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Research article

Impact of diet, exercise and diet combined with exercise programs on plasma lipoprotein and adiponectin levels in obese girls

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Abstract

We studied the effect of three programs, diet restriction (D), individualized exercise training (E) at the maximal lipid oxidation point (LIPOXmax) and diet combined with exercise (D+E), on body mass, plasma lipoprotein and adiponectin levels in obese girls. Eighteen obese adolescents girls aged 12-14 years were studied. A longitudinal intervention was carried out, consisting of a two-month diet (D; –500 kcal·day^{-1}), of individualized exercise (E; 4 days/week, 90 min·day^{-1}) and of diet combined with exercise (D+E). Body mass, body mass index (BMI), body fat mass, waist circumference, substrate crossover point, LIPOXmax point, homeostasis model assessment (HOMA-IR) index, fasting levels of lipids and circulatory adiponectin, were measured in all subjects before and after the program. In subjects of the D+E group, body mass, BMI, body fat mass, waist circumference, HOMA-IR, low-density lipoprotein cholesterol (LDL-C) and total cholesterol / high-density lipoprotein cholesterol (TC/HDL-C) ratio were significantly lower, and HDL-C and adiponectin were higher after the program than that of subjects in the D or E groups. Diet/exercise improved the ability to oxidize lipids during exercise (crossover point: + 18.5 ± 3.4 of % Wmax; $p < 0.01$ and fat oxidation rate at LIPOXmax: + 89.7 ± 19.7 mg·min^{-1}; $p < 0.01$). In the D+E group, significant correlations were found between changes in body mass and adiponectin and between changes in the TC/HDL-C ratio and LIPOXmax. These findings show that the combined program of diet restriction and individualized exercise training at the LIPOXmax point is necessary to simultaneously improve body mass loss, adiponectin levels, as well as metabolic parameters, in obese girls.

Key words: Obese girls, lipoprotein, adiponectin, exercise training, diet restriction.

Introduction

Obesity is a major independent risk factor for cardiovascular disease (Scaglione et al., 2004). Skeletal muscle is largely involved in the development of obesity (Perez-Martin et al., 2001). 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 (Dumortier et al., 2003).

However, it is now well accepted that adipose tissue is a major endocrine organ producing a variety of factors that regulate energy metabolism and insulin sensitivity (Kershaw and Flier, 2004). An increased adipose tissue mass is associated with insulin resistance, hyperglycemia, hypertension and other components of the metabolic syndrome (Després, 2006).

Adiponectin levels decrease with obesity (Ariata et al., 1999), and low adiponectin concentration is associated with insulin resistance (Hotta et al., 2000). Body mass reduction is followed by an increase in plasma adiponectin concentration (Esposito et al., 2003) and a lowering of several indicators of cardiovascular risk, such as plasma lipids (Gerhard et al., 2004).

Obesity is characterized by three primary lipoprotein abnormalities: increased triglyceride-rich lipoproteins, increased small low-density lipoprotein (LDL) particles, and reduced levels of high-density lipoprotein (HDL) (Grundy, 1998). Moreover, HDL-C concentrations are commonly a reflection of insulin resistance (Karhapaa et al., 1994).

Several studies have shown that endurance exercise training has a beneficial effect on conventional plasma lipoprotein lipids and on circulatory adiponectin levels (Kraus et al., 2002; Kriketos et al., 2004). Indeed, Kang et al. (2002) demonstrated that physical activity had a beneficial effect on LDL particle diameters in obese adolescents. A recent meta-analysis reported that diet combined with exercise favours the reduction in LDL-C and triglycerides but lessens the increase in HDL-C, when compared with exercise alone (Leon and Sanchez, 2001). In addition, Kriketos et al. (2004) reported that fasting adiponectin levels increased by 260% above baseline values after 2-3 bouts of low to moderate intensity exercise.

Recently, exercise calorimetry has been developed by several teams in order to target more closely training protocols for both adults (Dumortier et al., 2003; Perez-Martin et al., 2001) and adolescents (Brandou et al., 2003) 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 adolescents.

Therefore, in this study we investigated separately the effects of two-month diet, individualized exercise training at the point where fat oxidation was maximal (LIPOXmax) and diet combined with exercise on the body composition, plasma lipoprotein, metabolic parameters and circulatory levels of adiponectin in obese girls.

Our working hypothesis was that exercise training

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combined with diet restriction would decrease body fat mass, insulin resistance and LDL-C and increase adiponectin and HDL-C via its effect on fat oxidation during exercise.

