Effets d’un programme de deux mois d’entraînement individualisé associé ou non à un régime hypocalorique sur les niveaux plasmatiques des adipocytokines chez des adolescentes obèses

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Résumé

Objectifs. – Étudier les effets d’un programme d’entraînement individualisé associé à un régime hypocalorique sur les concentrations plasmatiques des adipocytokines chez des adolescentes obèses. Matériels et méthodes. – Vingt-sept adolescentes obèses ont été réparties (ordre randomisé) en trois groupes avec trois interventions différentes sur deux mois : (1) régime hypocalorique (n = 9), (2) entraînement individualisé au niveau du Lipoxmax (puissance pour laquelle le taux d’oxydation des lipides est maximal) (n = 9) et (3) programme combiné régime/entraînement (n = 9). La masse corporelle (MC), l’indice de masse corporelle (IMC), le pourcentage de masse grasse (%MG), l’estimation de la résistance à l’insuline par le modèle d’évaluation homéostasique (HOMA-IR) et les niveaux plasmatiques à jeun des adipocytokines ont été évalués avant et après les deux mois d’intervention. Résultats. – Le programme régime/entraînement a induit un décalage du niveau de Lipoxmax vers une intensité supérieure (+27,8 ± 5,1W ; p < 0,01) et une augmentation de l’oxydation des lipides à Lipoxmax (+96,8 ± 16,2 mg/min ; p < 0,01). L’augmentation de l’oxydation des lipides a été corrélée significativement (p < 0,01) avec les améliorations induites par le programme régime/entraînement sur le %MG (r = −0,47), le HOMA-IR (r = −0,66), la leptine (r = −0,41), le TNF-α (r = −0,48), l’Il-6 (r = −0,38), l’adiponectine (r = 0,43) et la résistine (r = 0,51). Conclusion. – L’entraînement au niveau du Lipoxmax combiné avec un programme de régime hypocalorique a amélioré la capacité à oxyder les lipides pendant l’exercice, et cette amélioration a été associée aux améliorations des niveaux plasmatiques des adipocytokines chez des adolescentes obèses.

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Mots clés : Adolescentes obèses ; Adipocytokines ; Activité physique ; Restriction calorique

Abstract

Objectives. – To examine if, in young obese patients, an individualized training programme in association with a caloric restriction programme which had an effect on whole-body lipid oxidation, was able to induce changes on plasma adipocytokine concentrations. Materials and methods. – Twenty-seven obese female adolescents participated in the study. Whole-body lipid oxidation during exercise was assessed by indirect calorimetry during a graded cycle ergometer test. Body mass (BM), body mass index (BMI), percentage of body fat (%BF), insulin homeostasis model assessment (HOMA-IR) and fasting levels of circulating adipokines were assessed prior and after a two-month diet programme, individualized training programme targeted at Lipoxmax corresponded to the power at which the highest rate of lipids was oxidized and combined diet/training programme.

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1. Introduction

The prevalence of obesity continues to rise, and a sedentary lifestyle is recognized as a key-risk factor. Adipose tissue is not merely a fat storage deposit, but has been recognized as an endocrine organ able to produce biologically active proteins termed ‘adipocytokines’. These adipocytokines include leptin, adiponectin, resistin, tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) which may be related to the increase of obesity-mediated adverse effects on glucose and lipid metabolisms [1,2].

Adiponectin levels decrease with obesity [3] and low adiponectin concentration is associated with insulin resistance [4]. TNF-α is elevated in obesity and contributes to insulin resistance, possibly through down-regulation of GLUT-4 and inhibition of insulin receptor function and signaling [5]. IL-6 secreted by fat cells induces hepatic synthesis of C-reactive protein (CRP), and both molecules are associated with obesity and cardiovascular disease [6]. Leptin plasma concentration is directly related to the severity of obesity, as an increase of fat mass is associated with an increase of leptin [7]. Kondo et al. [8] showed that changes in circulating adipocytokine levels, due to increased exercise, were involved in the improvement of a negative energy balance. Moreover, caloric restriction increases adiponectin levels and decreases insulin resistance in obese children [9]. Many of the controlled intervention studies have shown that exercise improves adipocytokine levels, with a concomitant improvement in BM and/or body composition [10].

