A Submaximal Exercise Patterns Intervention on the Plasmatic Leptin Level and Insulin Resistance for Overweight / Obesity Boys Adolescences

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Received: 16 June 2020

Accepted: 20 November 2020

Published in December 2020

Abstract

Objective: Obesity is a medical problem that increases the risk of health problems like diabetes. Hormones secreting from fat tissue, Leptin, are correlated with body mass index. Leptin reduces the person's appetite by acting on specific centers of their brain to reduce their urge to eat. And insulin, a hormone produced by the pancreas, is essential for regulating carbohydrates and the metabolism of fat. A positive relationship between leptin levels and insulin resistance in children showing in this study is to determine the effect of continues-endurance and interval-endurance on leptin serum levels and insulin resistance in over-weighted youths.

Materials and Methods: This study is a semi-experimental design. Thirty boys were voluntarily selected as a matched two experimental groups and a control. Physical profiles included aged 16-19, BMI; 37.75 (±4.46) KG/M^2, base functional capacity: 32.8 (±3.6) ml/kg/min which dividing by three equal groups: the submaximal training interval, a continuum and interval and control group performed the submaximal running program for six weeks on 3 D/W. The selected parameters were evaluated for an intervention pre and post-conditions.

Results: weight and body mass index variables were decreased by about 2.5-3% after exercise intervention. Plasmatic leptin levels were reduced by about 45% in the continuum and interval control groups. HOMA-IR & QUICKI indexes altered in the EG (8.9 %, 7.8%) (*P*-value < 0.05).

Conclusion: Presumably, 2 exercise program patterns lower than lactate threshold could change body composition profile, basal leptin level, and blood glucose /insulin concentrations, which probably induced changes in resistance to insulin.

Keywords: Leptin hormone, Insulin hormone, Submaximal exercises, Resistance, Obesity

Introduction

Obesity is considered as a significant health problem worldwide recently. Obesity and the overweight phenomenon is associated with several health problems that are often summarized together as metabolic syndrome and involve the development of insulin resistance, type 2 diabetes, cardiovascular disease, and fatty liver

[DOI: 10.18502/ijdo.v12i4.5182]

disease. Obesity is characterized by increased adipose tissue mass (1). Adipose tissue, as an active endocrine organ, is involved in obesity-related disorders by secreting cytokines that influence energy homeostasis (2). Leptin, the anti-inflammatory adipocytokine secreted from white adipose tissue, is effective on energy homeostasis and body weight control by affecting the hypothalamus and decreasing appetite and by increasing sympathetic nervous activity and lipolysis (4). The exact mechanism in the control of leptin secretion is not yet fully known; however, given the role of Leptin in regulating energy expenditure, the use of two therapeutic strategies of increasing physical activity and the caloric restriction would affect leptin levels by adjusting the level of energy intake and changing the amount of energy (5). Researchers have looked into the changes of Leptin resulted from endurance exercise, and in some cases, reduction (3, 6, 4, 7, 8, 15, 16, 17, 10) of Leptin has been observed. Some researchers relate changes in plasma leptin to changes in adipose tissues (18), yet some others consider a reduction in plasma leptin concentration or expression independent of changes in fat mass. It is, therefore, possible that, except for aerobic exercise, other factors contribute to the reduction of plasma leptin concentrations (9). In animal or human studies, when the level of Leptin is at its lowest natural limit, it acts as insulin sensors. It may lead to increased insulin resistance when increasing the base level of Leptin. Higher serum leptin levels in obese adolescents than their normal-weight counterparts indicate that insulin resistance to leptin levels decreases from early childhood (10). The Elsedfy study in 2014 showed that 54 % of children with a high body mass index have a more elevated insulin resistance index than the cutoff range (11). Therefore, the prevention and control of childhood and adolescent childhood are far more effective than childhood obesity prevention (12). Ghobadi et al (2013) also found the effect of two types of aerobic exercise significant decrease in serum leptin levels in the children's aerobic group (19).

Since insulin modulates the synthesis and secretion of leptin and insulin levels, some researchers have suggested insulin as the primary candidate for such control (20). Mechanisms responsible for such management are unknown to date. A limited number of studies have examined the concurrent changes of blood leptin and hormones that are effective in response to endurance exercise. However, Bouassida and colleagues demonstrated performance in the short time protocol cycling submaximal (45 Minutes) and long-term (85 Minutes), despite the reduction in plasma leptin and insulin plasma levels, has no significant effect (21). The result of studies protocol is due to differences in design features. Weight loss and doing exercise are common clinical interventions for insulin resistance treatment (13, 14). Moderate to vigorous aerobic exercise improves the inactivation of inactive skeletal muscle glucose transducers and insulin function (22). Since skeletal muscle is a significant site of glucose uptake following insulin stimulation, impaired insulin sensitivity, a decrease in the amount of access to GLUT4 protein, which plays a crucial role in whole-body insulin sensitivity and glucose tolerance, can lead to decreased uptake glucose, and subsequently, increased systemic blood sugar (23)