Methods

Subjects

We examined eighteen obese adolescent girls from two colleges in the centre of Tunisia. Obesity was defined as a body mass index (BMI; $kg \cdot m^{-2}$) greater than the 97th percentile defined by Cole et al. (2000). The pubertal stage was evaluated according to the Tanner classification (Tanner et al., 1966) by a trained paediatrician. Prepubertal adolescents comprised those subjects who where in Tanner stage I, pubertal adolescents those in Tanner stage II-III and post-pubertal adolescents in Tanner stage IV-V.

Criteria for participation in the present study included: no past history of cardiovascular disease, no history of smoking, no history of prescribed medicine, and no regular exercise. This study was approved by the Research Ethics Committee of the Faculty of Medicine, University of Sousse, Tunisia. The subjects were randomly assigned in 3 evenly divided program groups of 6 subjects: diet restriction (D), individualized exercise training (E) and diet/exercise (D+E). The adolescents and their parents gave a written informed consent for the experimental protocol.

Anthropometric measurements

Height was measured to the nearest 0.1 cm, waist circumference on the skin at the level of the navel to the nearest 0.2 cm, and total body mass to the nearest 0.1 kg on a digital scale (OHAUS, Florhman Park, NJ). Participants were nude or wearing only underwear for measurements of body mass. Body mass index (BMI) was calculated using the standard formula: body mass in kilograms divided by height in meters squared ($kg \cdot m^{-2}$).

The body fat percentage (BF%) was calculated by using the equation of Slaughter et al. (1988) for children with triceps and subscapular skinfolds < 35 mm: Girls = 1.33 (sum of 2 skinfolds) – 0.013 (sum of 2 skinfolds^2) – 2.5. BF% for children with triceps and subscapular skinfolds > 35 mm: Girls = 0.546 (sum of 2 skinfolds) + 9.7

Two skinfold thicknesses (triceps and subscapular) were measured in the subjects of the three groups, by the same trained observer to the nearest 0.1mm. Measurements were made on the right hand side of the body using a Harpenden calliper. Three measurements were taken at each site and the closest two measurements were averaged for use in the analysis. The test-retest data were then used to calculate the precision of all body composition measurements. Each anthropometric measurement was performed by the same technician for all participants before and after the two-month intervention.

Biochemical measurements

Fasting adiponectin and insulin were measured before and after the two-month intervention program. Plasma adiponectin was determined using an ELISA kit (B-Bridge international, inc). Insulin was assayed using an IRMA Insulin kit (Immunotech, France). Assays were carried out following the manufacturer's instructions.

Total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C) and glucose levels were measured in all subjects before and after the programs following 12 hours fasting using standardized techniques described by Wegge et al. (2004). Low-density lipoprotein cholesterol (LDL-C) was calculated as described by the Friedewald formula (Friedewald et al., 1972).

Homeostasis model assessment (HOMA-IR) was used to estimate the degree of insulin resistance, and calculated using the formula: HOMA-IR = [insulin ($mU \cdot liter^{-1}$) × glucose ($mmol \cdot liter^{-1}$)] / 22.5

To distinguish normal from impaired insulin sensitivity, HOMA-IR > 4.0 was the cut-off level employed for adolescents, according to the normal values provided by a previous study (Annunzio et al., 2004). Definition of the metabolic syndrome in adolescents was made according to the World Health Organization criteria (Alberti and Zimmet, 1998).

Exercise testing

The subjects performed an exercise test on an electromagnetically braked cycle ergometer (Ergo-line, Bitz, Germany) connected to a breath by breath device (ZAN 600, Meβgeräte, Germany) for gas exchange measurements (VO_2 and VCO_2). The conditions and requirements of the exercise testing were explained to each subject before the test. The laboratory temperature and relative humidity were between 22-24°C and 76% respectively during the test period.

Maximum oxygen consumption (VO_2max) and theoretical maximal working capacity (Wmax) were calculated for each subject before exercise testing using the predictive equations of Wasserman et al. (1986) for obese children. These equations take into account sex and anthropometric characteristics: Girl: $VO_2max = (52.8 \times M) - 303.4$. $Wmax = (VO_2max - 10 (\times M)) \times (10.3)^{-1}$. M is the body mass of the subject in kg.

The test consisted of a progressive increase in workload every 6 min with 5 steady-state workloads corresponding to 20, 30, 40, 50, and 60 % of Wmax. Heart rate was monitored electrocardiographically throughout the test (ZAN ECG 800, Meβgeräte, Germany). The subjects underwent a test with the same relative incremental workload and were compared at the same percentage of their Wmax. The results of this test were used to determine the exercise training intensity.