Regular endurance training has been shown to favourably modify the balance of substrate oxidation, especially in patients with metabolic defects such as obesity [11] and type 2 diabetes [12]. Indeed, exercise training helps to restore fat oxidation capacity [13], which can be linked to improvement in insulin sensitivity [12]. Given its advantages, indirect calorimetry remains the most commonly used technique for assessing the balance between fat and carbohydrate (CHO) oxidation during exercise.

During progressive exercise, it provides a simple index: the maximum fat oxidation rate point (Lipoxmax) [14]. This index can be obtained from gas exchange during submaximum graded exercise up to 60% of the theoretical maximum aerobic power [13], and are useful for prescribing an individualized exercise programme [15] in those with metabolic diseases such as obesity and type 2 diabetes.

Thus, the aim of our study was to investigate in obese female adolescents, the effects of a two-month diet programme, individualized training programme, carried out at the level of the Lipoxmax (power intensity at which lipid oxidation is maximum), and combined programme (diet/training) on whole-body lipid oxidation and plasma adipocytokine concentrations. We hypothesized that an increase in lipid oxidation and an improvement in plasma adipocytokine concentrations in obese girls will be better after a mixed two-month diet/training programme than after separate diet or training programmes.

2. Patients

The Research Ethics Committee of the Faculty of Medicine, Sousse, Tunisia approved this study. Each participant and parents signed an informed consent form before actively engaging in the study. Twenty-seven 13-year-old obese female adolescents participated in the study. Obesity was defined as body mass index (BMI) greater than the 97th percentile according to the French references [16]. None of the children was involved for more than three hours per week in structured programme of physical activity or sports training. No patients had diabetes-related complications, and no medications were administered. Non-compliant participants were not included. Individuals were excluded if they had ischemic heart disease or other medical conditions for which the prescribed exercise might be contraindicated. Each individual came to the laboratory for a medical examination and anthropometric measurements performed by a paediatrician.

The adolescents were randomly assigned to one of the three programme groups: diet group, individualized training at Lipoxmax group and training combined with diet group.

3. Methods

3.1. Anthropometric measures

Height was measured with a standing stadiometer and recorded with a precision of 1 mm. Body mass (BM) was measured to the nearest 0.1 kg with a digital scale (OHAUS, Florham Park, NJ). Body mass index (BMI) was calculated as BM in kilograms divided by height in squared meters (kg/m²). Two skinfold thicknesses (triceps and subscapular) were measured by the same trained technician (Harpenden caliper).
The percentage of body fat (%BF) was calculated using the equations of Slaughter et al. for girls [17]: with triceps and subscapular skinfolds less than 35 mm and more than 35 mm respectively:

\[
\% BF = 1.33 \times (\text{sum of 2 skin folds}) - 0.013 \times (\text{sum of 2 skin folds})^2 - 2.5 \\
\% BF = 0.546 \times (\text{sum of 2 skin folds}) + 9.7
\]

The test-retest data were then used to calculate the precision of all body composition measurements. Pubertal stage was evaluated according to the Tanner classification [18] by a trained paediatrician: pre pubertal children comprised children who were in stage I, pubertal children in stage II-III, post pubertal children in stage IV-V.

### 3.2. Exercise testing

For the second visit children came to the laboratory between 08:00 and 09:00 a.m. after an overnight fast to perform a cycling exercise. Children performed a five six-minutes stage exercise at 08:00 and 09:00 a.m. after an overnight fast to perform a cycling exercise testing for each adolescent using the predictive equations of Wasserman for obese girls [20]:

\[
\text{V}_{O_2 \text{max}} = (52.8 \times M) - 303.4 \\
W_{\text{max}} = (\text{V}_{O_2 \text{max}} - 10 \times (\text{M})) \times (10.3)^{-1} (M : \text{body mass in kg}).
\]