According to research shreds of evidence, exercise can significantly affect serum leptin levels and insulin resistance. However, few studies have examined the effect of exercise on leptin in adolescents. Also, the effect of exercise patterns: (endurance - continuous) and (endurance - periodic) have not been studied simultaneously. Therefore, the aim of the present study was to investigate the effect of these two types of exercise methods simultaneously on serum leptin levels and insulin resistance in overweight adolescent boys.

[DOI: 10.18502/ijdo.v12i4.5182]

Materials and Methods

This study is semi-experimental study. The subjects are selected from a high school by voluntary overweight and obese students. Twenty seven people were selected as a sample and randomly divided into three groups of 9 people. Participants completed questionnaires, demographic checklist, Physical Activity Readiness Questionnaire (PAR-Q), health status, and written consent. Inclusion criteria were: boys aged 16 to 19 years, weight range 65 to 110 kg, body mass index of 85 to 97% of CDC chart (Z SCORE≥1.04-1.64). The exclusion criteria were: smoking, metabolic syndrome, regular exercise, restriction diet programs. The intervention groups participated in 6 weeks of an intermittent and continuous-interval exercise program for three weeks in three sessions per week and 15-25 minutes each at the track. However, the submaximal program was a 15-minute continuum, which was added five minutes every week to the 65-60 HR-max exercise intensity. The alternate subroutine program was 3-minute running intervals with 70-75% HRmax intensity with 3-minute active rest (walking and jogging) at 30- 45% HRmax. The combined exercise group did one continuous exercise session and one intermittent exercise session.

In the first sample, ten ccs of fasting blood were obtained at 9 am from the brachial vein of the three study groups at the Medical Diagnostic Laboratory to measure plasma levels of Leptin, glucose, and insulin by standard methods. The second blood sampling was repeated after 72 hours of intervention.

To measure fasting plasma insulin and glucose levels, quantitative luminescence was measured using the German-made LIAISON device (24), and serum leptin levels were measured using the Mediagnost Elisa kit Refe07 made in Germany. The insulin resistance index was estimated in terms of baseline glucose (mg/dl) and fasting insulin (μ IU / ml) (19) HOMA-IR indices, QUICKI logarithmic relationship, FGIR (fasting glucose to insulin ratio) were also used to estimate insulin resistance indices (25).

Statistical analysis

Descriptive statistics were used to determine the mean and standard deviation (SD) of each variable compare the mean ± SD in the pre- and post-test, paired sample T-test inferential statistics, and one-way ANOVA with default distribution for the difference between groups. Data were analyzed using a Kolmogorov-Smirnov test (*P*-value= 0.632) and variance homogeneity Levenes test (*P*-value= 0.098). Tukey post hoc test was used at a significantly less than 5%. All statistical operations and graphs were performed by SPSS 18 software.

Ethical considerations

The percentage of changes in each of the dependent variables was also evaluated in terms of the difference between biochemical blood variables in the post- and pre-intervention conditions. This study was approved with the Ethical Code: IR.BASU.REC.1398.011

Results

Comparing the mean biochemical variables of boys in the two interventions and control groups were not different (Table 1).

Analysis of variance- one way between the dependent variables after exercise intervention showed a significant difference in each of the serum levels of Leptin, glucose, insulin, and selected indices of insulin resistance in both the interval and concurrent groups (*P*-value< 0.05). However, the comparison between the FGIR and HOMA-B groups showed no significant difference (*P*-value> 0.05) (Table 2).

Table 3 shows that no significant difference was found in the mean of the dependent variables in the control group. But fasting blood glucose in the interval exercise group decreased 50.16% from 106 to 75.89 mg/dl (*P*-value= 0.004) and in the combination exercise group decreased 41.13% to 75.98 to 5.86

[DOI: 10.18502/ijdo.v12i4.5182]

Table 1. Comparison of variables measured before exercise intervention (One-Way ANOVA)

Variable	control group Mean (±SD)	submaximal interval training Mean (±SD)	Continoum interval training Mean (±SD)	*P*-value
Number in the group	9	9	9	
fasting glucose (mg/dl)	99.37 (±3.07)	106 (±4.81)	98.75 (±3.46)	0.354
fasting insulin (µIU/ml)	6.40 (±0.27)	6.65 (±032)	6.27 (±0.5)	0.78
leptin (Ng/dl)	16.38 (±3.08)	13.08 (±1.54)	15.42 (±1.27)	0.727
HMOA-IR	1.56 (± 0.10)	1.73 (± 0.14)	1.56 (± 0.17)	0.61
QUICKI	0.36 (±0.002)	0.35 (±0.003)	0.35 (±0.003)	0.602
FGIR	15.58 (±0.48)	16.07 (±0.72)	16.13 (±0.81)	0.829
HOMA-B	67.33 (±5.3)	57.77 (±4.54)	63.12 (±3.38)	0.342