Carbohydrate (CHO) and fat oxidation rates were calculated from the gas exchange measurements according to the non-protein respiratory quotient (R) technique (Peronnet and Massicote, 1991): CHO oxidation rate ($mg \cdot min^{-1}$) = $4.585VCO_2 - 3.2255VO_2$. Fat oxidation rate ($mg \cdot min^{-1}$) = $1.6946\ VO_2 - 1.7012\ VCO_2$.

VO_2 and VCO_2 were determined as the means of measurements during the fourth and sixth minutes of each work load, according to MacRae et al. (1995). This technique provided CHO and lipid oxidation rates at different levels of exercise.

The percentage of CHO and fat oxidation were

Table 1. Subjects characteristics before and after the two-month program. Data are means (± SD).

	Diet (n = 6)		Exercise (n = 6)		Diet/Exercise (n = 6)	
	Before	After	Before	After	Before	After
Age (years)	13.4 (.2)		13.1 (.1)		13.0 (.4)	
PS (I / II-III / IV-V)	1 / 2 / 3		0 / 3 / 3		0 / 2 / 4	
Weight (kg)	79.8 (11.2)	75.6 (10.5) *	81.7 (12.2)	80.3 (12.9)	78.9 (9.2)	73.1 (8.6) **
BMI ($kg \cdot m^{-2}$)	30.5 (2.2)	28.6 (2.1) *	30.6 (2.3)	29.5 (1.8)	30.0 (2.2)	27.7 (1.5) **
Body fat (kg)	32.4 (4.7)	28.3 (5.1) *	32.5 (4.4)	30.8 (4.5)	33.7 (5.4)	27.1 (3.4) **
WC (cm)	98.2 (7.4)	95.3 (8.5) *	103.4 (8.4)	101.7 (7.4)	96.8 (6.0)	91.1 (6.7) **
Glucose ($mmol \cdot l^{-1}$)	4.52 (0.13)	4.46 (.17)	4.47 (.16)	4.41 (.10)	4.55 (.13)	4.19 (.16) **
Insulin ($\mu U \cdot ml^{-1}$)	20.8 (5.3)	19.6 (4.7)	20.4 (5.1)	16.5 (4.4) *	22.3 (5.1)	13.4 (4.8) **
HOMA-IR	4.18 (1.8)	3.89 (1.3)	4.05 (1.4)	3.23 (1.6) *	4.51 (1.2)	2.50 (1.7) **
Adiponectin ($\mu g \cdot ml^{-1}$)	2.13 (.7)	2.62 (1.1) *	1.97 (0.5)	2.73 (0.9) *	2.21 (1.1)	3.35 (1.0) **
TG ($mmol \cdot l^{-1}$)	1.33 (.11)	1.27 (.13)	1.36 (.17)	1.15 (.12) *	1.41 (.19)	1.13 (.15) **
TC ($mmol \cdot l^{-1}$)	4.42 (.36)	3.99 (.57) *	4.28 (.32)	4.12 (.44)	4.48 (.49)	3.82 (.56) **
HDL-C ($mmol \cdot l^{-1}$)	1.12 (.10)	1.10 (.13)	1.08 (.08)	1.16 (.11) *	1.04 (.09)	1.19 (.16) **
LDL-C ($mmol \cdot l^{-1}$)	2.69 (.21)	2.31 (.23) *	2.58 (.15)	2.43 (.26)	2.79 (.22)	2.11 (.23) **
TC/HDL-C	3.95 (.62)	3.63 (.41) *	3.96 (.23)	3.55 (.36) *	4.31 (.42)	3.21 (.19) **

PS: Pubertal status, BMI: body mass index, WC: waist circumference, HOMA-IR: homeostasis model assessment index for insulin resistance, TG: triglycerides, TC: total cholesterol, HDL-C: high-density lipoprotein cholesterol, LDL-C: low-density lipoprotein-cholesterol. * $p < 0.05$ and ** $p < 0.01$ before versus after program.

calculated by using the following equations (McGilvery and Goldstein, 1983): % CHO = ((R – 0.71) / 0.29) × 100. % Fat = ((1 – R) / 0.29) × 100, in which R is the respiratory quotient VCO_2/VO_2. These equations are based on the assumption that protein breakdown contributes little to energy metabolism during exercise (MacArdle et al., 1986).