Tests were all performed on an electromagnetically braked cycle ergometer (Ergoline, Bitz, Germany). \( V_O_2 \) and \( V_CO_2 \) were measured breath-by-breath through a mask connected to an \( O_2 \) and \( CO_2 \) analyzers (ZAN 600, Medgeräte, Germany). Ventilatory parameters were averaged every 30 s during submaximal exercise testing. ECG was monitored for the duration of tests. As previously described [19], we calculated a parameter representative of the whole-body lipid oxidation during exercise, which is the maximum lipid-oxidation point (\( \text{Lipox}_{\text{max}} \)), expressed in watts (W) which corresponds to the exercise intensity at which the highest rate of lipid oxidation is achieved (lipid oxidation at \( \text{Lipox}_{\text{max}} \), expressed in mg/min), according the following equation:

\[
\text{Lipid oxidation (mg/ min)} = 1.6946 \times V_O_2 - 1.7012 \times V_CO_2 \text{(with } V_O_2 \text{ and } V_CO_2 \text{ expressed in ml/ min}).
\]

### 3.3. Intervention programmes

Based on the identified risk factors for the participants, the intervention focused on changes in diet and physical activity to reduce the cardiovascular disease risk by encouraging the participants (diet and diet/training groups) to decrease saturated fat and cholesterol intake, increase consumption of a variety of fruits and vegetables, increase the consumption of dietary fiber and limit salt. The diet programme consisted of a 500 kcal daily caloric reduction below the actual energy requirement. The nutritional education programme was conducted four days per week at two local schools in the community where the participants lived. Lectures using Powerpoint® presentations, videos, games, and role-play scripts were designed for trainers to use during the education programme.

Subjects of training and diet/training groups received a two-page summary of information about exercise prepared by an exercise physiologist. It included a list of the general health benefits of regular exercise as well as various recommendations and precautions about exercise. An exercise prescription specifying the duration, frequency, and intensity of exercise based on the participant’s heart rate corresponding to \( \text{Lipox}_{\text{max}} \) assessed at the first visit, was also provided. The training programme consisted of 90 minutes of supervised activity per day at a heart rate that corresponded to \( \text{Lipox}_{\text{max}} \), less than four days per week during eight weeks.

### 3.4. Biochemical analysis

Blood samples were obtained between 7:00 am and 8:00 am after an overnight fast. Samples were collected in EDTA containing tubes and immediately centrifuged at 4 °C. Plasma samples were kept on dry ice during transportation from the testing sites and were stored at −80 °C until analyzed. Plasma glucose concentrations were measured using an automated device (AU2700, Olympus, France). The interassay coefficient of variability (CV) was 1.7%. Plasma insulin was assayed by an IRMA Insulin kit (Immunotech, France). The intra- and interassay CVs were, respectively, 3.3–4% and 3.7–4.8%. Plasma adiponectin and leptin were determined using an ELISA kit (B-Bridge international, Inc). The intra- and interassays CVs were, respectively, 4.1 and 4.7% for adiponectin and 3 and 3.2% for leptin.

Resistin was measured by an enzyme-linked immunoassay kit obtained from Biovendor Laboratory Medicine Inc (Brno, Czech Republic). The intra- and interassay CVs were, respectively, 4.5 and 7.8%. Plasma levels of IL-6 and TNF-α were measured with saline using quantikine ELISA-kits from R&D systems (cat. nos. HS600 and HSTA00C, respectively). The intra-assay CV for TNF-α and IL-6 assays was less than 10%. The interassay CV was 12.2% for TNF-α and 18.2% for IL-6.

Insulin resistance was assessed using the homeostatic model assessment for insulin resistance (HOMA-IR). The HOMA-IR has been validated in children and adolescents [21] and was computed as follows: HOMA-IR = [insulinemia (\( \muU/ml \times \text{glycemia (mmol/l}) \)]/22.5.

### 3.5. Statistical analyses

All values are expressed as mean ± S.D. Paired Student’s \( t \)-test was used for comparison within the three groups and unpaired Student’s \( t \)-test was used for group’s comparisons. Repeated-measure ANOVAs were used to compare the responses of different groups, at different times of the test, pre- and postprogram. Interclass correlation coefficients (ICC) were
calculated to evaluate the reliability of all body composition measurements [22]. Correlation between Lipoxmax and other parameters was determined by Pearson’s correlation. All statistical analyses were performed using SPSS version 8.0. P < 0.05 was considered statistically significant.

