Research Article

Effects Over 24Hr of Exercise Targeted on Lipid versus Carbohydrate Oxidation on Eating Behaviour in Normal Weight Volunteers -

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ABSTRACT

Exercise reverses muscular metabolic defects involved in the impairment of lipids oxidation in obesity, and modifies eating habits, exerting both an orexigenic and a satietogenic effect.

Our working hypothesis is that the orexigenic effect, which is triggered by glycogen depletion and high intensity exercise, is minimized when exercise is performed at the level of the LIPOXmax, and thus uses lipids and spares glycogen. Accordingly, its weight-loving effect could be related to a mostly satietogenic with little orexigenic effect, while exercises at higher intensities would be more orexigenic because of an important use of carbohydrates.

We studied over 24 hours the effects of an exercise targeted by exercise calorimetry, in 10 normal weight volunteers. Subjects performed at random order three sessions: 45 minutes of steady state exercise targeted on the LIPOXmax, an isoenenergetic exercise targeted at the LIPOXzero (exercise intensity in which there is no longer lipid oxidation and only carbohydrate oxidation) and a morning without exercise.

The evolution of appetite and satiety is measured with an analogous visual scale (Hill and Blundell). Capillary blood glucose is also measured. 20 minutes after exercise and after the morning without activity a meal test is proposed, along with a scale of palatability.

Energy ingested over the 24 hours is calculated with a food notebook filled by the subjects.

In these subjects after the LIPOXmax session, the following breakfast contains fewer calories and fewer carbohydrates. After the LIPOXzero session, the breakfast remains unchanged. Blood glucose decreases during the LIPOXzero session and remains stable during the LIPOXmax session. When the blood glucose decreases there is a parallel increase in hunger.

These findings are in agreement with our working hypothesis. Orexigenic effects of exercise mostly occur at levels where carbohydrate oxidation predominates, resulting in a decrease in blood glucose. On the other hand, satietogenic effects prevail at exercise levels close to the LIPOXmax, where lipid oxidation results in a sparing of carbohydrates.

The paradox of the fair weight-reducing effect of moderate volumes of LIPOXmax exercise in spite of its low intensity, while exercise at higher intensities is deceiving for this purpose unless it is used at high volumes, is likely to be due (at least in part) to their different effects on eating behaviour.

Keywords: Obesity; Exercise; LIPOXmax; Metabolism; Lipids oxidation; Appetite; Satiety

INTRODUCTION

Exercise is an essential component of the management of obesity. Its weight-loving effect is classically assumed to be explained by the energy deficit it induces, but there are clearly other mechanisms that have been highlighted more recently. Exercise has been shown to reverse metabolic muscular defects that result in a decrease of lipid oxidation in sedentary obese patients [1]. In addition, it also modifies eating behaviour. This last effect is complex, since it includes both an orexigenic and a satietogenic component [2].

Therefore exercise can either increase or decrease food ingestion according to the balance between these two effects. The literature on this topic is quite complex [3].

Recently the Energy Balance Study demonstrated in a series of 421 subjects that low levels of physical activity are associated with dysregulation of energy intake and fat mass gain, with a threshold of activity at 7116 steps/day [4]. However, which exercise procedure can reverse this dysregulation and restore an energy balance sufficient for activity at 7116 steps/day [4]. However, which exercise procedure can reverse this dysregulation and restore an energy balance sufficient for correcting obesity is not clearly established in the current literature. According to a recent review by John Bludell, individual responses of appetite to exercise are highly variable and difficult to predict [5].

Our working hypothesis is that (a) orexigenic effects are mostly triggered by exercise bouts that involve glycojen depletion and/or hypoglycaemia, and thus predominate after high intensity exercise, and (b) on the opposite, orexigenic effects are minimal when exercise is performed at the level of the LIPOXmax which burns less carbohydrates and allows glycogen sparing. Therefore, we hypothesize that the weight-loving effect of endurance training targeted at low intensity (50% VO2max or below) in the zone of lipid oxidation, is at least in part explained by a favourable balance between satietogenic and orexigenic effects, favouring satiety, while exercise at higher intensity are more orexigenic because of a more important use of carbohydrates.

