CLINICAL STUDY

Absence of exercise-induced variations in adiponectin levels despite decreased abdominal adiposity and improved insulin sensitivity in type 2 diabetic men

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Abstract

Objective: We investigated the effect of an intensive training program on fasting leptin and adiponectin levels.

Methods: Sixteen middle-aged men with type 2 diabetes were randomly assigned to either a training or control group. The training program consisted of 8 weeks of supervised endurance exercise (75% VO2peak, 45 min) twice a week, with intermittent exercise (five 2 min exercises at 85% VO2peak separated by 3 min exercises at 50% VO2peak) once a week, on an ergocycle.

Results: Training decreased abdominal fat by 44%, increased mid-thigh muscle cross-sectional area by 24%, and improved insulin sensitivity by 58% without significant change in body weight. Compared with controls, no significant variation in leptin or adiponectin levels was observed. However, in the trained group, change in adiponectin correlated with change in body weight (Spearman rank correlation, rs: 0.76, P = 0.03) but not with insulin sensitivity or abdominal adiposity variations.

Conclusions: An 8 week intensive training program inducing a marked reduction in abdominal fat and increase in insulin sensitivity does not affect adiponectin and leptin levels in men with type 2 diabetes.

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Introduction

Circulating leptin levels are correlated with body weight and more specifically with total body fat in humans and decline in subjects following weight loss (1). By contrast, adiponectin concentrations are lower in obese subjects (2), type 2 diabetic patients (3) and patients with coronary artery disease (3) compared with healthy controls, and increase after weight loss (4). Both adipocytokines are related to insulin action (5, 6). Exercise training improves insulin sensitivity (7) and body fat (8), but to date, the effect of exercise training on adiponectin levels in diabetic patients has not been investigated. We have previously reported that an intensive supervised training program improves insulin sensitivity and decreases visceral and subcutaneous adipose tissue in middle-aged type 2 diabetic males (9). In the present report, we analyzed the effects of this program on circulating adiponectin levels in relation to abdominal fat and insulin sensitivity in type 2 diabetic men.

Patients and methods

Sixteen type 2 diabetic men (known duration < 10 years) aged 45.4 ± 7.2 (S.D.) with glycosylated hemoglobin of 8.1 ± 1.7% and stable body mass index (BMI, 29.6 ± 4.6 kg/m²), reporting no participation in regular exercise for at least 6 months prior to inclusion, were recruited and randomly assigned to an eight-subject training group and an eight-subject control group. A randomized list was generated using the SAS program (SAS Institute, Cary, NC, USA). The trained group was assigned to an 8 week training program (three times/week) consisting of two different kinds of exercise: first, a continuous exercise for 45 min at 75% of their VO2peak twice a week; secondly, an intermittent exercise, once a week, consisting of five exercises at 85% VO2peak for 2 min separated by 3 min exercise at 50% VO2peak. The control subjects were seen weekly to exercise on the bicycle ergometer at a constant rate of 60 r.p.m. for 20 min at low intensity (30 W). All subjects had been followed in our department for at least 1 year.
Table 1 Anthropometric and biological parameters of the trained (n = 8) and control (n = 8) groups before and after the study period.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Trained group Before</th>
<th>Trained group After</th>
<th>Control group Before</th>
<th>Control group After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>42.90±5.20</td>
<td>—</td>
<td>47.90±8.35</td>
<td>—</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>86.90±13.40</td>
<td>85.00±13.75</td>
<td>90.40±11.50</td>
<td>88.75±11.30</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28.30±3.90</td>
<td>27.60±4.30</td>
<td>30.85±5.20</td>
<td>30.35±5.20</td>
</tr>
<tr>
<td>Visceral adipose tissue (cm²)</td>
<td>253.5±28.55</td>
<td>—</td>
<td>156.85±23.40</td>
<td>150.35±23.25</td>
</tr>
<tr>
<td>Subcutaneous adipose tissue (cm²)</td>
<td>241.5±49.55</td>
<td>198.00±39.00*</td>
<td>262.50±69.10</td>
<td>260.00±70.40</td>
</tr>
<tr>
<td>Mid-thigh muscle cross-sectional area (cm²)</td>
<td>148.30±36.10</td>
<td>184.35±35.85*</td>
<td>157.40±43.20</td>
<td>151.40±44.70</td>
</tr>
<tr>
<td>Glycemia (mmol/l)</td>
<td>9.35±1.20</td>
<td>9.70±1.65</td>
<td>8.50±2.15</td>
<td>8.55±1.95</td>
</tr>
<tr>
<td>Insulinemia (mU/l)</td>
<td>21.30±7.25</td>
<td>22.35±8.20</td>
<td>21.60±7.65</td>
<td>24.30±14.00</td>
</tr>
<tr>
<td>Leptin (µg/l)</td>
<td>6.05±3.40</td>
<td>5.60±4.30</td>
<td>7.26±3.85</td>
<td>7.40±3.95</td>
</tr>
<tr>
<td>Adiponectin (µg/ml)</td>
<td>6.50±2.75</td>
<td>6.00±3.50</td>
<td>7.30±2.55</td>
<td>7.05±2.10</td>
</tr>
<tr>
<td>KITT (%/min)</td>
<td>2.15±0.65</td>
<td>3.25±0.85**</td>
<td>1.95±1.00</td>
<td>1.80±0.90</td>
</tr>
</tbody>
</table>

