.7	29.1 ∂ 3.9	NS
Fat free mass, kg	48.8 ∂ 3.03	57.6 ∂ 6.01	NS

BMI=body mass index.

Table II
Training parameters at Lipox max (LI) and at Lipox max + 40% (HI).

	LI n = 7	HI n = 8	P
Total increase in energy expenditure over 2 months (KJ)	198 688 ∂ 18 650	195 000 ∂ 25 230	NS
Intensity (% Wmax th)	50.8 ∂ 2.6	60.8 ∂ 5.3	NS
Intensity (Watts)	85.1 ∂ 5.6	120.8 ∂ 12.2	0.02
Duration (min)	25.7 ∂ 2.8	19.8 ∂ 1.4	0.01
Heart rate (bpm)	148.2 ∂ 3.06	156 ∂ 3.4	NS

Discussion

Our study shows that, in obese boys already submitted to a balanced hypocaloric diet, the effect of exercise training is influenced by its intensity level. While a low-intensity exercise training program maintains (but does not improve) the ability to oxidize fat at exercise, a high intensity training

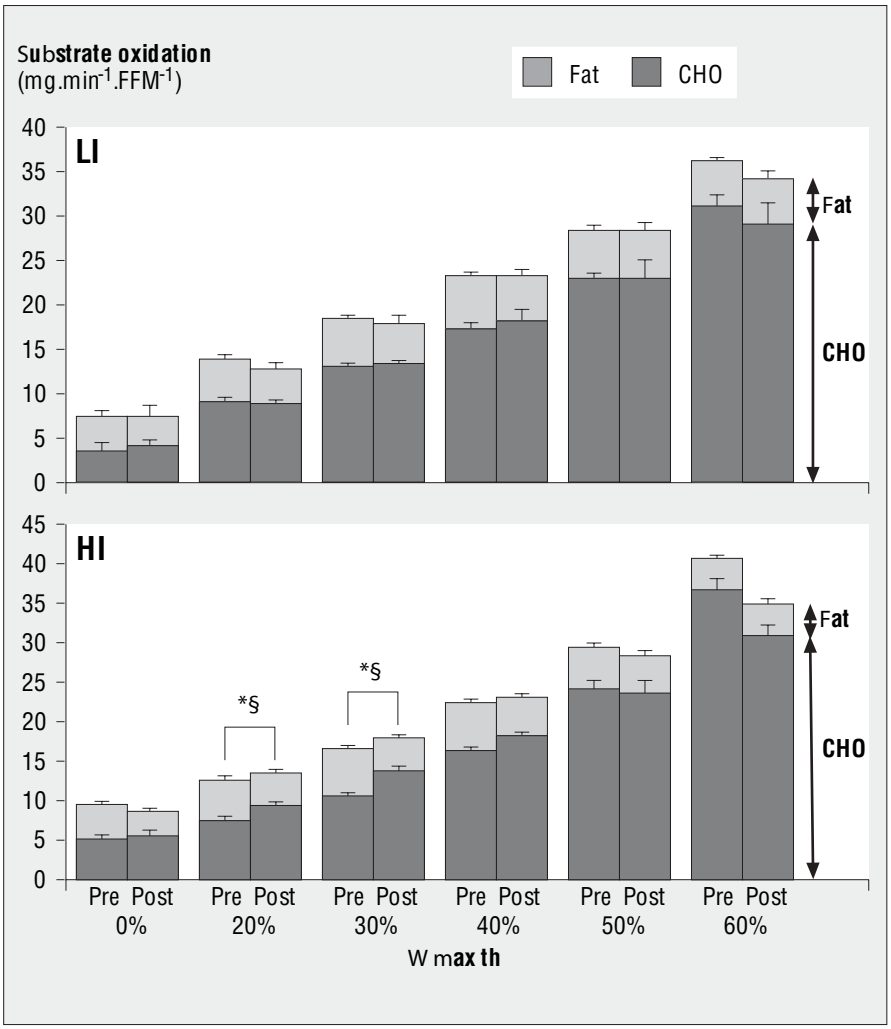

Figure 2
2a. Mean absolute substrate oxidation rates ($mg.min^{-1}.FFM^{-1}$) in the LI group. Comparison between pre and post exercise training. Pre = pre training; Post = post training.
2b. Mean absolute substrate oxidation rates ($mg.min^{-1}.FFM^{-1}$) in the HI group. Comparison between pre and post exercise training.
* $P < 0.05$ significant difference in fat oxidation between pre and post training.
§ $P < 0.03$ significant difference in CHO oxidation between pre and post training.

actually shifts towards CHO the balance of substrate oxidation during exercise.