We determined two parameters representative of the balance between fat and CHO utilization (Perez-Martin et al., 2001): i) crossover point of substrate oxidation expressed as a percentage of the Wmax. This point corresponds to the power at which energy from CHO derived fuels predominates over energy from lipids.

This power intensity is thus employed here as a standardized index of substrate balance during exercise. ii) maximal fat oxidation point (LIPOXmax), also expressed as a percentage of the theoretical maximal working capacity, and corresponding to the exercise intensity at which the highest rate of fat oxidation was observed. This power was used to set the intensity of the training program.

Dietary program

A balanced and personalized dietary restriction program was established by a dietician after an initial dietary assessment in order to define the total amount of calories consumed per day. In this objective, subjects of the D and D+E groups recorded the times and amounts of food and fluid intake for a week before the beginning of the program.

The dietary program was set at – 500 kcal/day below the initial dietary records. It was composed of 15% proteins, 55% carbohydrates and 30% lipids. Adolescents recorded, in a specifically designed notebook, the quantity of food and the time at which it was eaten (4 times a week). The foods were selected according to the subjects' dietary habits. PowerPoint presentations, videos, games and role-play scripts were designed for trainers to use during the educational program.

Each individual's diet was designed using a Bilnut 4 software package (SCDA Nutrisoft, Cerelles, France), a computerized database that calculates the food intake and composition from The National Institute of Statistics of Tunis 1978. The body mass was measured every week to assess the immediate effect of the nutritional modifications.

Exercise training program

The exercise training program was performed in a gymnasium and supervised by a teacher of physical education. Subjects of the E and D+E groups trained for two-months, completing four sessions of 90 min per week. The intensity of the exercise was fixed at a heart rate that corresponded to the LIPOXmax point assessed at the first visit, and it was controlled by monitoring the heart rate with a Sport-tester device (Vantage NV, Polar Electro, Kempele, Finland).

In order to enhance the adolescents' motivation, the prescribed exercises were varied and included warming-up, running, jumping and playing with a ball. However, the intensity at LIPOXmax was maintained within a narrow range despite these diverse physical activities.

Statistical analyses

All analyses were performed with SPSS for Windows. Results are expressed as mean ± standard deviation (SD). Paired Student's t-test was used for comparison within the three groups (D, E and D+E). Repeated-measure ANOVA was used to compare the responses of different groups, at different times of the test, before and after the program. The Tukey post-hoc test was used to compare means. In order to evaluate the relationship among various parameters, a Spearman correlation analysis was carried out. Intraclass correlation coefficients (ICC) were calculated to evaluate the reliability of all body composition measurements and the statistics for minimum difference (MD) needed to be considered real as calculated by Weir et al. (2005). A value of $p < 0.05$ was considered to be statistically significant.

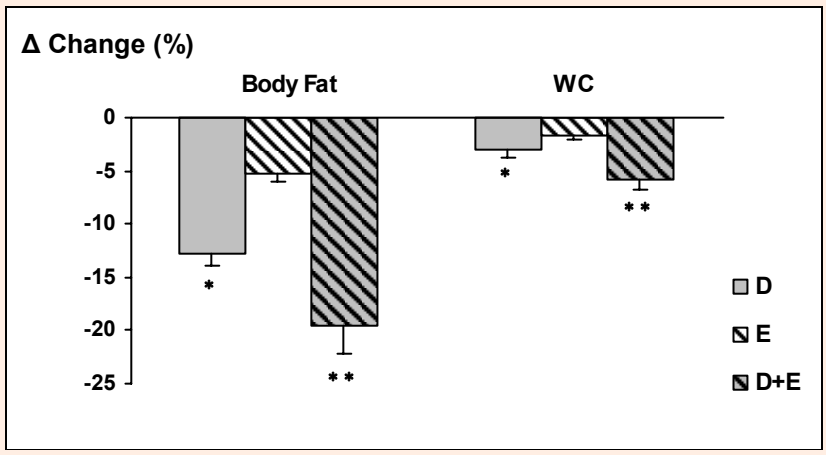

Figure 1. Percentage change in body fat mass and waist circumference (WC) from pre to post-program.
* p < 0.05 and ** p < 0.01 between pre- and post-program. D : diet, E : exercise, D+E : diet+exercise.

Results

The reliability for body composition data was as follows: body mass (ICC = 0.98), height (ICC = 0.99), BMI (ICC = 0.98), body fat mass (ICC = 0.98), waist circumference (ICC=0.98) and skinfold thickness (ICC = 0.97).