4. Results

Anthropometric, physical fitness, and related biochemical characteristics before and after the intervention programmes are shown in Table 1.

For all body composition measurements, the reliability was excellent; ICC >95%. Comparisons of the three groups before the intervention showed that they were matched for anthropometric parameters, age, and pubertal stage.

Before the programme, plasma adiponectin and leptin levels were similar in all subjects. However, the plasma resistin, IL-6 and TNF-α level were significantly different between the three groups in the preprogramme. Indeed, IL-6 and TNF-α level were higher in the diet/training subjects than in the diet or training subjects.

After the eight-week intervention programme, BMI decreased by 6.1, 1.5, 11.5%, and %BF by 7.3, 3.6, 15.1% in diet, training and diet/training groups respectively (Table 1).

At the end of the programme, diet/training group had a significant decrease in leptin, TNF-α and IL-6, and a significant increase in adiponectin and resistin at the end of the programme.

These changes were more pronounced in the diet/training group compared to the other groups (Fig. 1).

Exercise training had, however, marked effects on whole-body lipid oxidation. After subsequent training, the Lipoxmax was raised to a higher power (+19.3 ± 3.4 W; p < 0.05 and +27.8 ± 5.1 W; p < 0.01) in the training and diet/training groups, respectively (Fig. 2A).

Fig. 2B shows that the post-training lipid oxidation at Lipoxmax was significantly increased compared with pretraining values (+61.7 ± 9.6 mg/min; p < 0.05 and +96.8 ± 16.2 mg/min; p < 0.01) in the training and diet/training groups, respectively.

There was no significant change for this parameter in the diet group (Lipoxmax: +6.1 ± 2.4 W; p = 0.7 and lipid oxidation at Lipoxmax: +23.2 ± 6.6 mg/min; p = 0.4).

The postprogram usual index of insulin resistance (HOMA-IR) was significantly improved in training (p < 0.05) and diet/training (p < 0.01) groups. Diet group did not present a significant reduction in HOMA-IR (Fig. 3).

In the diet/training group, Lipoxmax exhibited a significant (p < 0.01) negative correlation with BMI (r = −0.31), BF% (r = −0.47), leptin (r = −0.49), TNF-α (r = −0.48), IL-6 (r = −0.38), HOMA-IR (r = −0.66) and a significant (p < 0.01) positive correlation with adiponectin (r = 0.43), resistin (r = 0.51).

In the training group, Lipoxmax exhibited a significant (p < 0.05) negative correlation with leptin (r = 0.36), TNF-α (r = 0.41), IL-6 (r = 0.30), HOMA-IR (r = 0.44) and a signifi-

