



The Response of Plasma Leptin and Some Selected Hormones to 24-weeks Aerobic Exercise in Inactive Obese Women

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Abstract:

Background: Leptin resistance is one of the influential factors on hyperinsulinism and finally on lack of the glucose tolerance in overweight and its related diseases. Thus the goal of this research is to study the response of Leptin Plasma, 17-beta estradiol serum and insulin to 24-weeks aerobic exercise in inactive obese women.

Method: The research was of semi-experimental kind. 15 inactive obese women were chosen randomly and were categorized in two different groups; i.e. aerobic exercise and control. The exercise process included aerobic exercises for 6 months (three 60-minute sessions per week). We used repeated measure for variation the time \times group. The Independent sample T test was applied to compare the average amounts between groups.

Result: The results represent that the influence of time has not been identical on amount of leptin serum, insulin, the body mass index and fat amount, and a 24-weeks aerobic exercise caused a significant decrease in this field ($P < 0.05$). After 24-weeks aerobic exercises, duration of time in two different control and aerobic exercise groups showed no important effect on the level of 17-beta estradiol serum ($P = 0.393$).

Conclusion Results showed that aerobic exercises reduce the body mass index and consequently reduce leptin and insulin in inactive obese women via increasing the capacity of muscle in using the fat, which can be considered as a preventing factor of obesity and its related diseases, such as obesity and cardiovascular diseases.

Keywords: Leptin, 17-Beta Estradiol, Insulin, Aerobic Exercise, Obese, Inactive Women

Introduction

Obesity is in company with increasing the leptin blood level. According to some scientific sources, leptin does not hold the insulin secretion (Ahrén et al., 1999). However other findings show that leptin resistance is an influential factor on hyperinsulinism and finally on lack of the glucose tolerance in overweight and its related diseases (Morioka et al., 2007). Some studies suggest that leptin sensors exist in pancreas beta cells and play an important role in insulin secretion (Kieffer et al., 1996). Hormonal changes that can be seen in obesity, especially in the upper part of the body, lead to cortisone and insulin increasing and hormone serum level decreasing. Whether such changes are consequence of overweighting or not, and whether it can be considered as primarily reasons of obesity and the existing relation between that and kind of fat distribution in the body is not clear (Haffner, 2000). Plus, researches have shown that leptin products are controlled in vivo and vitro conditions by different hormones and chemical materials. Mentioned studies have suggested that leptin products are controlled by positive insulin regulation, glucocorticoid, NPY and negative regulation of cAMP (DeVos et al., 1995; Zhang et al., 1996).

Ovarian hormones may affect the body via several potential mechanisms. It is claimed that estradiol controls the lipase lipoprotein performance in fat tissue. Contradictory results were reported

about the effects of estradiol and follicle stimulant hormone (FSH) in producing the leptin by fat cells (Bouvattier et al., 1998; Riad-Gabriel et al., 1998; Messinis et al., 1999; Rechberger et al., 1999). Serum estradiol density changes eminently during the women's life. leptin serum level may change parallel to the estradiol level as well (Yamada et al., 2003).

It is reported that 1) Estradiol does not regulate the leptin secretion or its effects on a fat mass directly. 2) Leptin does not regulate the estradiol secretion or its effects on a fat mass directly. Nevertheless, leptin and estradiol may affect the fat consumption directly and indirectly (Pelleymounter et al., 1999). Insulin secretion directly to white fat and causes a pure catabolic response by the brain, exactly the same as leptin. Leptin and insulin stimulate the arch of hypothalamus (Zolandz et al., 2005); they are the regulator of leptin gene (ob) secretion as well (Kolaczynski et al., 1996).

A mechanism that reduces the leptin amount, may affect the nervous system due to physical activities (Mantzoros et al., 1996; Considine et al., 1996). Exercises affect the fat and carbohydrate metabolism, in other words, leptin is connected to health condition, glucose metabolism and free fat acids (Bouassida et al., 2010). Recently, many contradictory effects of the exercise on the leptin density were found. But some researchers observed no changes in leptin (Jurimae et al., 2005; Pop et al., 2010). Vatansever et al (2011) studied the effect of a 120-minute exercise on jogging with maximum 50 to 70 percent of oxygen consumption in 10 healthy men. Their results showed that the exercise does not affect the leptin serum level.

