ORIGINAL ARTICLE

Strength training improves insulin sensitivity and plasma lipid levels without altering body composition in overweight and obese subjects

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Abstract

Objective: To assess the effect of long-term strength training on insulin sensitivity, lipid profile, and body composition in overweight and obese subjects.

Materials and methods: A prospective, randomized, interventional study of 16 overweight or obese subjects aged 18-35 years who were investigated before and at the end of 8 weeks of strength training. The experimental group (n = 8) followed a strength training program consisting of 4 sessions per week at 50% to 80% of repetition maximum (RM), estimated through the 1RM test. The control group (n = 8) did not perform the training program. Glucose, insulin, total cholesterol, triglycerides, HDL-C, VLDL-C, and LDL-C levels and arterial index were determined. Insulin sensitivity was measured by calculating HOMA-IR (Homeostatic Model Assessment-Insulin Resistance). Indicators of body composition included weight, height, waist circumference, body fat, fat weight, muscle mass, somatotype chart and distance.

Results: At the end of intervention, the experimental group showed decreased insulin sensitivity (3.5 ± 0.9 vs. 2.9 ± 1.2; p = 0.04), LDL-C (106.9 ± 20.8 vs. 95.5 ± 14.2; p = 0.03) and arterial index (4.0 ± 0.6 vs. 3.5 ± 0.5; p = 0.01) values and increased HDL-C levels (43.7 ± 8.8 vs. 46.9 ± 5.6; p = 0.04), while the control group remained stable. There were no significant differences between the groups regarding body composition, somatotype chart or distance after training.

Conclusions: In overweight and obese subjects, strength training for eight weeks improved insulin sensitivity and lipid profile without altering body composition.

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KEYWORDS
Exercise; Strength training; Overweight; Obesity; Insulin sensitivity; Lipids

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Introduction

The association of a sedentary lifestyle and obesity has been identified as a risk factor that increases the chances of experiencing various non-transmissible chronic diseases, including coronary artery disease and cerebrovascular disease, as well as some types of cancer. In addition, epidemiological studies conducted in Latin America and the Caribbean show that approximately 50%-60% of overweight and obese adults have a sedentary lifestyle and a two-fold greater risk of suffering metabolic syndrome. In countries such as Mexico and Chile, approximately 45% of adults are overweight, and in Colombia the proportion rises to 55%.

This problem has led international organizations such as the American College of Sports Medicine, the American Heart Association, the Center for Disease Control and Prevention, and the Department of Health in London to recommend at least 30 minutes of physical activity of moderate intensity or more days a week or, failing this, 20 minutes of physical activity of high intensity 3 or more days a week as a strategy for controlling body weight and sedentary lifestyle. These recommendations are based on strong evidence showing the impact of both aerobic and anaerobic or strength building physical exercise (PE) on the treatment and prevention of several cardiometabolic diseases such as high blood pressure, atherosclerosis, obesity, and MS.

At the molecular level, strength training induces initiation signals leading to the translocation of GLUT-4 (a glucose transporter) through the phosphorylation of adenosine monophosphate-activated protein kinase (AMPK). On the other hand, strength training is an attractive model for research into the effects of PE on metabolic health despite a controversial, albeit highly desirable, body weight loss. Several authors have shown strength training to improve insulin sensitivity (as measured through HOMA-IR: Homeostatic Model Assessment–Insulin Resistance) thus significantly contributing to lipid metabolism and an improved glucose uptake by tissue.