Table 2. Comparisons of mean fasting serum levels of leptin, glucose, insulin and insulin resistance indices of the three groups after exercise intervention (Tukey post hoc test)

Groups		leptin	Fasting glucose (mg/dl)	Fasting insulin (µIU/ml)	HOMA-IR	QUICKI	FGIR	HOMA-B
Control group	Continues interval training	*0.039	*0.008	*0.047	*0.001	*0.002		
	Submaximal interval training	0.339	*0.045	*0.047	*0.004	*0.004	0.280	0.412
Continues interval training	Submaximal interval training	0.468	0.702	1.00	0.891	0.957		

* *P*-value< 0.05

Table 3. Intergroup analysis of variance between serum levels of Leptin, glucose, insulin, and selected indices of insulin resistance before and after exercise (T-test).

Variable	Control group Mean (±SD)			Submaximal interval training Mean (±SD)			Continuous interval training Mean (±SD)		
	Baseline	Post test	*P*-value	Baseline	Post test	*P*-value	Baseline	Post test	*P*-value
Weight (KG)	96.38 (±7)	96.28 (±7.79)	0.861	80 (±10)	78 (±9.8)	0.005*	81.4 (±7.79)	79.5 (±8.31)	0.019*
BMI KG/M^2	29.98 (±1.91)	29.86 (±2.06)	0.574	26.65 (±2.14)	25.86 (±2.15)	0.002*	26.61 (±1.25)	26.23 (±1.33)	0.021*
leptin (Ng/dl)	16.83 (±3.08)	15.80 (±2.76)	0.369	13.08 (±1.54)	11.76 (±1.54)	0.269	15.42 (±1.27)	8.42 (±1.27)	0.011*
fasting glucose (mg/dl)	99.37 (±3.07)	95.12 (±1.12)	0.168	106 (±4.81)	88.5 (±1.62)	0.004*	98.75 (±3.46)	86.5 (±2.06)	0.047*
fasting insulin (µIU/ml)	6.40 (±0.27)	5.96 (±0.22)	0.102	6.65 (±0.32)	5.00 (±0.33)	0.01*	6.27 (±0.5)	5.00 (±0.22)	0.017*
HOMA-IR	1.56 (±0.10)	1.40 (±0.59)	0.61	1.73 (±0.14)	1.10 (±0.18)	0.002*	1.56 (±0.17)	1.06 (±0.046)	0.020*
QUICKI	0.36 (±0.2)	0.3562 (±0.004)	0.095	0.3525 (±0.003)	0.38 (±0.004)	0.001*	0.35 (±0.003)	0.381 (±0.6)	0.013*
FGIR	15.58 (±0.48)	16.04 (±0.57)	0.530	16.07 (±0.72)	18.69 (±1.86)	0.065	16.13 (±0.81)	17.55 (±0.87)	0.118
HOMA-B	67.33 (±5.3)	69.14 (±3.09)	0.766	57.77 (±4.54)	71.81 (±8.19)	0.112	63.12 (±3.38)	82.34 (±9.00)	0.096

* *P*-value <0.05

mmol equivalent respectively. Grams occurred in dL (*P*-value= 0.047).

In the two intervention groups, fasting insulin concentration decreased significantly (*P*-value= 0.01). Serum leptin level in the interval group decreased by 10.09% (*P*-value= 0.269). But in the combination training group, a significant decrease was observed in 39.45%

[DOI: 10.18502/ijdo.v12i4.5182]

(*P*-value= 0.011). The decrease in HOMA-IR index in both groups was also significant (*P*-value= 0.002). A similar pattern was observed in the insulin resistance index (*P*-value= 0.01). In contrast, the mean increase in HOMA-B glucose to insulin ratio in the intervention groups was not significant (*P*-value> 0.05).

Discussion

The results of this study showed that serum leptin levels in the continuous-interval training group significantly decreased. Science has suggested a link between leptin changes and negative energy balance, sympathetic activity, and some metabolites. Among the regulators of Leptin, the release is exercise-induced stress, altered fuel displacement, systemic hormone concentrations, and the effect of energy expenditure (4). Bernard Gutin et al. (2014) Changes in plasma leptin concentration in obese children aged 7-11 years over four months of intense aerobic exercise 5 times per week and 40 minutes of endurance exercise with an average heart rate of 150 and then a four-month non-exercise period. Their results showed that this exercise program significantly reduced serum leptin levels but returned to baseline after an interval of inactivity (18).