MATERIALS AND METHODS

Subjects

The study was performed in 10 normal weight volunteers, (age: 24.1 ± 0.46; Body mass index: 22.2 ± 1.36 kg/m²; theoretical VO2max: 44.2 ± 8.26 ml/min). Characteristics of these subjects are given on table 1.

Exercise calorimetry

Exercise calorimetry was performed as previously described [6] in order to analyse the balance of energetic substrates. Patients should remain fasting for 12 hours. The test is composed of 4 steps of 6 minutes steady state exercise set at respectively 30, 40, 50 and 60% of the maximal predicted power on cycloergometer connected with a gas exchange analyser allowing an analysis cycle by cycle with control of ECG and measurements of VO2, VCO2 and respiratory exchange

Table 1 : Characteristics of the 12 subjects of the study (3 females and 9 males).

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>Fat Free mass (kg)</th>
<th>Muscle mass (kg)</th>
<th>%fat</th>
<th>waist circumference (cm)</th>
<th>Hip circumference (cm)</th>
<th>BMI (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>24.92 ± 1.69</td>
<td>69.80 ± 2.43</td>
<td>175.54 ± 2.39</td>
<td>58.54 ± 2.32</td>
<td>31.61 ± 2.09</td>
<td>15.98 ± 2.53</td>
<td>79.13 ± 2.68</td>
<td>87.50 ± 2.15</td>
<td>22.65 ± 1.28</td>
</tr>
</tbody>
</table>
RESULTS

On the average the LIPOXmax of these subjects occurred at 42 ± 0.06 % of VO2 max and the LIPOXzero (level of exercise where the use of lipids is none) was at 76 ± 0.14 % of VO2max.

Eating behaviour

As shown on figures 1 & 2, it was observed that in these subjects both exercise bouts increased in a similar manner on the Hill’s scale the drive to eat ($p < 0.01$) and its intensity ($p < 0.01$) and have a similar orexigenic effect.

Dietary questionnaire

As shown on figure 3, after the exercise session performed at the LIPOXmax, the breakfast eaten in the following morning contained less calories ($p < 0.02$) and less carbohydrates ($p < 0.02$) compared to the breakfast of the preceding day. After the LIPOXzero exercise, the breakfast remained unchanged compared to the morning following the session without activity. There was a nonsignificant tendency to eat more over the 24 hr after LIPOXzero ($11059.7 ± 1506$ kJ/24hr) compared to LIPOXmax ($9678.6 ± 854$ kJ/24hr) and controls ($9718 ± 925$ kJ/24hr).

Blood glucose

During all exercise sessions, capillary glucose was monitored.

Figure 1: Design of the study.

Figure 2: Evolution of subjective feelings of appetite and satiety after a single exercise bout. $p < 0.05$. Both exercise bouts increase hunger on a rather similar manner, and the satietogenic effects of LIPOXmax exercise that are evidenced after a series of training sessions [10] are not observed here.
Blood glucose was shown to significantly decrease during the session performed at LIPOXzero (-0.06 ± 0.02 mmol/l, p < 0.02). In this case the decrease occurred 8 times on 10. By contrast there was no significant change in capillary glucose during exercise performed at LIPOXmax, since in 50% of the cases it increased and decreased in 50% of cases. As shown on figure 4, all exercise sessions that resulted in a decrease in blood glucose induced an increase in hunger on the VAS (p < 0.001). By contrast when exercise did not decrease blood glucose, it was followed by a significant decrease in calorie intake over the next 24 hours (p < 0.005) as shown on figure 5.

**DISCUSSION**

This randomized controlled study shows that spontaneous food intake is decreased over the period following a bout of exercise performed at the LIPOXmax. More precisely, the next breakfast contains less calories and less carbohydrates. By contrast, after an exercise bout performed at the LIPOXzero, the breakfast remains unchanged. Blood glucose decreases during the exercise bout performed at the LIPOXzero while it remains stable (on the average) during an exercise bout performed at the LIPOXmax. When the exercise bout induces a decrease in blood glucose, it increases hunger, while exercise that does not induce a fall in blood glucose results in a lower calorie intake over the following 24 hours.

These results lead to think that in normal weight subjects, 45 min of exercise performed at the intensity where lipid oxidation is optimal has a satietogenic effect. This satietogenic effect is associated on the average with a stability of blood glucose. By contrast when exercise is performed at the LIPOXzero and thus uses only carbohydrate as a fuel, it frequently determines a decrease in blood glucose and an increase in hunger.