In addition to reducing all-cause mortality, anaerobic or strength training (e.g. weight and machine training circuits) has been shown to improve health and decrease the risk of a number of non-transmissible chronic diseases such as type 2 diabetes mellitus, osteoporosis, obesity, and depression, amongst others. The objective of this study was therefore to assess whether a directed strength training program for 8 weeks improved insulin sensitivity, lipid profile, and body composition in overweight and obese subjects, as this type of training is increasingly popular among patients with excess weight and diabetes.
Materials and methods

Design and participants

A prospective, randomized, interventional study was conducted on 16 overweight or obese young subjects from a higher education center in Cali (Colombia). The sample was selected by advertising and sampling by intention. Subjects with a medical or clinical diagnosis of major systemic disease (including malignant conditions), type 1 or 2 diabetes mellitus, high blood pressure, hypothyroidism/hyperthyroidism, body mass index (BMI) < 25 kg/m², history of drug or alcohol consumption, use of multivitamin preparations, statin use, and inflammatory (trauma, contusions) or infectious conditions were excluded from the study. Written informed consent was obtained from each participant, and the ethics committee of the academic center approved the intervention in compliance with the ethical standards set out in the Declaration of Helsinki and the applicable legal regulations in Colombia governing research in humans (Decision 008430, of 1993, of the Ministry of Health).

Biochemical measurements

Ten milliliters of blood were drawn into Vacutainer tubes with no additive by puncture into an antecubital vein. Metabolic markers were measured using the following procedures: glucose, total cholesterol, triglycerides (TG), and high density lipoprotein cholesterol (HDL-C) were tested using a direct colorimetric method in an automated spectrophotometer solubilization with detergent (Biosystems, Spain)26. Very low density lipoprotein cholesterol (VLDL-C) and low density lipoprotein cholesterol (LDL-C) were calculated using the Friedewald et al equations27: VLDL-C = TG/5; and LDL-C = total cholesterol – HDL-C – VLDL-C. Arterial index was similarly calculated using the formula: total cholesterol (HDL-C). Insulin levels were measured by a chemiluminescence assay (Immulite 1000 kit, San Jose, CA)28. Insulin sensitivity was measured by calculating the HOMA-IR index (Homeostatic Model Assessment-Insulin Resistance) using the formula: HOMA index = (basal insulin in mU/L) x basal blood glucose in mmol/L)/22.5.

Measurement of body composition

The following anthropometric variables were measured in each participant: height, weight, skin folds (triceps, subscapular, biceps, iliac, supraspinial, abdominal, middle thigh, and medial calf), circumferences (relaxed and contracted arm, forearm, wrist, chest, waist, hip, middle thigh, calf, and ankle), bone diameters (bistyloid, biepicondylar, and bicondylar). Procedures standardized by López et al29 were used for these measurements. Somatotype, somatic chart, and somatotype distance by groups were calculated using the equations described by Carter30 and the principles laid down by Robert and Baimbridge31 respectively. Body composition was studied using the Parízcová and Buzcova method32, and the results obtained were expressed as kilograms of fat, kilograms of muscle mass, and percent of body fat.

Intervention

Participants were randomized to an experimental group (n = 8) which followed an 8-week training program consisting of 4 sessions per week at an intensity ranging from 50% to 80% of repetition maximum (MR), estimated through the 1RM test using the following equation: 1RM = lifted weight/((100 – [number of repetitions x %]) x 100. Eight exercises with machines and free weights involving most muscle segments and groups were selected. The control group (n = 8) did not follow the training program.

Statistical analysis

An exploratory analysis was first performed to determine the frequency and distribution of each of the variables tested. Outcome measures were compared between groups, before and after intervention, using a Wilcoxon signed rank test33 because of sample distribution and size. All statistical tests were performed using software SPSS 15.0 for Windows (Graphpad Instat, Graphpad Software, University of London, London [United Kingdom]). A value of p ≤ 0.05 was considered statistically significant.