It is important to emphasize that the design of this study, which consists of comparing two training protocols in children already submitted to a restrictive diet, is different from other studies in which training was studied isolately, or in which both training and diet were simultaneously undertaken. This design is likely to explain some discrepancies with other previous studies. Actually it was applied here in order to fit more closely with a usual clinical situation, where long term dietary prescriptions are employed, standardized training protocols being added later for a more limited duration of time. Thus, the baseline situation in this study is that of children already submitted to a restrictive diet, which may, as discussed later, induce by its own a modification of the balance of substrates at exercise.

The matching of our two groups of children is an important issue, since gender, age and adiposity may all be confounding factors, while both the body mass index and the percentage of fat increase during puberty. For this reason we tried to match very carefully 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 50^{th} percentile and a Z-score of +2.0 is equivalent to the 97^{th} percentile. In our study, Z-scores of respectively 3.98 and 4.13 are found for the Z-score for the BMI in LI and HI groups, ie, quite similar values that are not statistically different. Such a Z-score confirms obesity and shows that LI and HI children were correctly matched in terms of adiposity.

During exercise, the intensity and duration will determine the energy expenditure. Because the high-intensity physical training group used more energy per minute, they were scheduled to exercise for fewer minutes per session than the low-intensity group.

It should be pointed out that the individual level of exercise training calculated from the results of exercise calorimetry occurs at a level of the theoretical maximal heart rate (% HRmax) which is highly variable among individuals. If we apply the equations of Londeree [22], for converting

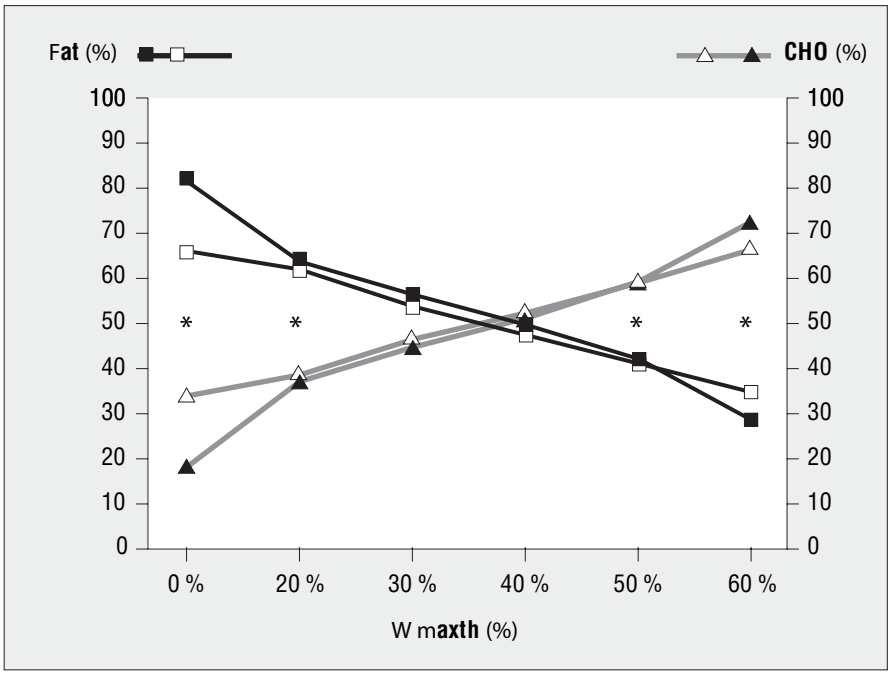

Figure 3a
Fat and CHO oxidation rate, expressed as percentage, as the function of exercise intensity expressed as relative power output in the low intensity (LI) group.Closed square = Fat (%) and closed triangle = CHO (%) before training.Open square = Fat (%) and open triangle = CHO (%) after training.
* $P < 0.0001$ significant difference between fat and CHO oxidation on the whole group, before and after training.

