Effects of Four Weeks of High-Intensity Intermittent Training and Continuous Walking on Atherogenic Indices of Obese Middle-Aged Men

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ABSTRACT

Background and objectives: Elevated blood lipids and physical inactivity are known risk factors of atherosclerosis. The objective of this study was to compare effects of four weeks of high-intensity intermittent training (HIIT) and continuous walking on atherogenic indices of obese middle-aged men.

Methods: Study population consisted of 36 male teachers aged 35-50 years, with mean body mass index (BMI) of 30.7 ± 3.5 kg/m² who were working in the city of Galikesh, northeastern Iran. The subjects were enrolled via purposeful sampling and were randomly divided into two experimental groups and a control group. Before and after the intervention, anthropometric characteristics (height, weight and BMI) and blood pressure of each subject were measured. Fasting blood samples were taken from the left brachial vein 12 hours before the first exercise session and 12 hours after the last exercise session to determine lipid profile. Data were analyzed using SPSS 18 at significance level of 0.05.

Results: The four-week walking exercise significantly decreased serum levels of low-density lipoprotein-cholesterol (LDL-C) (P=0.001) and triglyceride (P=0.001). In addition, the HIIT program significantly increased high-density lipoprotein level (HDL-C) (P=0.004) and significantly reduced LDL-C (P=0.049), LDL/HDL (P=0.002), triglyceride (P=0.01), BMI (P=0.027) and blood pressure (P=0.002). In addition, the results of ANOVA and (Scheffe test) showed a significant increase in HDL-C (P=0.042) values and a significant decrease in VLDL-C (P=0.032), LDL/HDL (P=0.041), triglyceride (P=0.024), BMI (P=0.043) and blood pressure (P=0.016) of HIIT group compared to the control group.

Conclusion: Our findings indicated that HIIT has beneficial effects on some risk factors of atherosclerosis and cardiovascular disease.

Keywords: risk factors, Blood Pressure, High-Intensity Interval Training.
INTRODUCTION

Lipids are small hydrophobic molecules that play a critical role in energy storage, signaling pathways and cellular responses (1). However, abnormal lipid levels can contribute to the development of atherosclerosis (2-4). Obesity and overweight are independent risk factors for atherosclerotic cardiovascular disease (2,5). A body mass index (BMI) of more than 25 kg/m² and 30 kg/m² indicates overweight and obesity, respectively (2,6-8). People with visceral obesity have evidently impaired plasma lipids, including increased triglycerides, apolipoprotein B and low-density lipoprotein-cholesterol (LDL-C) concentrations and decreased high-density lipoprotein-cholesterol (HDL-C) levels (2,9).

Men are at a higher risk of developing obesity since fat mostly accumulates in the pelvic area and lower extremities of women (gynoid fat) but in the abdominal and visceral areas of men (android fat). Accumulated upper body fat increases the risk of stroke, diabetes, hypertension and biliary disease, which is more common in android obesity than in gynoid obesity (10).

Exercise can boost body metabolism, muscle strength, cardiovascular function, metal health and sex appeal, all of which are associated with a high level of confidence (1). Regular exercise prevents various diseases such as hypertension, heart disease, type 2 diabetes and obesity in both men and women (2, 11, 12). Frequent exercise, mostly regular aerobic exercise, such as jogging is one of the best non-pharmacological strategies in the prevention and treatment of cardiovascular disease (2, 11, 13). Exercise causes a drastic increase in post-heparin lipoprotein lipase (LPL), which in turn increases triglyceride clearance and decreases HDL-c clearance from plasma (7). LPL is the most important enzyme in plasma triglyceride catabolism and is thought to be elevated in skeletal muscle, adipose tissue and plasma of individuals with an active lifestyle (14). Such adaptation is usually enhanced by increased fatty acid delivery and oxidation in the skeletal muscle promoted by LPL and adaptation of the carnitine palmitoyl transferase system even under high-intensity interval training (HIIT) (15). Previous studies have shown that metabolic adaptation and oxidation are usually higher following intense exercise compared to endurance training (16, 17).

In a study by Sheikh al-Islami et al. (2011) on obese women, both HIIT and moderate endurance training resulted in a significant decrease in visceral fat oxidation (18). Given the well-established positive effects of medium- and long-term exercise on lipid oxidation, this study aims to determine effects of short-term HIIT and light walk on risk factors of obesity and atherosclerosis in overweight middle-aged men.

