

Effect of progressive resistance training on insulin resistance and plasma adiponectin concentration in overweight men

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Abstract

Background and objectives: The purpose of the present study was to examine the effect of a progressive resistance-training program beside an energy deficit diet on body Composition, insulin resistance and plasma adiponectin levels in overweight adult men.

Methods: Twenty-five overweight men (age = 32.1 ± 4.3 years, BMI = 28.9 ± 1.3 kg.m⁻²) were randomly assigned into one of the two groups: resistance training+ diet (RD, n =15); and diet only (DO, n =12). Both groups undertook a 12-week weight loss program using a moderate isocaloric energy deficit.

Results: After 12 weeks intervention, there was equivalent significant weight loss for both groups ($p < 0.01$). Total and abdominal fat mass, fasting insulin concentration and HOMA-IR score were decreased in both groups ($p < 0.01$). All previously mentioned variables except fasting insulin levels were more significantly reduced in RD compared to DO group. There was a significant decrease in lean body tissue only in DO ($p < 0.01$) group. We did not find any significant changes in plasma adiponectin concentration of the experimental groups. Reduction in insulin resistance had a significant positive correlation with abdominal and total fat loss ($p < 0.05$) and a negative correlation with lean mass loss ($p < 0.05$).

Conclusion: Adding a progressive resistance-training program to an energy deficit diet, without any change on magnitude of weight loss could prevent the loss of lean body mass and improve the reduction of total and abdominal fat mass and insulin resistance. Moreover, the change in body composition and insulin resistance was accompanied with no significant change in plasma adiponectin.

Keywords: Resistance training; HOMA; Adiponectin; Weight Loss

Introduction

Adiponectin is an adipose tissue-derived cytokine protein that has been closely associated with insulin action (1). Total

plasma adiponectin levels have been proposed to negatively correlate with obesity and insulin resistance (2). The possible mechanisms for adiponectin action on insulin sensitivity are the reduction of hepatic

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glucose production (3), and increase of fatty acid oxidation, thus decreasing plasma free fatty acid levels (4). Based on the existing evidence, interventions such as weight reduction and exercise training accompanied with a decrease in insulin resistance (5), may achieve this through modulation of adiponectin levels.

Diet only interventions known as effective strategies for managing the metabolic disorders in obese people (6), are accompanied with a significant reduction in fat free mass (FFM) which leads to impaired energy metabolism including reduced fat oxidation (7). On the other hand, the study results show that a decrease in lean body mass is associated with decreased insulin sensitivity (8). Therefore, it has been suggested that adding suitable exercise training to a diet program in order to reserve FFM (9) causes greater improvement in insulin action than diet only (10). Although some studies have shown that performing resistance training beside a low calorie diet preserve lean body mass during weight loss program (11), the number of studies conducted to detect the effects of resistance training on circulating adiponectin and the biological role of adiponectin on the insulin sensitivity are narrow and controversial ((12); (13); (14)). Some evidence indicates that the high intensity resistance training has

increased plasma adiponectin levels with accompanied improved insulin action (12). However, all resistance training interventions are not associated with significant changes in plasma adiponectin concentrations despite enhanced insulin sensitivity (14). In this context, some investigators reported that performing resistance training beside a diet, is accompanied with even a reduction in plasma adiponectin levels, despite significant improvements in insulin sensitivity (13).

The present study intended to define the role of progressive resistance training during weight loss program on insulin resistance and the level of serum adiponectin in overweight men. Consequently, the main hypothesis of this study was that adding the 12 weeks of resistance training to hypocaloric diet can result in the improvement of insulin resistance that might be related to alterations in circulating adiponectin.

Materials and Methods

Subjects

Twenty-seven adult men (age = 24-39 years) were recruited for the present study. The subjects had been registered in a weight loss program by Iranian Health Clinic, Isfahan, Iran. All subjects undertook a full medical examination, filled up the physical activity questionnaire and weight and height were

measured before inclusion. We omitted the participants who were smokers, had any severe illness (diabetes, cardiovascular disease...), or were taking medication that could affect laboratory test results. The Participants were overweight ($27 \leq \text{BMI} \leq 30 \text{ kg.m}^{-2}$) with a sedentary lifestyle (<20 min exercise twice per week). Each subject signed an informed consent document before participation. This study was approved by Research Ethical Board of Tehran Azad University.