% VO_2max into % HR max, we obtain 65-77% for LI group and 60-77% for HI group, so that we cannot give theoretical general recommendations for a level o % HR max that would correspond to the targeted levels of exercise used in this study. Unless further research provides us predictive equations for the Lipox max suitable in this population, exercise calorimetry seems to be indispensable for targeting exercise training at a defined level of lipid oxidation.

In the current study, LI exercise training seems thus to maintain but not to increase, fat oxidation during exercise in obese children. It may be considered as surprising because in the literature, endurance exercise training is known to enhance fat oxidation during sub maximal exercise [2, 7, 19]. Although training for endurance exercise markedly enhances the muscle's capacity to metabolize fat, several factors regulate the lipids utilization during exercise. One of them is diet. Hypocaloric diet modifies substrate utilization during exercise, since it induces a decline in fat oxidation [23]. According to the kind of diet used, this decline will be more or less marked. Indeed, we know that

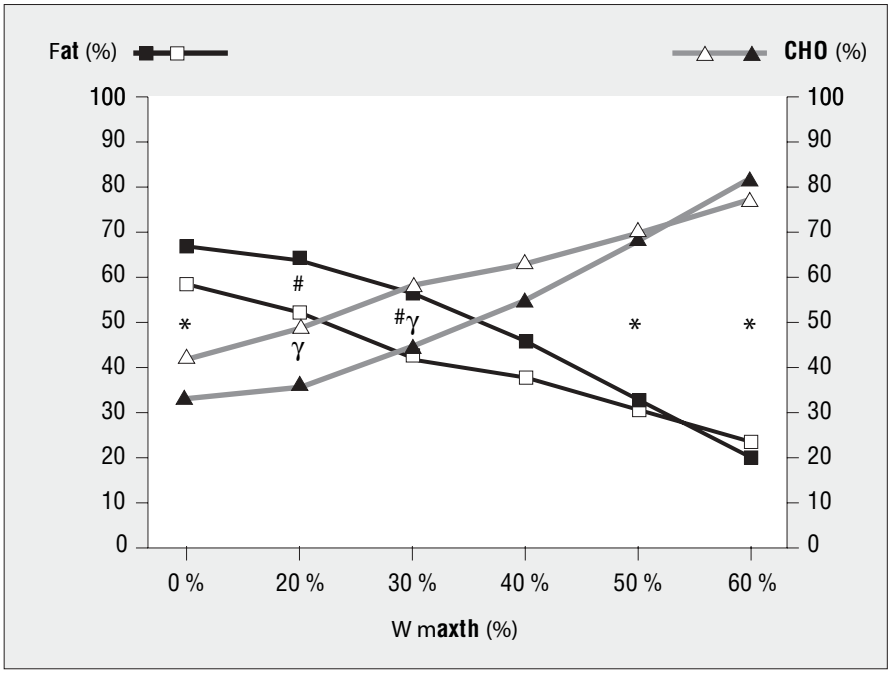

Figure 3b
Fat and CHO oxidation rate, expressed as percentage, as the function of exercise intensity expressed as relative power output in the high intensity (HI) group.Closed square = Fat (%) and closed triangle = CHO (%) before training.Open square = Fat (%) and open triangle = CHO (%) after training.
* $P < 0.0001$ significant difference between fat and CHO oxidation, on the whole group, before and after training.
# $P < 0.02$ significant difference in fat oxidation between pre and post training.
ν $P < 0.2$ significant difference in CHO oxidation between pre and post training.