MATERIALS AND METHODS

Study population consisted of 36 male teachers aged 35-50 years, with mean BMI of 30.7 ± 3.5 kg/m² who were working in the city of Galikesh, northeastern Iran. Inclusion criteria included willingness to participate and no history of metabolic and orthopedic disorders, medication use, smoking and physical activity in the last six months. The subjects were enrolled via purposeful sampling and were randomly matched and equally divided into two experimental groups and a control group.

Before and after the intervention, anthropometric characteristics (height, weight and BMI) and blood pressure of each subject were measured. The subjects participated in a briefing session and signed a consent form. Fasting blood samples (7 ml) were taken from the left brachial vein 12 hours before the first exercise session and 12 hours after the last exercise session to determine lipid profile.

For subjects in the HIIT group, a 20 m distance was marked by three cones that were placed 10 m apart. The subjects ran at maximum speed from the start point (cone 1) to cone 2 (path A), returned and then ran at the opposite direction for 20 m towards cone 3 (route B) and finally back to the start point (route C). The total distance covered was 40 m. This protocol was performed with four repetitions and at 75% maximum heart rate in the first two sessions. The intensity was gradually increased to eight repetitions and 90% maximum heart rate by session 12. A 30-sec rest interval was given between each repetition.

Subjects in the second experimental group performed 30 minutes of continuous walking at 50% maximum heart rate in the first session. The walking intensity increased to 75% maximum heart rate in the 12th session (5). The control group did not perform any exercise activity during the study. Serum
concentrations of triglyceride, total cholesterol, HDL-C and LDL-C were measured using Pars Azmoon kits and a photometric device. The level of very-low-density lipoprotein (VLDL) was calculated by dividing the total triglyceride by 5.

Obtained data were expressed as mean ± standard deviation. The Shapiro-Wilk test was used to assess normality of data. In addition, one-way ANOVA (Scheffe test) and t-test were used to analyze inter- and intra-group changes, respectively. All statistical analyses were performed in SPSS 18, at significance level of 0.05.

RESULTS
The descriptive characteristics of the subjects are shown in table 1. Our results showed that the four-week walking exercise significantly decreased serum LDL-C (P=0.001) and triglyceride (P=0.001) levels. In addition, HIIT significantly increased HDL (P=0.004) and significantly reduced LDL-C (P=0.049) levels. No significant change in the cholesterol/HDL ratio was observed (Table 2).

Table 1- The characteristics of the subjects in the study groups

| Variable       | Control group (n=12) | Walking group (n=12) | HIIT group (n=12) |
|----------------|----------------------|----------------------|-------------------|
| Height (cm)    | 176.3±7.1            | 171.6±7.7            | 174±3.9           |
| Weight (kg)    | 88.4±15.1            | 84.8±9.6             | 82.6±6.8          |
| Age (years)    | 42.9±4.3             | 43.3±5.5             | 40.2±4.9          |
| BMI (kg/m2)    | 28.22±5              | 29.7±2.2             | 29.5±3.5          |