Experimental design

The participants after being matched for age and BMI were randomly assigned into one of two intervention groups for 12 weeks: (1) the resistance training + diet group (RD; n=15); or (2) the diet only group (DO; n=12). Those in the RD group experienced a supervised resistance training period beside a dietary regimen. The participants in the DO group were asked to follow their sedentary lifestyle along with a dietary regimen.

Interventions

Training. The first 2 weeks were used to familiarize RD group to the exercise techniques and to measure one repetition maximum (1-RM) action in each exercise. This period also used in order to instruct all subjects about the planed diet and prepare

them for the 12-week intervention. To determine the 1RM, Subjects received warm-up in a set of six repetitions at the load that could be raised about 15 times. Thereafter, four to five separate, single efforts were executed until the participants were unable to complete the one repetition lift. All exercise sessions were performed under the supervision of an exercise physiologist to assure each participant exercising in the correct form and reasonable technique. A progressive resistance raining program was performed in the RD group. Each training session included six exercises for the main muscle groups of the body (leg press, bench press, knee curl, shoulder press, lateral pull-down, abdominal crunch). During the initial 5 weeks of the training program the participants exercised by lifting of 50–70% of the individual 1RM. At this stage, participants completed 12–15 repetitions per set and three sets for each exercise. Throughout the last 5 weeks of the program, the loads were 70–80% of the 1RM that were executed in three sets and 8-10 repetition per set. Rest periods of about 1 minute were assumed between each set during the training session. The total training program was carried on for 12 weeks, three times per week (Saturday, Monday, and Wednesday). Each main exercise session lasted about 40 to 45 min.

Diet. Daily caloric requirements were predicted using body weight, height, age, sex, and physical activity level. The weight loss diet involved an energy deficit diet by 2000-3000 Kcal/week in RD group and 3000-4000 kcal/week in DO group. The dietary protocol was a balanced healthy diet consisting of the six categories of foods. The balanced diet contained 50-55% carbohydrate, 20-25% protein, and 25-30% fat. This diet was planned to produce about 0.5 kg weight loss per week. During the 12-week intervention period, subjects visited the nutritionist once a week to take their diet, general knowledge on diet and weight reduction and weighing.

Experimental assessments

The same assessor at both the baseline and after the 12-week intervention assessed all subjects. Baseline testing was completed during two days before starting of program and the posttests were carried out 48 h after the last exercise session. The participants were not performed exercise 48 hours prior to testing.

Anthropometrics. The anthropometric characteristics of the present study were included the body weight, height and BMI. Body weight, without shoes and excess clothing, was determined in kilograms (measuring accuracy: 0.1 Kg), and height was measured with bare feet in standing position

(measuring accuracy: 1.0 cm). The measurements were performed in a fasted state on the test day in the morning between 8.00 -9.00 am. During the intervention period, the participants were instructed to monitor their weekly body weight after waking up and before breakfast without the excess clothes. BMI was calculated as body mass (kg) divided by the square of height (m²).

Body composition. Body composition measurement was attained using Dual-energy X-ray absorptiometry (DEXA) scanning (Hologic Discovery-A; Integrity Medical Systems). To decline deviations in hydration grade, the scans were gotten after 8–9 hours of fasting. Daily calibration was done to ensure that the devices provided equal results. We standardized subject location for the scans and followed the manufacturer recommendations. To assess abdominal adiposity, one trained assessor intercepted a section between T12 and L4 from the whole-body DEXA scans.

Blood analysis. Blood samples were taken in the morning after an 8-9 h overnight fast. Blood samples were centrifuged for 10 min at 4°C and plasma were stored at -80°C for following analyses. The blood samples collection were executed three days before starting of training program and 48 h after the last exercise session. Plasma adiponectin

levels were measured using a specific ELISA kit (Phoenix Peptides, Karlsruhe, Germany). The intra-assay coefficient variation (CV) was about 5%. Human insulin ELISA kit were used to measure the plasma insulin concentration (DiaMetra, Foligno-Perugia, ITALY) with an intra-assay CV less than 4.5%. The glucose oxidase technique was used to measure Blood-glucose levels. Insulin resistance was calculated by the homeostasis model assessment (HOMA) for all subjects. HOMA-IR was calculated by (fasting glucose (mmol/l) \times fasting insulin (μ u/ml) / 22.5) (15). A single assessor took all samples in duplicate for each factor.