In addition, the statistics of minimum difference (MD) for all body composition measurements were 0.33 kg, 0.31 kg·m^{-2}, 0.01m, 0.18 kg, 0.26 cm and 1.1 mm respectively for body mass, BMI, height, body fat mass, waist circumference and skinfold thickness.

Table 1 summarizes the anthropometric characteristics and the metabolic parameters of the three groups at the beginning and at the end of the program. There were no significant differences among groups for age, body mass, body fat mass, BMI and pubertal stage before the study. Main baseline BMI of 30.4 ± 2.2 kg·m^{-2} indicates that, on average, these girls were obese at the beginning of the program.

After the two-month program, body mass, BMI, waist circumference and body fat mass show a significant reduction in the D group (-4.2 ± 1.1 kg, -1.9 ± 0.3 kg·m^{-2},

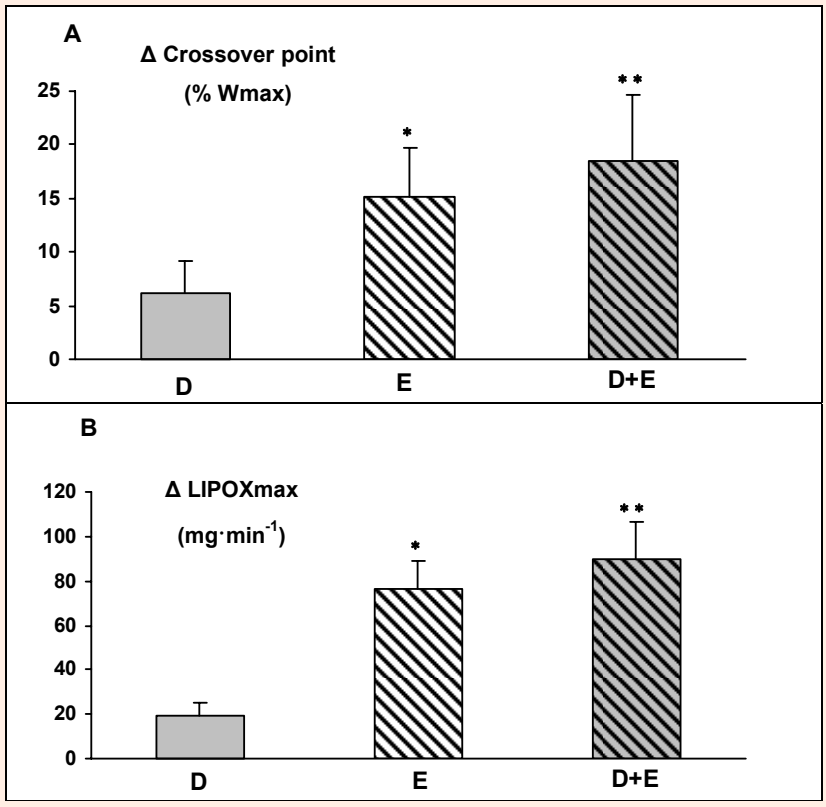

Figure 2. Delta comparison (difference pre- and post- program) in obese girls: (A) The crossover point (% Wmax). (B) Fat oxidation at LIPOXmax (mg·min^{-1}). * p < 0.05 and ** p < 0.01; difference between pre- and post- program.

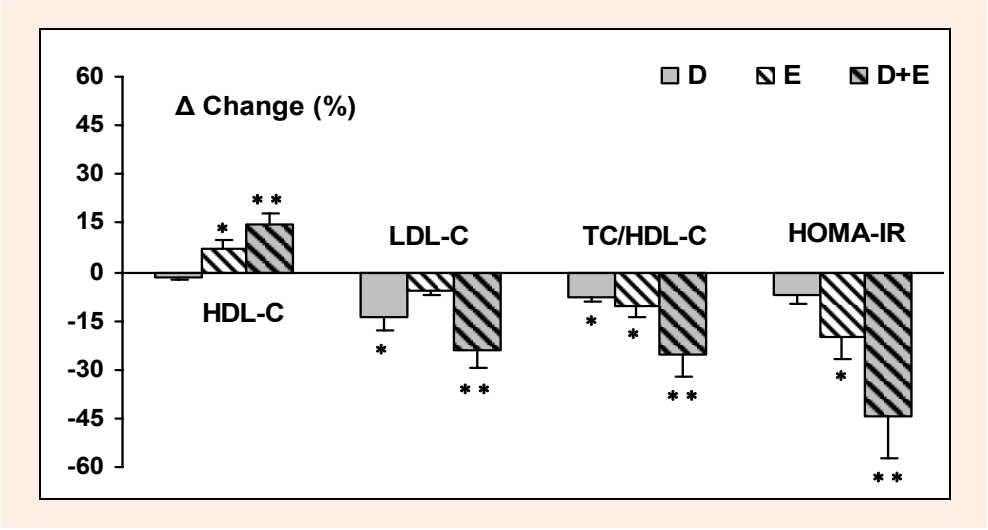

Figure 3. Percentage changes of plasma HDL-C, LDL-C, TC/HDL-C ratio and HOMA-IR in the three groups after the program. * $p < 0.05$ and ** $p < 0.01$; difference between pre- and post- program.