In another work dealing with obese subjects followed during a few months, we observed that LIPOXmax training increases the feeling of satiety, decreases gradually the orexigenic effect of an isolated session and decreases the caloric intake which is correlated to a decrease of food thoughts and leads to weight loss in 72% of subjects [10]. Therefore, this controlled randomized study extends to normal weight subjects what we previously reported in a follow-up open study of obese patients.

These findings are likely to have important implications for exercise prescription in the context of weight management. Currently, the importance of physical activity in the management of obesity is no longer a matter of controversy. It is now well established that exercise is efficient for preventing obesity, for stabilizing weight after weight loss, and also to decrease body fat stores. However, for the later purpose, most studies show a rather modest effect, and there is no consensus about the intensity level that should be targeted [11].

It has become clear since decades that high volumes of physical activity, above 15 hours per week, are very efficient for losing weight, and even more if exercise is practiced at high intensity: this has been remarkably demonstrated by the RESOLVE study [12]. The important calorie deficit induced by these high volumes of exercise is likely to explain most of the efficacy of this procedure. However, large series of patients treated by this approach are not available, since it is rather difficult for most obese sedentary people to find enough time and motivation to add such volumes of physical activity to their regular life.

Interestingly, even low volumes of exercise (3-4 hr/week) are able to determine weight loss, as clearly evidenced by recent studies [11,13]. A common belief is that, when exercising at low volumes, high intensity could be more efficient because it wastes more energy. However, high intensity exercise mostly involves carbohydrate oxidation and thus decreases glycogen stores and
blood glucose concentrations, resulting in increased hunger, and, in turn, in many cases, paradoxical weight gain [14]. The results of the current experiment provide a very likely explanation for this apparently paradoxical observation. In individuals whose blood glucose decreases during such short and intense exercise bouts, there may be a predominance of the orexigenic effect of exercise and thus overeating and weight gain.

The Study of Targeted Risk Reduction Interventions through Defined Exercise (STRIDE) confirms that, beside high training volumes that are obviously a powerful weight-lowering procedure, low volumes of activity (<4h/week) are also efficient on weight either at low or high intensity, and are even more efficient for improving body composition and carbohydrate metabolism at low intensity [13]. Our current observations are in agreement with that study and provide an additional explanation for this efficiency: clearly, the effects of exercise on appetite and satiety are not similar at high and low intensity, and low intensity exercise is an interesting tool to regulate appetite.

The methods used in this study require some comments. Since its first introduction at the end of the nineties, exercise calorimetry has been the matter of some controversies. In a recent review [15] we made an attempt to synthesize all this literature. It can be reasonably considered that the averaged VO2 and VCO2 values of a 6 min steady state exercise bout reflects the level of substrate oxidation that could be measured over a period of 45 min. By contrast, the often employed measurement on 3 min steps results in oversatimation of lipid oxidation rate. The prediction of the power at which occurs maximal lipid oxidation by the derivative of the curve of lipid oxidation plotted against power intensity (LIPOXmax) is fairly reproducible when measured in standardized conditions, but is quite variable among individuals and is modified by a host of situations (previous exercise, feeding, etc…). Actually this level represents the intensity of exercise that would be spontaneously selected for exercising for a long time without being stopped by signs of exhaustion such as breathlessness or excess sweating... Recently some reports have shown that low volumes (3x45 min/week) of exercise targeted at this level induce or excess sweating... Recently some reports have shown that low volumes (3x45 min/week) of exercise targeted at this level induce... and low intensity exercise is an interesting tool to regulate appetite.

On the whole, this study supports the hypothesis that the weight-reducing effect of low intensity exercise is at least in part explained by its effects on eating behavior. They are in agreement with an observational follow-up study on obese subjects [10]. These data allow to understand better the efficiency of this soft exercise training protocol but are still preliminary and must be confirmed. On a practical point of view, they lead to prompt clinicians involved in exercise prescription for obesity to carefully take into account the effects of exercise on appetite and satiety, which are likely to markedly influence the efficiency of training.

ACKNOWLEDGEMENT

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REFERENCES