Results

Participants had a mean age of 23.7 ± 5.4 years (range 19-35 years), a mean body weight of 84.3 ± 11.9 kg (range 57-97 kg), a mean height of 168.0 ± 9.3 cm (range 148-185 cm), and a mean BMI of 28.5 ± 2.1 kg/m² (range 27-32 kg/m²). At study start, there were no significant differences in any of the tested variables, except for baseline triglyceride levels (Table 1). At the end of intervention, participants in the experimental group showed, as compared to baseline values, an 18% decrease in insulin sensitivity as measured with the HOMA index (3.5 ± 0.9 versus 2.9 ± 1.2; p = 0.04), a 10% decrease in LDL-C levels (106.9 ± 20.8 versus 95.5 ± 14.2 mg/dl; p = 0.03), and a 13% decrease in the arterial index (4.0 ± 0.6 versus 3.5 ± 0.5; p = 0.01), as well as an 8% increase in HDL-C levels (43.7 ± 8.8 versus 46.9 ± 5.6; p = 0.04) (Table 1). No significant differences were found in the endomorphic, mesomorphic, and ectomorphic components of somatotype, somatic chart, and somatotype distance (X and Y coordinates) by groups or time points (Fig. 1), or in body composition (Table 1).

As expected, after 8 weeks of training, the experimental group achieved an improvement close to 30% in maximum strength, as measured by the RM test (1RM = 127 at baseline versus 1RM = 168 after intervention; p = 0.04). Of the 8 subjects did not complete the whole program, and adherence in the experimental group was therefore 75%.

Discussion

The hypothesis of this pilot study was to assess whether strength training for 8 weeks would improve insulin sensitivity, lipid profile, and body composition in overweight or obese subjects. This hypothesis was confirmed, because 8 weeks of training resulted in a positive change in the HOMA-IR resistance index and lipid markers: LDL-C, arterial
Table 1  Results of body composition and metabolic profile variables by group before and after intervention