-2.9 ± 0.6 cm and -4.1 ± 1.4 kg; $p < 0.05$, respectively) and the D+E group (-5.8 ± 1.3 kg, -2.3 ± 0.4 kg·m^{-2}, -5.7 ± 1.6 cm and -6.6 ± 2.2 kg; $p < 0.01$, respectively). No significant change was observed for the E group. The percentage change of body fat mass and waist circumference are presented in Figure 1.

Substrate utilization was modified by the exercise training, as well as the diet/exercise programs (Figure 2). The crossover of substrate utilization increased significantly in the E and D+E groups after the two-month program (15.2 ± 4.6%; $p < 0.05$ and 18.5 ± 3.4%; $p < 0.01$ of Wmax, respectively) (Figure 2 A).

The fat oxidation rate obtained at the LIPOXmax point increased significantly in the E, and D+E groups after the program (77.4 ± 10.4 mg·min^{-1}; $p < 0.05$ and 89.7 ± 19.7 mg·min^{-1}; $P < 0.01$, respectively). In the diet group there was no significant change in the crossover and LIPOXmax points (Figure 2).

Plasma glucose and insulin concentrations and lipid profile did not differ between the three groups before the program. After the program, the fasting insulin level decreased significantly in the E (-3.9 ± .8 µU·ml^{-1}; $p < 0.05$) and D+E (-8.9 ± 3.2 µU·ml^{-1}; $p < 0.01$) groups (Table 1). Plasma insulin and glucose concentrations did not change after the two-month diet program (Table 1).

The usual index of insulin resistance changed significantly after exercise alone and exercise combined with diet. HOMA-IR decreased significantly in the E and D+E groups (-20.2 ± 6.7%; $p < 0.05$, and -44.6 ± 12.4%; $p < 0.01$, respectively). No change was found in the D group (Figure 3).

The lipid profile improved significantly after the two-month diet combined with exercise program. Diet/exercise increased HDL-C by 14.4%, and decreased LDL-C and TC/HDL-C by 24.4% and 25.5% respectively (Figure 3).

Adiponectin levels increased significantly in the D ($p < 0.05$), E ($p < 0.05$) and D+E groups ($p < 0.01$) (Figure 4).

In the subjects of the D+E group, adiponectin levels exhibited a significant negative correlation with body mass ($r = -0.41$; $p < 0.01$) and HOMA-IR ($r = -0.59$; $p < 0.01$). In addition, the TC/HDL-C ratio was positively correlated to HOMA-IR ($r = 0.46$; $p < 0.01$), body mass ($r = 0.35$; $p < 0.01$) and negatively correlated to LIPOXmax ($r = -0.52$; $p < 0.01$).

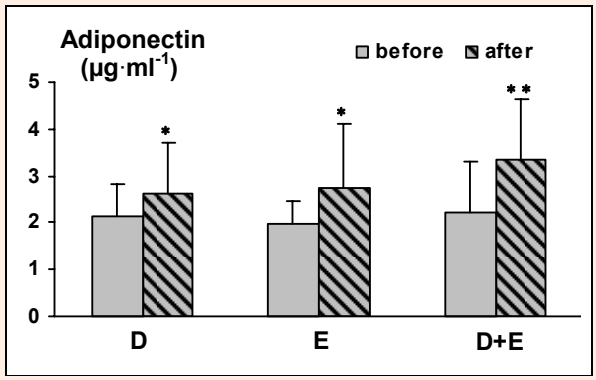

Figure 4. Adiponectin levels (before and after programs) in obese girls. $p < 0.05$ and ** $p < 0.01$ compared with the concentrations before the program.

In the E group, adiponectin levels were negatively correlated to HOMA-IR ($r = -0.33$; $p < 0.05$) and the TC/HDL-C ratio was positively correlated to HOMA-IR ($r = 0.28$; $p < 0.05$) and negatively correlated to LIPOXmax ($r = -0.30$; $p < 0.05$).