Statistical analyses

Descriptive data were reported as means \pm standard deviations. Group differences at baseline for all variables were examined using independent Student's t-test. Paired Student *t* test were used to determine pre–post differences within each group. The two-way repeated measures ANOVA method was applied to measure group–time interactions (group \times time; 2 \times 2). Pearson correlation was used to examine associations between body composition, insulin resistance and plasma adiponectin levels change. The Alpha level was primarily set at $p < 0.05$ to define statistical significance. SPSS version 15.0

(SPSS Inc., Chicago, IL, USA) was used to analyze all data.

Results

Table 1 shows all characteristics at baseline. There were no significant differences between the two groups on any parameter before intervention (Table 1). Body weight reduction after the 12-week intervention in the DO group were 5.4% (5.0 ± 1.4 kg, $p < 0.01$) and in the RD group 5% (4.6 ± 2.1 kg, $p < 0.01$) respectively. In DO group, significant decreases were observed in fat mass ($p < 0.01$), abdominal fat mass ($p < 0.01$), lean body mass ($p < 0.01$), plasma insulin concentration ($p < 0.01$) and HOMA-IR ($p < 0.05$). Whereas, there was no significant change in blood glucose level and plasma Adiponectin, in RD group, we found significant reduction in fat mass ($p < 0.01$), abdominal fat mass ($p < 0.01$), plasma insulin concentration ($p < 0.01$), blood glucose level ($p < 0.01$) and HOMA-IR ($p < 0.01$). However, no significant variation in total lean body mass and plasma Adiponectin was observed in this group (Table 2).

A significant group–time interaction was observed for some parameters. In comparison with the DO group the RD group had greater decreases in blood glucose level ($p < 0.05$), total fat mass ($p < 0.01$), abdominal fat mass ($p < 0.01$), HOMA-IR score ($p < 0.05$) without

redaction in total lean body mass ($p < 0.05$). However, there were no significant group–time interaction in body weight, plasma insulin concentration, and plasma Adiponectin level (Table2).

In the baseline, insulin resistance was positively correlated with body weight ($r = 0.375$, $P < 0.05$), total body fat ($r = 0.433$, $P < 0.05$), abdominal fat mass ($r = 0.374$, $P < 0.05$) and negatively correlated with adiponectin levels ($r = -0.546$, $P < 0.05$)

whereas, no correlation was observed with lean body mass. After 12-week intervention, the changes in HOMA-IR has shown a strongly positive correlation with changes in total body fat ($r = 0.633$, $P < 0.01$) and abdominal fat mass ($r = 0.408$, $P < 0.05$) and a negative association with changes in lean body mass ($r = -0.470$, $P < 0.05$). Moreover, no correlation was observed between HOMA-IR and adiponectin level change.

Table 1: Baseline values

	RD (n=15)	DO (n=12)	p
Age (years)	31.5 ± 4.4	32.6 ± 4.4	0.502
Height (cm)	175 ± 4	179 ± 3	0.062
Body Weight (kg)	89.4 ± 8.0	92.1 ± 5.6	0.335
BMI (kg.m⁻²)	29.1 ± 1.4	28.7 ± 1.2	0.403
Fat Mass (kg)	33.9 ± 7.5	34.5 ± 5.0	0.815
Abdominal Fat Mass (kg)	4.8 ± 1.1	4.5 ± 0.9	0.426
Lean Body Mass (Kg)	52.8 ± 3	53.5 ± 4	0.076
Glucose (mmol.l⁻¹)	5.1 ± 0.4	4.9 ± 0.7	0.082
Insulin (μu.ml⁻¹)	8.5 ± 1.5	9.5 ± 1.6	0.125
HOMA-IR	1.94 ± 0.47	1.92 ± 0.4	0.894
Adiponectin (μg.ml⁻¹)	3.1 ± 0.5	2.8 ± 0.5	0.20

RD, diet and resistance training combined; DO, diet only; BMI, body mass index. Data are presented as means ± SD.