**Table 1**

Subject characteristics pre- and postintervention programme in the diet group (n = 9), the training group (n = 9) and the diet/training group (n = 9).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Diet Pre</th>
<th>Diet Post</th>
<th>Training Pre</th>
<th>Training Post</th>
<th>Diet/training Pre</th>
<th>Diet/training Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>13.2 ± 0.3</td>
<td>-</td>
<td>13.1 ± 0.9</td>
<td>-</td>
<td>13.1 ± 0.8</td>
<td>-</td>
</tr>
<tr>
<td>BMI (kg)</td>
<td>80.2 ± 10.1</td>
<td>75.3 ± 9.8*</td>
<td>80.5 ± 11.4</td>
<td>79.3 ± 10.8</td>
<td>82.3 ± 8.7</td>
<td>72.8 ± 9.4**</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>30.5 ± 2.2</td>
<td>28.5 ± 1.3*</td>
<td>30.4 ± 1.8</td>
<td>29.8 ± 1.7</td>
<td>31.2 ± 2.1</td>
<td>27.6 ± 1.2**</td>
</tr>
<tr>
<td>BF%</td>
<td>38.2 ± 4.9</td>
<td>35.4 ± 5.3*</td>
<td>39.3 ± 4.2</td>
<td>37.9 ± 6.1</td>
<td>39.1 ± 5.0</td>
<td>33.2 ± 4.3**</td>
</tr>
<tr>
<td>Energy intake (kcal/day)</td>
<td>3144</td>
<td>2629 ± 92**</td>
<td>3208 ± 228</td>
<td>3226 ± 119</td>
<td>3242 ± 123</td>
<td>2734 ± 97**</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>4.42 ± 0.11</td>
<td>4.37 ± 0.14</td>
<td>4.51 ± 0.14</td>
<td>4.44 ± 0.16</td>
<td>4.62 ± 0.12</td>
<td>4.26 ± 0.15**</td>
</tr>
<tr>
<td>Insulin (µU/ml)</td>
<td>21.3 ± 4.2</td>
<td>20.6 ± 4.6</td>
<td>21.6 ± 5.3</td>
<td>17.8 ± 3.1*</td>
<td>22.7 ± 4.2</td>
<td>14.3 ± 3.4**</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>4.18 ± 1.3</td>
<td>4.00 ± 1.6</td>
<td>4.33 ± 1.7</td>
<td>3.51 ± 1.4</td>
<td>4.66 ± 1.5</td>
<td>2.71 ± 0.9**</td>
</tr>
<tr>
<td>Resistin (ng/ml)</td>
<td>7.4 ± 1.3</td>
<td>8.9 ± 1.4*</td>
<td>6.2 ± 2.4</td>
<td>7.8 ± 1.1*</td>
<td>7.6 ± 2.4</td>
<td>9.9 ± 1.2*</td>
</tr>
<tr>
<td>Adiponectin (µg/ml)</td>
<td>4.2 ± 0.4</td>
<td>4.7 ± 0.6</td>
<td>4.4 ± 0.5</td>
<td>5.2 ± 0.6*</td>
<td>4.1 ± 0.3</td>
<td>5.8 ± 0.5**</td>
</tr>
<tr>
<td>Leptin (ng/ml)</td>
<td>19.8 ± 5.2</td>
<td>16.2 ± 5.3*</td>
<td>20.1 ± 6.1</td>
<td>16.2 ± 5.3*</td>
<td>19.5 ± 6.4</td>
<td>13.9 ± 3.3**</td>
</tr>
<tr>
<td>TNF-α (pg/ml)</td>
<td>7.4 ± 2.4</td>
<td>5.6 ± 2.9*</td>
<td>7.1 ± 3.7</td>
<td>5.3 ± 3.1*</td>
<td>8.6 ± 2.1</td>
<td>5.2 ± 1.4**</td>
</tr>
<tr>
<td>IL-6 (pg/ml)</td>
<td>4.7 ± 1.1</td>
<td>4.4 ± 1.3</td>
<td>5.5 ± 1.6</td>
<td>4.7 ± 1.2*</td>
<td>5.8 ± 2.1</td>
<td>4.3 ± 1.2**</td>
</tr>
</tbody>
</table>

Numbers are mean ± S.D. *: p < 0.05 and **: p < 0.01 post-versus preprogram in the three groups.

BF%: percentage of body fat; BM: body mass; BMI: body mass index; HOMA-IR: homeostasis model assessment index for insulin resistance; HR: heart rate; Lipoxmax: the power at which the highest rate of lipids is oxidized; IL-6: interleukin-6; PS: puberty stage; TNF-α: tumor necrosis factor-alpha.
Changement du point d’oxydation maximale des lipides (Lipoxmax) entre avant HOMA-IR après le programme. * p < 0,05 et ** p < 0,01.

Changement en pourcentage de l’index usuel de la résistance à l’insuline ; programme. * p < 0,05 et ** p < 0,01. A : Lipoxmax exprimé en watts. B : Taux de l’oxydation des lipides au niveau du Lipoxmax, exprimé en mg/min.

Correlation between Lipoxmax and other parameters over the 2-month programme.

Coefficients de corrélation entre Lipoxmax et les autres paramètres après le programme de deux mois.