In the D group, significant correlations were observed between adiponectin levels and body mass ($r = -0.31$; $p < 0.05$) and between the TC/HDL-C ratio and body mass ($r = 0.26$; $p < 0.05$).

Discussion

This study shows that the two-month program of diet restriction combined with exercise training at a working intensity corresponding to the LIPOXmax leads to greater improvements in obese girls than diet or exercise alone.

Combined diet and exercise induce a substantial body fat mass loss (19.6% of initial body fat mass), a decrease in waist circumference and an increased ability

to oxidize lipids during exercise (LIPOXmax increased from 0.13 to 0.22 $g \cdot min^{-1}$). The levels of plasma adiponectin and HDL-C were higher and those of LDL-C, and the TC/HDL-C ratio were lower at the end of the two-month diet/exercise program.

In this study, we were interested in providing relative reliability estimates of body composition measurements (Weir et al., 2005), and we found excellent test-retest reliability for all the tests examined. Skinfold thickness and circumference measurements showed excellent test-retest reliability, with the ICC estimates exceeding 0.95. Our study has produced excellent reliability of each participant during anthropometry testing. Moreover, all body composition measurements reached the minimum difference needed to be considered real.

Our investigations are based on the technique of indirect calorimetry, which appears to be valid for measurements of substrate oxidation during sub-maximal steady-state exercise bouts. Such measurements have shown that obese people oxidize fewer lipids during exercise than lean matched controls (Perez-Martin et al., 2001) and that low intensity exercise training markedly reverses this defect in both adult and adolescent obese subjects (Brandou et al., 2003; Dumortier et al., 2003).

In the obese population, low-intensity exercise training may be preferable to high-intensity because of the lower risk of musculoskeletal injuries and better adherence to the training schedule (Bouchard et al., 1993). In addition, 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 body mass loss in the post-diet period. This effect may be mediated by maintenance of sympathetic nervous system sensitivity, which tends to be reduced after body mass loss alone (Van Aggel-Leijssen et al., 2001).

Obesity is considered as a major independent risk factor for cardiovascular disease (Scaglione et al., 2004). The National Cholesterol Education Program (NCEP) has demonstrated that diet alone reduces serum TC and LDL-C in normal people. However, a drop in the HDL-C levels is also often reported (Hellenius et al., 1997; Yancy et al., 2004). The results of the present study demonstrate that the diet restriction alone induced an improvement in LDL-C but without any effect on HDL-C. Reduced HDL-C concentrations are an established risk factor for coronary heart disease, and it has been estimated that a 1 $mg.dl^{-1}$ increase in HDL-C reduces heart disease risk by 4% (Gordon et al., 1986).

HDL-C levels are higher in well-trained endurance athletes (Thompson et al., 1991) and increase in sedentary subjects after exercise training (Thompson et al., 1997).

The present study shows that changes in HDL-C (14.4%) and LDL-C (-24.4%) in the diet/exercise group are considerably greater than those of the diet or exercise group alone suggesting a decrease in heart disease risk in the subjects of the D+E group. Accordingly, Wood et al. (1991) have shown that a one year combined program was more effective in improving the lipoprotein profile than exercise or diet alone and was associated with a rise in HDL-C and a drop in LDL-C.

In addition, it has been evidenced that the ratio of TC/HDL-C is a better predictor of cardiovascular disease (CVD) risk reduction than HDL-C, LDL-C, or the TC value alone (Natarajan et al., 2003). Our results show that exercise does not influence TC or LDL-C. However, exercise favourably influences HDL-C (7.4% and 14.4% in the E and D+E groups, respectively) and the TC/HDL-C ratio (-10.6% and -25.5% in the E and D+E groups, respectively). These results agree with the findings of Varady et al. (2007) who showed that eight weeks of endurance exercise three times a week increased HDL-C by approximately 10%. It appears that exercise only, although not affecting changes in TC and LDL-C, has a beneficial effect on the TC/HDL-C ratio. Moreover, the current study demonstrates that combined diet with training improves the lipid profile (TC, LDL-C, HDL-C and TC/HDL-C) in obese girls.

In the present study, we noted a small decline in fasting glucose (7.9%) accompanied by a much larger decrease in insulin (39.9%) after the diet/exercise program. Kang et al. (2002) reported an improvement in fasting insulin in overweight children after a combined exercise/diet program. In the same way, three days/week of aerobic exercise improved HOMA-IR in overweight subjects (Balagopal et al., 2005).