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control group at baseline (n= 8)</th>
<th>Experimental group at baseline (n= 8)</th>
<th>p*</th>
<th>Control group at study end (n= 8)</th>
<th>p</th>
<th>Experimental group at study end (n= 6)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight, kg</td>
<td>89.2 ± 8.0</td>
<td>78.9 ± 13.4</td>
<td>0.10</td>
<td>88.0 ± 11.6</td>
<td>0.62</td>
<td>75.9 ± 13.4</td>
<td>0.87</td>
</tr>
<tr>
<td>Height, cm</td>
<td>172.0 ± 6.9</td>
<td>164.0 ± 8.9</td>
<td>0.89</td>
<td>171.0 ± 6.9</td>
<td>0.42</td>
<td>163.1 ± 10.2</td>
<td>0.70</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>30.1 ± 1.9</td>
<td>29.1 ± 2.8</td>
<td>0.45</td>
<td>29.7 ± 3.1</td>
<td>0.94</td>
<td>28.3 ± 2.2</td>
<td>0.72</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>26.4 ± 2.2</td>
<td>26.4 ± 2.4</td>
<td>0.99</td>
<td>25.8 ± 3.6</td>
<td>0.67</td>
<td>24.9 ± 2.5</td>
<td>0.28</td>
</tr>
<tr>
<td>Fat weight, kg</td>
<td>11.6 ± 2.6</td>
<td>9.9 ± 3.9</td>
<td>0.34</td>
<td>11.1 ± 4.2</td>
<td>0.51</td>
<td>8.0 ± 2.7</td>
<td>0.58</td>
</tr>
<tr>
<td>Muscle mass, %</td>
<td>37.4 ± 1.9</td>
<td>38.5 ± 2.8</td>
<td>0.39</td>
<td>37.7 ± 1.8</td>
<td>0.52</td>
<td>39.8 ± 2.0</td>
<td>0.57</td>
</tr>
<tr>
<td>Endomorphic component</td>
<td>8.27 ± 0.48</td>
<td>8.45 ± 0.36</td>
<td>0.31</td>
<td>7.92 ± 0.56</td>
<td>0.56</td>
<td>8.17 ± 0.85</td>
<td>0.60</td>
</tr>
<tr>
<td>Mesomorphic component</td>
<td>5.87 ± 1.10</td>
<td>6.00 ± 1.10</td>
<td>0.17</td>
<td>5.78 ± 1.17</td>
<td>0.66</td>
<td>5.90 ± 0.79</td>
<td>0.35</td>
</tr>
<tr>
<td>Ectomorphic component</td>
<td>0.20 ± 0.48</td>
<td>0.10 ± 0.50</td>
<td>0.73</td>
<td>0.38 ± 0.79</td>
<td>0.19</td>
<td>0.27 ± 0.41</td>
<td>0.97</td>
</tr>
<tr>
<td>Waist circumference</td>
<td>95.7 ± 6.5</td>
<td>92.6 ± 8.6</td>
<td>0.91</td>
<td>93.8 ± 8.7</td>
<td>0.98</td>
<td>89.5 ± 8.2</td>
<td>0.17</td>
</tr>
<tr>
<td>Waist/hip ratio</td>
<td>0.84 ± 0.08</td>
<td>0.84 ± 0.08</td>
<td>0.35</td>
<td>0.87 ± 0.09</td>
<td>0.33</td>
<td>0.85 ± 0.06</td>
<td>0.34</td>
</tr>
<tr>
<td>Insulin, µU/mL</td>
<td>17.7 ± 2.5</td>
<td>17.2 ± 9.1</td>
<td>0.29</td>
<td>19.0 ± 7.4</td>
<td>0.80</td>
<td>14.1 ± 8.2</td>
<td>0.38</td>
</tr>
<tr>
<td>Glucose mg/dL</td>
<td>77.4 ± 7.2</td>
<td>84.0 ± 6.8</td>
<td>0.76</td>
<td>82.5 ± 6.6</td>
<td>0.34</td>
<td>83.6 ± 6.4</td>
<td>0.58</td>
</tr>
<tr>
<td>HOMA index</td>
<td>3.6 ± 0.7</td>
<td>3.5 ± 0.9</td>
<td>0.33</td>
<td>3.6 ± 1.4</td>
<td>0.65</td>
<td>2.9 ± 1.2</td>
<td>0.04</td>
</tr>
<tr>
<td>Triglycerides, mg/dL</td>
<td>163.9 ± 85.8</td>
<td>99.2 ± 40.9</td>
<td>0.07</td>
<td>223.8 ± 140.8</td>
<td>0.08</td>
<td>119.3 ± 55.3</td>
<td>0.38</td>
</tr>
<tr>
<td>Cholesterol, mg/dL</td>
<td>161.4 ± 30.3</td>
<td>170.5 ± 18.3</td>
<td>0.57</td>
<td>170.5 ± 14.8</td>
<td>0.86</td>
<td>166.3 ± 13.3</td>
<td>0.24</td>
</tr>
<tr>
<td>HDL-C, mg/dL</td>
<td>48.4 ± 9.4</td>
<td>43.7 ± 8.0</td>
<td>0.71</td>
<td>39.2 ± 7.4</td>
<td>0.44</td>
<td>46.9 ± 5.6</td>
<td>0.04</td>
</tr>
<tr>
<td>LDL-C, mg/dL</td>
<td>85.6 ± 23.1</td>
<td>106.9 ± 20.8</td>
<td>0.34</td>
<td>89.4 ± 27.0</td>
<td>0.88</td>
<td>95.5 ± 14.2</td>
<td>0.03</td>
</tr>
<tr>
<td>VLDL-C, mg/dL</td>
<td>32.8 ± 17.2</td>
<td>23.8 ± 11.0</td>
<td>0.12</td>
<td>35.6 ± 15.6</td>
<td>0.63</td>
<td>19.8 ± 8.2</td>
<td>0.38</td>
</tr>
<tr>
<td>Arterial index</td>
<td>3.5 ± 0.5</td>
<td>4.0 ± 0.6</td>
<td>0.99</td>
<td>4.2 ± 0.8</td>
<td>0.69</td>
<td>3.5 ± 0.5</td>
<td>0.01</td>
</tr>
</tbody>
</table>

HDL-C: high density lipoprotein cholesterol; LDL-C: low density lipoprotein cholesterol; VLDL-C: very low density lipoprotein cholesterol; BMI: body mass index.
Values are mean ± standard deviation, adjusted for sex, age, and BMI.
Group differences by Wilcoxon signed rank test.