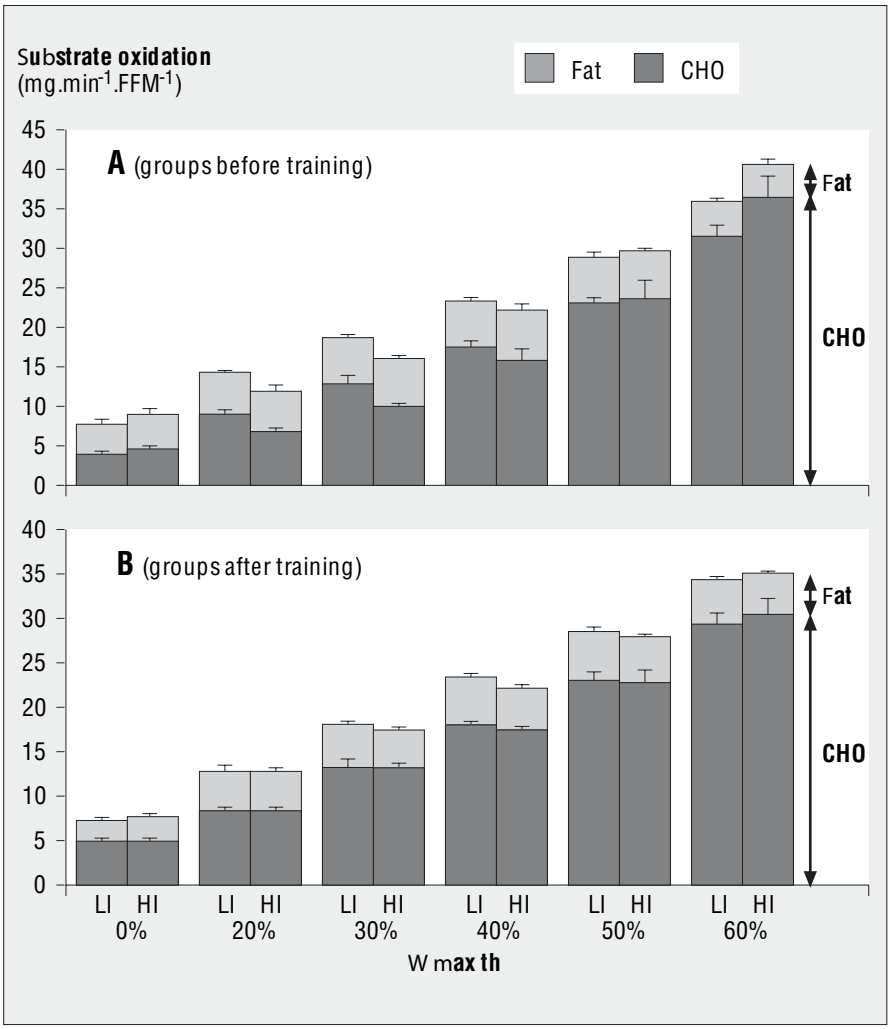

Figure 4
4a. Mean absolute substrate oxidation rates ($mg.min^{-1}.FFM^{-1}$) between low intensity (LI) and high intensity (HI) groups before training.4b. Mean absolute substrate oxidation rates ($mg.min^{-1}.FFM^{-1}$) between low intensity (LI) and high intensity (HI) groups after training.

caloric restriction decreases adipocyte lipolytic responsiveness due to reduced expression and activation of hormone-sensitive lipase (HSL), the rate-limiting enzyme for lipolysis [24, 25]. Caloric restriction also leads to decline in fat oxidation [23]. Moreover, some authors [23, 26] showed that fat oxidation is lower after a period of restricted caloric intake and subsequent weight loss. In our study, caloric restriction was started 2 weeks before the onset of the training protocol, so that the ability to oxidize lipids may have been decreased for this reason. The effects of exercise need to be interpreted on this background. Endurance exercise training increases adipose tissue lipolytic responsiveness, by enhancing expression and activation of HSL [27]. Thus, exercise ought to prevent the decline of fat oxidation induced by the restrictive diet [28, 29]. Since we apparently have now a rather limited information on the effects of various nutritional conditions on the balance of substrate oxidation at exercise, the explanation we propose for our findings remain speculative. We suggest that the beneficial effect of low intensity training on lipid oxidation at exercise helps to counteract the lipid sparing effect induced by the post-diet homeostatic adaptations that develop in traditional hypocaloric strategies and that play a role in resistance to slimming. This issue which remains largely unknown in adolescents requires, of course, additional investigation.