Table 2: pre and post intervention values

	RD			DO			(G × T)
	pre	post	p	pre	post	p	p
Age (years)	31.5 ± 4.4	-	-	32.6 ± 4.4	-	-	-
Height (cm)	175 ± 4	-	-	179 ± 3	-	-	-
Body Weight (kg)	89.4 ± 8.0	84.8 ± 6.9	<0.001**	92.1 ± 5.6	87.1 ± 5.9	<0.001**	0.586
BMI (kg.m⁻²)	29.1 ± 1.4	27.6 ± 1.4	<0.001**	28.7 ± 1.2	27.00 ± 1.3	<0.001**	0.517
Fat Mass (kg)	33.9 ± 7.5	29.0 ± 6.1	<0.001**	34.5 ± 5.0	31.9 ± 5.8	<0.001**	0.001†
Abdominal Fat Mass (kg)	4.8 ± 1.1	3.7 ± 0.8	<0.001**	4.5 ± 0.9	4.2 ± 1	0.001**	<0.001†
Lean Body Mass (Kg)	52.8 ± 3	53.6 ± 2.7	0.069	53.5 ± 4	51.0 ± 2.2	0.001**	<0.001†
Glucose (mmol.l⁻¹)	5.1 ± 0.4	4.3 ± 0.3	<0.001**	4.9 ± 0.7	4.8 ± 0.4	0.560	<0.001†
Insulin (µu.ml⁻¹)	8.5 ± 1.5	7.0 ± 1.3	<0.001**	9.5 ± 1.6	8.2 ± 2.7	0.003**	0.651
HOMA-IR	1.94 ± 0.47	1.36 ± 0.3	<0.001**	1.92 ± 0.4	1.68 ± 0.5	0.021*	0.002†
Adiponectin (µg.ml⁻¹)	3.1 ± 0.5	3.2 ± 0.54	0.116	2.8 ± 0.5	2.6 ± 0.4	0.220	0.076

(G × T), group and time interaction. * Significant change from pre to post in RD and DO groups (P < 0.05).

** Significant change from pre to post in RD and DO groups (P < 0.01). † Significant group and time interaction (P < 0.01). Data are presented as means ±SD.

Discussion

The results of the present study indicated that a progressive resistance training plus diet

intervention has the advantage of preventing the loss of lean body mass and improving the reduction of total and abdominal fat mass

rather than the diet only intervention while both resulted in the same weight loss values. Whereas both weight loss programs decreased plasma insulin levels and insulin resistance; still, the insulin resistance reduction was significantly more in RD than DO group. The change in body composition and insulin resistance was accompanied with no significant changes in plasma adiponectin concentration in both groups. Insulin resistance reduction had a significant positive correlation with total and abdominal fat loss and a negative correlation with lean mass loss in both groups. However, we did not find any correlation between HOMA-IR and adiponectin level changes.

Weight loss in RD group was accompanied with an increase (no significant) in lean body mass, but in DO group considerable fraction (40%) of weight, loss was due to lean mass reduction. The findings in the RD group indicate that 100% of the weight reduction was lost as fat tissue. This finding is in agreement with those of (16), (9) and (17), (18) that reported resistance training could preserve fat free mass in subjects consuming a weight-loss diet. Elevated anabolic hormones in response to resistance training can explain the lean body mass retention in RD group. In this line, some studies have shown that resistance training can cause muscle

hypertrophy even in participants suffering severe energy restriction (19). However, hypertrophy was only observed in trained muscles. Thus, the resistance training could not inhibit the loss of whole body lean mass superior to diet alone (20). In contrast to our findings, some studies that used a very low calorie diet have suggested that resistance training cannot prevent the loss of lean body mass (21). Thus, it is likely that a baseline level of calorie intake (more than 1200 kcals) is needed that resistance trainings can retain the lean body mass. Consequently, the anabolic effect of progressive resistance training versus the catabolic nature of weight loss program can preserve the lean body mass.