In the study of Zelia Bori et al. (2015), the effect of 41 minutes of aerobic training on a treadmill with 70-50% HRmax significantly reduced serum leptin in obese girls against 4 min of intermittent exercise, 33 min of treadmill activity in Fig. 4 × 3 minutes of running with 70 to 95% HRmax range (27).

Also, in the present study, submaximal and combined exercise significantly decreased the levels of glucose, fasting blood insulin, and insulin resistance indices (HOMA-IR and QUICKI). Sang et al. 2016 showed that aerobic activity of Korean children running 10 to 13 years within 12 weeks, the first month with 60% VO2max, the second month 65% VO2max, and the third month with 70% VO2max were associated with decreases in body mass index and insulin resistance (28). Lee's 2012 study also found that three months of aerobic training of obese adolescents without calorie restriction, three sessions per week for 60 minutes at 60-75% VO2max intensity, significantly improved abdominal fat despite significant abdominal fat and body mass index. Insulin sensitivity was not found to be consistent with the results of our study (29). Also, Ashokan, in 2015, examined the effect of 8-week aerobic exercise at 50-80% of maximal heart rate on insulin resistance index and overweight boys' body weight, which significantly decreased weight and BMI, but changes in insulin and HOMA-IR concentrations. It is inconsistent with the present study results (30), which may be searched for factors such as lifestyle intervention, daily nutrition, and a basic level of subjects' practical capacity. In a systematic review of the role of obesity in children and HOMA in 2019, Stephen noted that a dynamic lifestyle of running 30 minutes of aerobic exercise daily for 3 to 5 weekly sessions with sub-optimal intensity could improve insulin sensitivity. Also, the High-Intensity Intermittent Exercise Model (HIIT) can cause increased changes in children's insulin sensitivity (31).

Three possible mechanisms have been proposed to improve baseline blood glucose control following exercise, insulin responsiveness through insulin receptor phosphorylation and consequent activity (PIK3), as well as increased serine-protein phosphorylation. Kinase (AKT) stimulates the transfer of GLUT4 to the cell membrane. Furthermore, in the absence of insulin secretion, GLUT4 translocation following skeletal muscle contraction has been reported (32). Thus, biochemical adaptation such as increases in GLUT-4 glucose transporter activity, GLUT-4 glucose transporter content, carrier Glucose-dependent, insulin-dependent, content and activity levels of metabolic enzymes affect glucose, lipid metabolism, lead to the acceleration of liposomes and the reduction of glycogen pathways (34). Structural adaptations include increases in capillary density, blood flow, and blood

[DOI: 10.18502/ijdo.v12i4.5182]

distribution of skeletal muscle capillaries (34). Finally, other transformations such as decreases in abdominal obesity, systemic inflammation, hypertension, improvement of the fat profile, and increased aerobic fitness have been reported (33).

These results could reveal the role of submaxillary aerobic exercise intervention in weight loss and basal levels of plasma leptin hormone and insulin resistance index as one of the symptoms of diabetes in obese children. Clinical evaluation of metabolic outcomes adolescent obesity requires independent studies on cardio-metabolic factors and inflammatory markers and the effect of different modes of activity on it**.** These results can emphasize the effect of physical activity on reducing and preventing the effects of obesity in adolescents.

Conclusions

The results of this study indicated a continuous-interval group with significant reductions in blood glucose, insulin resistance, and plasma leptin. Although changes in leptin hormone were not substantial in the interval group, a significant decrease in serum leptin levels was observed following a reduction in blood glucose and insulin levels and decreased insulin resistance. Studies show that basal levels of leptin concentration play a prominent role in inducing glucose homeostasis. However, while serum leptin levels are in the lowest physiological range, they trigger increased insulin sensitivity. They may increase insulin resistance, depending on elevated basal plasma leptin levels in obese individuals (34). Of course, leptin receptors present in pancreatic beta cells play an essential role in insulin secretion (35). These results may underlie aerobic exercise intervention in weight loss and basal plasma leptin hormone levels in obese children. Clinical evaluation of adolescent metabolic outcomes on cardio-metabolic factors in obese children requires independent studies.

Acknowledgments

This is a grateful acknowledgment from all the people who have contributed to this thesis.

Funding

This article is based on the master's thesis approved by Bu - Ali University of Hamedan.

Conflict of Interest

All authors declare there were no conflicts of interest.

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[DOI: 10.18502/ijdo.v12i4.5182]

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[DOI: 10.18502/ijdo.v12i4.5182]