<table>
<thead>
<tr>
<th>Lipoxmax</th>
<th>Diet</th>
<th>Training</th>
<th>Diet/training</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI (kg/m²)</td>
<td>0.17</td>
<td>−0.12</td>
<td>−0.31**</td>
</tr>
<tr>
<td>BF%</td>
<td>−0.15</td>
<td>0.16</td>
<td>−0.47**</td>
</tr>
<tr>
<td>Leptin (ng/mL)</td>
<td>0.14</td>
<td>−0.36</td>
<td>−0.49**</td>
</tr>
<tr>
<td>TNF-α (pg/mL)</td>
<td>0.14</td>
<td>−0.41</td>
<td>−0.48**</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>0.11</td>
<td>−0.44</td>
<td>−0.66**</td>
</tr>
<tr>
<td>Adiponectin (ng/mL)</td>
<td>0.19</td>
<td>0.31*</td>
<td>0.43**</td>
</tr>
<tr>
<td>Resistin (ng/mL)</td>
<td>0.13</td>
<td>0.39*</td>
<td>0.51**</td>
</tr>
<tr>
<td>IL-6 (pg/mL)</td>
<td>0.16</td>
<td>−0.30*</td>
<td>−0.38**</td>
</tr>
</tbody>
</table>

BF% : percentage of body fat; BMI : body mass index; GH : growth hormone; HOMA-IR : homeostasis model assessment index for insulin resistance; IL-6 : interleukine-6; Lipoxmax : maximum rate of lipid oxidation; TNF-α : tumor necrosis factor-alpha.

* p < 0,05.
** p < 0,01.

5. Discussion

The present study showed that, in obese female adolescents, moderate training over two-month targeted at the level of Lipoxmax increases their ability to oxidize lipids during exercise, and that this increase is correlated with an improvement in both insulin sensitivity and plasma adipocytokine concentrations. Moreover, the group who performed the diet/training programme for two-month exhibited a significant correlation between lipid oxidation and body composition changes.

The first and most obvious effect of these interventions on food intake and exercise is the body weight loss and the improvement of the body image. This was evident in the diet group with a significant weight loss, but not in the training group for whom the weight-loss was rather slight. The combination of the two treatments, diet-training group, resulted in the best reduction of body weight, in account probably with a greater negative energetic balance. When examining more deeply the body composition, it appeared that the reduction in body fat mass was quite two-fold higher in the diet/training group than in the diet group, which is the expected effect of a slimming intervention.

Less visible are the changes in the glucose metabolism; in these adolescents, glucose levels were still in the normal range and did not change after the intervention. However, there was a tendency to insulin resistance (high insulin and HOMA-IR) which was reduced after the two-month of training with or without diet. Oppositely to the weight loss, the major effect was attributable to the exercise program, and one more time, the combination of the diet plus training was the more effective.

Skeletal muscle is largely involved in the development of obesity [15]. Moreover, muscular abnormalities alter the balance of substrate utilization, thus facilitating fat accumulation in adipose tissue. In contrast, regular exercise training, generally recommended for obese people, induces muscular metabolic changes, which can reverse these defects [19].

Obesity is characterised by an impaired ability for fat mobilisation and utilisation, so training at Lipoxmax is able to counteract this metabolic dysfunction and prevent the decline in fat oxidation induced by BM loss in the postdiet period. This effect may be mediated by maintenance of sympathetic nervous system sensitivity, which tends to be reduced after BM loss alone [23]. Recently, exercise calorimetry has been developed by several teams in order to target more closely training protocols for both adults [15] and adolescents [24] suffering from obesity. Consequently, it becomes important to know how diet combined with exercise modifies the balance of substrates as assessed with this technique in obese adolescent boys [19].

Leptin is thought to provide information about nutritional status and fat mass to neural centres regulating feeding behaviour,
power, an indicator of cardiovascular fitness, might attenuate, if
in Lipoxmax. The administration of leptin reduces hepatic fat
mass and improves insulin sensitivity in humans suffering from
this condition [26]. Suppression of the fatty-acid-synthesizing
enzyme stearil-CoA desaturase-I can correct the hypometabolic
phenotype of leptin deficiency, implying that leptin not only
works via central anorectic effects but also by increasing hepatic
fatty acid oxidation [27].

Our results showed that the decrease in plasma leptin lev-
els was associated with the decrease in HOMA-IR values in
the training and diet/training groups after the two-month pro-
gramme, suggesting the improvement of insulin sensitivity in
the subjects of these groups. A negative correlation between obe-
sity and circulating adiponectin has been well established, and
adiponectin concentrations increase concomitantly with weight
loss [28].

In the present study, adiponectin was significantly higher
for training and diet/training groups after the programme; this
increase was associated with the decrease in BMI and the sur-
rogate of insulin resistance (HOMA-IR) in the diet/training
group.