Sari et al (2007) showed that no changes can be observed in leptin serum level in 23 obese women after a 45-minute walking sessions at 60-80% of maximum heart rate. Some studies have reported that exercises reduce the leptin, depending on the time and energy consumption (Jurimae and Jurimae, 2005; Pop et al., 2010). For example in a recent study, a 30-minute exercise with maximum 80% of heart rate leads to a significantly leptin reduction (Kraemer et al., 1999). Although some other studies have represented that a 60-minute exercise by treadmill with 50% of Vo_{2max} in healthy men leads to no changes in leptin level (Torjman et al., 1999).

Nevertheless more researches are needed to clarify the quality of determining the metabolic materials and hormonal effects in leptin and to find a reason that why the leptin density has decreases in some cases and has not changed or has increased in some others. In the history of accomplished studies, the effects of short term or 12-weeks exercises on the level of mentioned factors were evaluated; and according to researchers, most of such data are not available completely. Thus the goal of our study is to evaluate the response of leptin plasma, 17-beta estradiol serum and insulin to 24-weeks aerobic exercise in inactive obese women.

Materials and Method

The research was of semi-experimental kind. 15 inactive overweight women aging 37 to 47 and (BMI>30) were chosen voluntarily and according to some criteria (such as being healthy on the basis of health questionnaire, no cigarette and drug usage, lack of menopause and inactivity on the basis of questionnaire) via random sampling and were categorized into two different groups: i.e. aerobic exercise (8) and control (7). Participants filled a written letter of satisfaction for attending in the research. Participants were allowed to enter the research after taking blood and heart examinations and measuring the blood pressure by an expert. The BMI weight and fat amount were determined by a body analyze machine (Inbody720, made by Biospace, Korea). Blood samples were collected after 12-14 hours of being fasting to evaluate the biochemical changes; they were collected before starting the exercises and 48 hours after the last session of a 6-month exercise. Blood sampling was taken between 7 a.m. to 8 a.m. in the laboratory via the left hand vein as much as 7 to 8 milliliters, while the participants were sitting and resting. Obtained serums were analyzed for measuring the leptin, estradiol and insulin. Hormones were measured via Elisa method by special kits.

Exercise Process: The exercise process included aerobic exercises for 6 months, three times a week as long as 60 minutes. Walking, jogging and aerobic movements with a same rhythm and 55 to 65 percent of heart beat intensity. The exercise has been controlled by a cardiograph (Polar, Made in Poland). The participants in the control group did no exercises during the research and followed their routine lifestyle.

Statistical analysis

All the data were analyzed using SPSS version 11.5. The average and standard deviation of data were calculated after checking the data distribution normalcy using Kolmogorov-Smirnov test. We used repeated measure for the effect of time group. The Independent sample T test was applied to

compare the average amount between groups (aerobic exercise and control). P values of less than 0.05 were considered to be statistically significant.

Result

Results show that the effect of time on two different groups is not the same and a 24-weeks aerobic exercise causes a significant decreasing in leptin level in aerobic exercise group (P= 0.036), although effect of time×groups showed no significant difference in 17-beta estradiol serum after a 24-weeks aerobic exercise either in aerobic exercise or in control group (P=0.393). Also it was revealed that time does not affect on the level of serum insulin in two aerobic exercise and control groups equally, and the 24-weeks aerobic exercise has reduced the serum insulin meaningfully in aerobic exercise group (P = 0.001). Time does not affect the body mass and fat amount equally in aerobic exercise and control groups as well; it shows a significant decreasing in aerobic exercise group after the 24-weeks exercise (P = 0.045).