Contrasting with the effect of low intensity training, high intensity (HI) exercise training decreases fat oxidation and increases CHO oxidation at 20 and 30% Wmax th. Clearly, the effects of exercise training at HI in obese boys, is not to increase total fat oxidation during sub maximal exercise. On the opposite, fat oxidation at exercise decreases. This result is in agreement with Van Aggel-Leijssen [7] who shows no effect of HI exercise training on total fat oxidation, in obese men. By contrast, it should be pointed out that high intensity training increases the ability to oxidize CHO during exercise, as already observed in elite cyclists trained above the ventilatory threshold [30]. This metabolic adaptation, which may have some interest in elite sports, is clearly not the goal of training protocols aiming at treating obesity, a situation already characterized by some degree of "CHO dependence" [1], perhaps because

of the greater abundance of the glycolytic as opposed to lipolytic enzyme systems in skeletal muscle. Obviously, the delayed effects of exercise training at high intensity on lipid oxidation at rest, which were not investigated here, should be important to take also into account and may provide a metabolic basis to the use of HI training besides LI training in the management of obesity [9].

Actually, the results in the literature regarding the effect of regular resistance exercise sessions are less consistent than those related to low intensity training. Poehlman et al. [31] studied 16 young lean sedentary women before and after 6 month resistance training program. The data showed that RER was 0.85 before and after the training program. Similar findings were reported in a group of young men after 3 month of strength training [32]. Further research is necessary to specifically investigate the effects of resistance training program on fat metabolism.

Moreover, in the current study, the HI training was associated with a non significant tendency for fat free mass to decrease. This decrease, if confirmed on a larger sample, could be due to the classical effect of restrictive diet. Clearly, loss in fat-free mass are not rare during diet-induced weight reduction in obese subjects, particularly when diet is very low in energy [33]. While the LI diet designed to improve lipid oxidation seems to preserve correctly the fat free mass in this protocol, we cannot affirm that this mass would be equally preserved with the HI protocol. To conclude, additional studies are needed.

However, on the whole, in regard to these results, HI training does not seem to be adapted for the obese adolescents. In the literature, low-intensity exercise is typically recommended for individuals interested in weight loss [34] because free fatty acids are a preferential fuel source during low-intensity exercise [35], and subjects who are deconditionned and carry excessive weight may not be able to safely engage in high-intensity exercise [34]. The higher proportion of fat oxidation during exercise after LI training was justified when we considered the physiological adaptations during exercise. However, recent works by the team of Tremblay and coll. [36] have evidenced some specific aspects of high-intensity exercise that may be interesting in this context. The most obvious is that this kind of training creates a more negative energy balance during post exercise than low-intensity exercise.

Several factors can influence the effects of exercise programs. The first is patient's adherence to exercise. A large body of research has focused on the difficulties of exercise adherence in children [37, 38]. However it is impossible to properly evaluate the influence of exercise unless subjects engage in the majority of the exercise sessions for the planned duration and intensity. The second concern is diet. A decrease in caloric intake through manipulation of diet is at present the most powerful way to lose weight. Diet changes have clearly a bigger impact on negative energy balance than calories expended as a result of exercise, although the combination of both can be synergistic and increase negative energy balance, thus theoretically improving weight loss compared to diet alone.

Conclusion

This study shows that in obese children submitted to a hypocaloric diet a two months training protocol at a targeted exercise level maintains the ability to oxidize fat at exercise if it is performed at low intensity while it both decreases fat oxidation and increases CHO oxidation at exercise when performed at high intensity. Thus, a low intensity training protocol seems to counteract to some extent the decline in lipid oxidation at exercise that occurs after a hypocaloric diet, and is thus likely to be synergistic to diet in the weight lowering strategy. By contrast, the high intensity training protocol mostly increases the ability to oxidize CHO at exercise and seems therefore less interesting to associate with diet in a slimming strategy. On a practical point of view the individual level for targeting this level of training cannot be predicted by a theoretical percentage of the HR max and can only be determined by a specific exercise-test.

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