In this study, HOMA-IR increased by 20.2% and 44.6% respectively in girls undertaking exercise and diet/exercise programs. The decreased risk of cardiovascular disease is associated with an increase in fat oxidation in the subjects of the E and D+E groups. Indeed, the TC/HDL-C ratio is significantly negatively correlated to LIPOXmax after a two-month program of exercise only ($r = -0.30$) or combined with diet ($r = -0.52$).

This study confirms that physical activity associated with reduced food intake improves insulin sensitivity more than exercise alone or controlled diet alone. An immediate effect of exercise is an increase in muscle glucose transporters, which secondarily improves insulin-mediated glucose disposal. Presumably, increased oxidation of fatty acids reduces lipid overload, which also increases insulin sensitivity (Goodyear and Kahn, 1998).

The marked improvement observed in serum TG is primarily due to the combination of the diet restriction and the exercise training at LIPOXmax. Because the consumption of processed carbohydrates is generally higher in obese children (St-Onge et al., 2003), the transition to a diet largely devoid of refined carbohydrates, along with a daily exercise regimen (Oscai et al., 1972) facilitated reduction of TG. In addition, the decrease in serum insulin may also play a role in reducing TG.

Obesity is the final consequence of a chronic positive energy balance, regulated by a complex network between endocrine tissue and the central nervous system (Cummings and Schwartz, 2003). Fat tissue is increasingly viewed as an active endocrine organ with a high metabolic activity. Adipocytes produce and secrete several proteins that act as real hormones, responsible for the regulation of energy intake and expenditure (Mora and Pessin, 2002).

Adiponectin may act as anti-atherosclerotic factor not only through direct effects on vascular endothelial cells, but also through improving insulin resistance and

lipid metabolism (Yamamoto et al., 2002). It has been reported that adiponectin levels lower in young and adolescent subjects who are obese (Zou et al., 2005). Seven months of moderate intensity exercise training increased adiponectin levels by 42.8% in obese young women (Kondo et al., 2006), and in the same way, a significant body mass loss was associated with a significant increase in adiponectin levels in 16 obese children after a one-year Obeldicks intervention program (Reinehr et al., 2004). Our present study demonstrated that circulatory adiponectin levels are significantly increased in the obese girls after the two-month intervention program; this increase is more pronounced in the D+E (51.6%) than the E (38.5%) or D (23%) groups.

Matsubara et al. (2002) have reported significant correlations between adiponectin levels and insulin resistance. Accordingly, a significant correlation was observed in our subjects between adiponectin levels and HOMA-IR after the two-month exercise alone or exercise combined with diet control, suggesting that adiponectin could be considered as an important determinant of insulin resistance.

Adiponectin is the first known adipocytokine that is down-regulated in obesity. The mechanism of this negative regulation remains unclear, because adiponectin is secreted exclusively by fat cells (Beltowski, 2003). Adiponectin secretion decreased when visceral adipose tissue was isolated and cultured in vitro (Beltowski, 2003). This effect was reduced by decreasing the amount of tissue cultured per dish. In addition, the effect was prevented by inhibitors of transcription and translation. Probably, the increasing mass of white adipose tissue in obesity reduces adiponectin protein synthesis by a feedback inhibition (Diez and Igleasis, 2003).

Furthermore, adiponectin secretion in vitro is lower in visceral as opposed to peripheral adipocytes in children (Sabin et al., 2003), pointing to an influence of body fat distribution. Because adiponectin is stimulated by insulin and inhibited by TNF-α, insulin resistance and enhanced TNF-α expression may contribute to hypoadiponectinemia (Beltowski, 2003). Glucocorticoids are also reported to inhibit adiponectin gene expression and secretion (Diez and Igleasis, 2003), suggesting that decreased adiponectin production could play a role in glucocorticoid-induced insulin resistance.

Conclusion

The results from the present study complement the previous findings by showing that the addition of a diet restriction to endurance exercise training significantly improves body composition, HDL-C, TC/HDL-C ratio, and insulin resistance. Diet combined with an exercise program is typically recommended for decreasing body mass, insulin resistance and LDL-C and increasing fat oxidation, HDL-C and adiponectin levels in obese girls.

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Key points

- Diet combined with exercise training improved body composition, adiponectin levels and metabolic parameters in obese girls.
- Diet only decreases body mass and LDL-C without improving fat oxidation and HDL- C.
- Individualized exercise training at LIPOXmax point improved the HDL-C and the circulatory adiponectin levels with any change of LDL-C and body composition.

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