Another finding of the present study was significantly more reduction in abdominal fat and total fat mass in the RT versus DO group. Few studies have conducted to consider the effects of performing resistance training beside a low calorie diet and even fewer studies have investigated this type of intervention on fat mass. Brochu et al (2009) concluded that dietary intervention in combination with resistance training was accompanied with more reduction of fat mass and body fat percent than dietary intervention alone (22). In a similar study (Hunter et al, 2002) reported that resistance training without

energy restriction showed a significant reduction in total body fat mass, despite no significant changes observed in body weight. Because resistance training in this study was not associated with more changes in body weight, the more reductions in fat mass occurred in the RD group may be related to maintaining or even increasing the fat free mass (16). In the diet only intervention, reduction in body weight is associated with decreases in both FFM and FM. Different changes in body fat were observed in the two groups may have been affected by differences in anabolic and catabolic hormones, such as cortisol and growth hormone. It has been shown that plasma growth hormone level is inversely related to body fat (23). Thus, a marked plasma growth hormone elevation after resistance exercise bout (24) could be an explanation for more reduction of fat mass in the RD group.

In both groups, we observed a significant improvement in insulin sensitivity, without any significant changes in plasma adiponectin levels. These results are in agreement with previous findings that indicated insulin resistance decreases after a moderate weight loss in the nonexistence of any change in serum adiponectin levels (25), (26). However, in contrast to our findings several studies have indicated that the increased insulin sensitivity following weight loss is

accompanied with increasing of serum adiponectin levels (27), (28), (29). The most likely explanation for this discrepancy is the difference in the amounts of weight loss. Indeed, the magnitude of weight loss in present study (5kg) compared to those of converse studies (>23 kg) was absolutely much less. This approves Madsen *et al.*'s results (2008) that demonstrated more than 10–11% weight loss is necessary in order to obtain a significant increase in serum adiponectin concentration in obese subjects (30). Collectively, these data show that moderate weight loss independent of serum adiponectin levels changing can improve the insulin resistance.

Regardless of adiponectin levels changing, our results showed that HOMA-IR scores were significantly reduced in both groups but the slope of reduction was significantly more in RD than DO group. Moreover, insulin resistance responsible for decreasing in insulin resistance can be the significant reduction in abdominal and total body fat mass due to negative energy balance. In this context, available evidence confirms a significant relationship between accumulation of abdominal and total body fat and insulin resistance (31). A possible explanation for greater effects of RD group on insulin sensitivity improvement may be the more reduction in abdominal and total fat loss in

this group. The other explanation may be the lean body mass preservation in RD group. In this line Poehlman et al (2000) reported that Insulin resistance improvement following resistance training is probably due to a mass effect (32). Based on this theory, the more skeletal muscle mass would enhance insulin sensitivity through a metabolic sink providing for glucose removal. The negative correlation between lean body mass loss and insulin resistance decline that observed in the present study can approve this suggestion. Whereas some studies suggested that resistance training-induced enhancement in insulin sensitivity could be due to increases in lean body mass (32), (33). The other findings provide the evidence that resistance training may contribute to improve insulin action through functional changes in skeletal muscle such as improvement in the insulin-signaling cascade, glycogen synthase and glucose transporter GLUT4 (34), (35). Thus, the both qualitative and quantitative changes in skeletal muscles may explain the improvement in insulin action following a resistance-training program.

Conclusion

The findings of the present study indicated that adding a progressive resistance-training program to an energy deficit diet, although dose not increase the magnitude of weight

loss, it could prevent the loss of lean body mass and improve the reduction of total and abdominal fat mass and insulin resistance. Therefore, the strength and conditioning professionals trying to help their clients to lose weight are recommended to design and implement resistance-training programs not only to burn calories more, but also to prevent the lean body mass loss also improve the health status. Moreover, the change in body composition and insulin resistance was accompanied with no significant change in plasma adiponectin concentration. These findings show that a progressive resistance-training program may be useful for patients who use an energy deficit diet at least for a period of 12 weeks. Further studies need to find the mechanisms related with these finding.

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Declarations

Human subjects

A written informed consent was obtained from all participants.

Conflict of interest

None

Authors' contributions

All authors contributed equally to this work

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