The mechanism underlying the role of adiponectin in lipid
oxidation may involve the regulation of production or activity
of proteins associated with triglyceride metabolism, including
CD36, acyl CoA oxidase, 5'-activated protein kinase, and
peroxisome proliferator-activated receptor γ (PPARγ) [29].
In our study we observed a significant positive correlation
between Lipoxmax and adiponectin in subjects of training and
diet/training groups.

Adiponectin decreases lipid synthesis and glucose produc-
tion in the liver and causes decreases in glucose and free fatty
acid concentrations in the blood. In addition, triglyceride pro-
duction is decreased and fat oxidation and energy dissipation
in the muscle are increased. Our results showed that in the
diet/training group the improvement in whole-body lipid oxida-
tion, and particularly the lipid oxidation rate at Lipoxmax during
exercise was significantly positively correlated with the increase
in plasma adiponectin concentrations. Adiponectin intracellular
signalling is connected with activation of adenosine monophos-
phate kinase [30], which has been previously shown to attenuate
β-adrenergic stimulation of lipolysis in fat tissue and muscle
[31].

It has been suggested that the strength of the association
between adipocytokines and insulin resistance is higher in
overweight- compared with normal-weight children. Similarly,
decreased physical activity levels have been associated with
increased insulin resistance [32].

Levels of these adipocytokines appear to respond favourably
to sustained physical activity [33] in children. It has been specu-
lated that increased but not decreased physical activity or aerobic
power, an indicator of cardiovascular fitness, might attenuate, if
existent, the relationship between insulin resistance and resistin,
TNF-α, and IL-6.

Aerobic training is considered to be a key-factor in the
treatment of obesity, and numerous studies have shown an
improvement in the metabolic as well the cardiovascular status
of obese individuals after a regular aerobic training programme.
Contradictory findings exist in the literature describing reduc-
tion [34] as well as no change [35] of plasma TNF-α induced
by diet or physical activity.

TNF-α produced by white adipose tissue is markedly up-
regulated in obesity and contributes to insulin resistance by
interfering with insulin receptor signaling [36]. This adipocy-
tokine inhibits adiponectin production in adipose tissue. Rubin
et al. [37] suggested that increased levels of adiposity, resulting
in decreased adiponectin and in increased TNF-α concentra-
tion, may link being overweight with insulin resistance during
adolescence. Our results show that the diet/training programme
decreases significantly the plasma levels of TNF-α in obese
girls and this decrease is associated with the improvement in
the insulin sensitivity.

In the present study we showed that the diet program did
not improve IL-6 concentrations. However, the improved body
composition induced by diet combined with training is associ-
ated with decreased serum concentrations of the IL-6 in obese
girls.

This finding was also reported in obese adolescents after
a three-week weight reduction programme including physical
activities [38]. Although being significantly higher in diet/training than in diet and training before the intervention,
TNF-α and IL-6 were decreased to the same level after the
intervention, showing the preponderant effect of training in this
improvement.

Resistin is a recently discovered adipocyte-released hormone
that has been positively correlated with several features of body
composition and insulin resistance [39]. Nevertheless, the phys-
iological role of resistin on obesity and insulin resistance is
unclear, with some studies reporting a significant association
between resistin levels, obesity, and insulin resistance [40],
whereas other studies were not able to show any significant
associations [41].

In addition, plasma levels of this protein have been scarcely
reported, and there is little information about its possible varia-
tions after weight loss. In the present study, changes of resistin
level observed in the training and diet/training groups were
significantly correlated with Lipoxmax and Lipoxmax was signifi-
cantly correlated with changes in insulin sensitivity presented in
this study by HOMA-IR. Therefore, our data support the hypo-
thesis that resistin antagonizes insulin action, suggesting that the
decrease of this hormone is associated with the improvement of
insulin sensitivity.

6. Conclusion

Our results show that in female obese adolescents an
increase in fat oxidation during exercise resulted obviously in
an improvement in body composition, but essentially in circu-
lating adipocytokine levels and the insulin resistance state by the
combination of diet and individualized exercise training targeted at Lipoxmax.

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References