Table 1: effect of Group * Time on leptin, 17-beta serum estradiol, insulin, body mass index, body fat percent in inactive obese women in two aerobic exercise and control groups

Group * Time	Assumed Freedom Level	F	P value
Leptin	1	5/143	0/036*
17-Beta Estradiol (pg/ml)	1	0/114	0/393
Insulin(Uu/ml)	1	8/19	0/001*
BMI (kg/m ²)	1	10/271	0/045*
Body Fat Percent	1	5/006	0/000*

Significant at the P<0.05 level

Table 2: Comparing the average amounts of measuring indices before and after testing in two aerobic exercise and control groups

Variable		aerobic	control	t	P value
Leptin	pre	18/81±5/7	23/51±5/8		
Leptin	post	22/35±9/9	22/42±6/5	0/852	0/036*
17-Beta Estradiol	pre	150/8±40/6	150/8±42/6		
17-Beta Estradiol	post	147/6±43/2	149/5±43	-3/191	0/007
Insulin	pre	9/33±3/6	11/46±3/7		
Insulin	post	7/36±3/17	11/26±3/7	-2/862	0/02*

¥ Independent sample T test (between groups)

Discussion and Conclusion

Inactive and obese people are at the risk of health threatening factors, such as cardiovascular diseases. Many studies have shown that physical exercises improve the glucose and fat metabolism in the body and decrease the risk of suffering cardiovascular disease. (Olive and Miller, 2003). Results represented a significant decreasing in leptin level after a 24-weeks aerobic exercise in aerobic exercise group. The role of leptin has been proved in producing the clot of blood, making oxidative pressure in heart endothelial cells, expanding the calcification of vessels and cell proliferation in flat muscle vessels in laboratorial models (Hojjati et al., 2008). Leptin is related to increasing the number of heart rate and probably the plackets accumulation and producing the clot of blood, in a way that increasing the blood leptin can be considered as an independent risk for the first

heart attack under the influence of ischemia (Hojjati et al., 2008). The fat accumulation in the body regulates the leptin level, and higher number of fat cells in a person's body leads to higher level of leptin in the blood (Kraemer et al., 2003). Also there is a direct relation between the levels of serum leptin and the body mass index (Bouassida et al., 2010). Furthermore, recent evidences have shown that higher level of leptin may increase the body weight and the fat mass (Fleisch et al., 2007). Regular physical exercise and calorie limitation (diet) are two strategies to decrease the leptin levels simultaneously; this is feasible because of losing the body weight (Lowndes et al., 2008). Various physiological factors affect the serum leptin level as well, like being empty stomach and exposing to cold, which each of them reduces the (ob) gene and consequently reduces the circulatory leptin level (Van et al., 1999). Therefore it can be deduced from this research that aerobic exercise and reducing the body mass index and fat amount decreases the level of leptin meaningfully. Experts in this field have claimed that reducing the leptin density probably happens due to weight losing, and our results conforms to Tang et al., (2000), Kramer et al., (2003), Haqiqi and Hamed, (2008), Piri et al., (2009), and Pope et al., (2010).