*p < 0.001, **p < 0.02 after/before values in the trained vs control group.
KITT, constant rate of plasma glucose disappearance during the ITT.
adiponectin levels. Individual variation of adiponectin levels in trained and control groups according to insulin sensitivity changes are shown in Fig. 2.

**Discussion**

Our data show that a supervised intensive training program did not induce significant changes in adiponectin and leptin levels despite a tremendous decrease in abdominal fat and improvement in insulin sensitivity in sedentary middle-aged type 2 diabetic men maintaining their usual dietary habits.

Due to reduced sample size and possible heterogeneity of the study population with regard to age, body weight, hypoglycemic treatment and diabetes control at baseline, we focused mainly on the individual variations of the defined target variables over the study period.

There is some evidence from the literature that only reductions in body weight above the threshold of 10% are likely to result in a significant decrease in circulating leptin levels (1, 10, 11). Such a threshold effect is likely to also apply to adiponectin variations, since an increase in circulating adiponectin levels has been reported in obese patients following a weight loss of 10% or more consecutive to restrictive diet or gastric banding (3, 12). This magnitude of weight loss was not achieved in our study (2.2% weight loss), probably because the reduction in visceral adiposity was balanced by the increase in muscle mass. The marked reduction in abdominal adipose tissue induced by our training program was not associated with crude increase in adiponectin levels. However, we found an inverse relationship between changes in body weight and changes in adiponectin levels in the trained group.
According to Tataranni’s group (6, 13), hypoadiponectinemia is more closely related to insulin resistance than adiposity. In the present study, the tremendous improvement in insulin sensitivity in the trained group was not associated with a significant change in adiponectin and leptin levels. These results are unlikely to be explained by hypoglycemic medications among the groups since no significant change in adiponectin levels are reported in patients taking metformin (14). With the exception of the study by Perusse et al. (15), other exercise training protocols did not affect leptin (16) or adiponectin levels (12).

Exercise improves insulin sensitivity at least in part through AMP kinase pathway activation (17). It has recently been shown that adiponectin also increases muscular insulin sensitivity through the same pathway (18). Thus elevation of adiponectin levels may no longer be necessary to increase insulin sensitivity during exercise training. Contrasting with diet-induced weight loss, the improvement of insulin sensitivity by training is not related to adiponectin variations.

In conclusion, despite a decrease in abdominal adiposity and improvement in insulin sensitivity, an 8 week intensive training program did not significantly affect fasting leptin and adiponectin levels, suggesting the absence of a direct cause–effect relationship between adiposity, insulin sensitivity and these adipocytokines.

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References


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