* p: comparison of both groups before intervention.
  † p: comparison of baseline versus 8 weeks in the control group.
  ‡ p: comparison of baseline versus 8 weeks in the experimental group.

![Figure 1](image)

Figure 1  Somatotype, chart, and somatotype distance (X and Y coordinates) by group.
index, and HDL-C (p ≤ 0.05). The metabolic effect seen in this study agrees with that reported by other studies using strength programs14,15. For instance, a study by Kretschmer et al16 showed that a 5-week combined training program (strength and resistance) was sufficient for body weight maintenance and reduction (expressed as percent of body fat) and increased insulin sensitivity. This significant effect is very important in patients with type 2 diabetes mellitus who have insulin resistance or are overweight, because an impairment occurs in the insulin-dependent signaling mechanisms that regulate glucose transport in tissue. Although weight training does not have a clear effect on the insulin receptor, the phosphorylation of IRS-1 proteins (an insulin receptor substrate), or PI3-K (phosphatidylinositol 3-kinase) activity17, GLUT-4 translocation clearly occurs through the action of proteins such as AMPK, CaMK (Ca2+/calmodulin-dependent protein kinase), and aPKC (atypical protein kinase), signaling molecules which are involved in glucose uptake stimulated by muscle contraction.

An interesting finding was that an improvement in the metabolic function markers was not associated with changes in the assessed body composition variables. However, we cannot state that weight training did not induce changes in other compartments such as visceral fat, limbs, etc. because these were not assessed in this study. This unique finding is supported by a meta-analysis of 3,476 subjects published by Shaw et al38 in 2005. As compared to no treatment, regular PE induced a small weight loss in the 43 studies analyzed. The combination of PE with a diet caused a greater mean weight decrease (MWD) than diet alone (MWD −1.1 kg; 95% confidence interval [CI], -1.5 to -0.6). PE of increased intensity achieved a greater weight loss (MWD −1.5 kg; 95% CI, −2.3 to −0.7). All studies, including combined training or circuits with weights, showed significant differences in the same parameters measured in this study, such as serum lipids, glucose, and fasting insulin. This agrees with our results.

Similarly, studies including high intensity protocols induce a greater reduction in fasting blood glucose than low intensity PE (−0.3 mmol/L; 95% CI, −0.5 to −0.2), a finding that was not observed in this study. Improvement in blood lipid levels, particularly the increase (△8%) in HDL-C as compared to sedentary subjects, was similar to that reported by Durstine et al39 and the Kelley and Kelley meta-analysis40, which reported that 8 weeks of PE were sufficient to significantly increase HDL-C levels in adults with characteristics similar to those included in this study without altering any other blood lipid. HDL-C is a lipoprotein that protects against atherosclerosis, partly because of its antiproliferative and antithrombogenic effects41. The same finding was observed in the experimental group of our study, as lower TG and LDL levels were seen after intervention with strength training.

The results of this study, where no adverse effects such as musculoskeletal lesions or cardiovascular events were seen, support strength training as an intervention that may improve the metabolic state, even if no changes are seen in body composition. Our results should be interpreted with caution due to two limitations in the study. The first was the small subject sample. The second was that feeding patterns, which may modulate metabolic response, were not taken into account. However, all things considered, our results represent a challenge to health care professionals and to everyone else responsible for the promotion of physical activity to reinforce the impact of different physical training proposals in this type of population. To sum up, this study showed that 8 weeks of weight training improves insulin sensitivity and lipid profile in overweight and obese males without modifying body weight.

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Conflict of interest

The authors state that they have no conflict of interest.

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