Also the researches showed that time affects the insulin level in two aerobic exercise and control groups meaningfully. Mentioned reduction is not the same in these groups. French and Castiglioni (2002) have claimed that a 12-weeks enduring swimming exercises reduces leptin and insulin level simultaneously. It seems that there is mutual relation among three parameters – resistance to insulin, body weight and energy negative balance—in regulating the leptin level. Exercise can increase responding to insulin via increasing the glucose carrier in muscle cells (GLUT-4) and insulin receptor substrating (IRS) and increasing the muscle mass (more than 75% of glucose that is caused by insulin stimulation, is related to muscle tissue). Fat acids produced by fat tissue with accumulation in muscle cells derange the GLUT-4 transferring to these cells, and exercise prevent muscle cell accumulation by increasing the oxidation of fat acids (Esteghamati et al., 2008). But it was cleared that enduring exercises and diet increase the sensitivity to insulin (Dyck, 2005). Reducing the sensitivity to leptin in skeleton muscle, which is a consequence of fat diet, may lead to fat acids transferring in cell membrane and this fat is a potential for reducing the insulin sensitivity (Dyck, 2005). It is distinguished that controlling the phosphatidylinositol 3-kinase (PI3K), which is an important enzyme in signaling to insulin, removes the ability of leptin in redistribution of fat acids toward oxidation and avoiding fat accumulation in some extent (Dyck, 2005). When the insulin secretion reduces, the blood glucose will increase, and it is observed in some studies that it decreases the plasma leptin level as well (Daryanoosh, 2009). Leptin affects the fat acid metabolism inside the skeleton muscle importantly. This increasing leads to a reduction in tri-acil glycerol inside the skeleton muscle (Wang et al., 2001). Thus leptin sensitivity reduction in the skeleton muscle may lead to reduction in insulin sensitivity in the tissue because of lipid accumulation inside the muscle cells and derangement in signaling way to insulin (Dyck, 2005). There is a close relationship among the plasma leptin level, body mass index and insulin density (Kraemer et al., 1999). In this research, considering the meaningful reduction of fat amount and body mass index in aerobic exercise group, insulin represented a significant changing as well.

Results showed that no important changes can be observed in insulin after 24 weeks. It is claimed that there is a direct relation between hormones and estradiol role in stimulation of leptin producing and secretion (Geber and Brandão, 2012). Physical exercise may affect the changes in adiposities independently, including the reduction of insulin level that leads to estradiol reduction (Geber and Brandão, 2012). Physical exercise also may reduce the estradiol level via following strategies: 1) Overall, physical exercise decreases the endogenous sexual hormones. Reduction of inner estradiol leads to delayed menstruation, menopause in younger ages and lack of ovulating after increasing the physical exercise (Haglund and Eriksson, 1993). Since estradiol is made of cholesterol, like other sexual steroids, exercise schedule may interrupt the serum estradiol reduction or the estradiol metabolism by affecting on cholesterol level. Physical exercise affects the estrogen metabolism as well. It changes the estradiol enzymes into androstenedione, and it changes into some other useful or harmful metabolites (Travis et al., 2003).

It is suggested in recent guidelines that adults do aerobic exercises at least 150 minutes a week lightly, or 75 minutes a week hardly (Christine et al., 2010). Christian et al., (2010) observed a reduction in estradiol level up to half of its main amount by doing aerobic exercise 150 to 225 minutes a week, and more reduction is possible by more exercising. In another study by McTiernan et al., (2005) which was accomplished to evaluate the effect of a regular 12-month exercise on serum density in estrogenic hormones (estradiol and androstenedione), it was cleared that the most amount of reduction in serum density in estradiol and androstenedione among women who did exercise, can be seen in those who had more than 2% reduction in their body fat.

The results of McTiernan et al. (2004) study showed that a 12-month aerobic exercise, 4 sessions a week and 45 minutes each, affects the reduction of 17-beta estradiol serum density meaningfully. In another study by Williams et al. (2004), which focused on the effect of a regular light exercise during 4 menstruation periods and 4 times a week with 20 to 30% energy reduction, no changes had been observed on serum estradiol and astron. The results of another study by Copland et al. (2002) and Kemmler et al. (2003), which only had studied the effect of a scheduled enduring and compound exercise on serum estradiol changes, revealed a significant increasing in serum estradiol. Lack of concordance between our results and mentioned studies may take place due to short period of exercise (i.e. 6 months), severity and duration of each exercise session in the inactive overweight women's program.

Obese people can be distinguished by the leptin resistance and increasing in their blood. This hormone accompanying insulin affects the performance of coronary and nervous system. Leptin increasing can be considered as an independent factor for coronary disease and wrong performance of vessel walls (Rayah et al., 2010), and considering obtained results in this study, it can be concluded that aerobic exercise decreases the leptin and insulin in inactive overweight women via increasing the capacity of muscle in using fat with reducing the body mass index. It can prevent suffering from obesity and consequently from its related diseases, including Cardiovascular diseases.

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