Table 2- The lipid profile of subjects in the study groups

| Variable       | Group   | Pretest       | Posttest      | Within-group changes |
|----------------|---------|---------------|---------------|----------------------|
|                |         |               |               | **I**                | **P**               |
| Cholesterol    | Walking | 186.3±52.7    | 179.1±35.8    | 0.0917               | 0.376               |
| (mg/dl)        | HIIT    | 210.6±27.8    | 206.5±29.4    | 0.20                 | 0.784               |
|                | Control | 182.6±19.5    | 181.5±31      | 0.607                | 0.680               |
| Triglyceride   | Walking | 183.5±76.9    | 144.2±55.3    | 4.172                | 0.001               |
| (mg/dl)        | HIIT    | 107.9±181.5   | 151.6±92.7    | 3.097                | 0.010               |
|                | Control | 180.1±104.7   | 190.6±99.7    | 0.743                | 0.786               |
| HDL            | Walking | 31.2±13.2     | 35.2±12.5     | -1.4                 | 0.179               |
| (mg/dl)        | HIIT    | 40.2±9.4      | 45.2±8.6      | -0.85                | 0.004               |
|                | Control | 42.1±16.8     | 38.9±7.6      | 0.557                | 0.284               |
| LDL            | Walking | 103.3±37.2    | 98.8±37.7     | 4.172                | 0.001               |
| (mg/dl)        | HIIT    | 143.3±27.4    | 132.5±30.7    | 2.186                | 0.049               |
|                | Control | 107±17.3      | 101±29.5      | 1.747                | 0.074               |
| VLDL           | Walking | 35.2±13.2     | 31.2±12.5     | -1.12                | 0.282               |
| (mg/dl)        | HIIT    | 40.6±24.7     | 31.3±18       | 0.316                | 0.758               |
|                | Control | 39.2±22.7     | 38.9±20.3     | 0.662                | 0.314               |
| LDL/HDL        | Walking | 2.9±1         | 2.9±0.8       | 0.098                | 0.923               |
|                | HIIT    | 3.4±0.7       | 3±0.7         | 4                    | 0.002               |
|                | Control | 2.5±0.8       | 2.6±0.6       | 0.680                | 0.70                |
| Blood pressure | Walking | 133.4±7.2     | 131.5±7.2     | 1.989                | 0.068               |
| (mm/hg)        | HIIT    | 125.3±12.6    | 121.9±11.5    | 4.154                | 0.002               |
|                | Control | 122±8.28      | 123.1±6.4     | 1.09                 | 0.101               |
| Cholesterol/ HDL | Walking | 4.9±1.2      | 4.6±0.77      | 1.037                | 0.319               |
|                | HIIT    | 5.3±1.13      | 5.2±1.4       | 0.326                | 0.750               |
|                | Control | 4.6±0.9       | 4.7±0.96      | -0.246               | 0.809               |
| BMI            | Walking | 29.5±3.5      | 28.8±2.2      | 1.004                | 0.566               |
| (kg/m2)        | HIIT    | 30.7±2.2      | 27.3±2.3      | 2.55                 | 0.027               |
|                | Control | 28.22±5       | 28.52±5.2     | -0.28                | 0.784               |

Table 3- The inter-group changes in the lipid profile variables

| Variable       | Df | Mean difference | P    |
|----------------|----|----------------|------|
| Cholesterol    | 2  | -24.143        | 0.142|
| Triglyceride   | 2  | 7.382          | *0.024|
| HDL            | 2  | -5.07          | *0.042|
| LDL            | 2  | 4.341          | 0.052|
| VLDL           | 2  | 5.166          | *0.032|
| LDL/HDL        | 2  | 0.293          | *0.041|
| Cholesterol/ HDL | 2  | 1.653          | 0.248|
| BMI            | 2  | 7.354          | *0.048|
| Blood pressure | 2  | 10.08          | *0.016|

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The results of ANOVA (Scheffe test) showed a significant increase in HDL-C values and a significant decrease in VLDL-C, LDL/HDL, triglyceride, BMI and blood pressure of HIIT group compared to the control group (Table 3).

DISCUSSION

Our results showed that the four-week walking exercise significantly decreased serum LDL-C and triglyceride levels. In addition, HIIT increased HDL-C and reduced LDL-C, LDL/HDL, triglyceride, BMI and blood pressure. HDL-C transports cholesterol from peripheral tissues to the liver, which then channels the excess cholesterol for excretion into the bile. It is known that elevated HDL-C levels may play a protective role against coronary artery disease (19, 20). However, our results are inconsistent with finding of Kozehchian (21), Pauli (22) and consistent with Ding (23). The mechanism of HDL-C alterations following exercise is complex; enzymes such as LPL, hepatic lipase and cholesteryl ester transfer protein (CETP) play important roles in altering HDL-C concentration. LPL is the most important regulator of HDL-C concentration through hydrolysis of plasma triglycerides (24). It seems that HIIT may contribute more to the hydrolysis of triglycerides and increased LPL activity than walking; however, the HDL-C elevation immediately after exercise is not related to increased LPL activity but rather to a possible decrease in CETP concentration or activity. Cholesteryl ester transfer protein is responsible for the transport of lipids in HDL-C and other lipoproteins. It has been reported that CETP might decrease after exercise, which slows down HDL catabolism thereby increasing HDL-C concentrations (3). One of the mechanisms that could increase HDL-C catabolism is elevation of blood triglycerides, which results in the formation of triglyceride-rich particles, which are suitable substrates for hepatic lipase. In addition to transporting cholesterol to the liver, HDL-C has been proposed as an antioxidant, anti-inflammatory and anti-thrombotic agent that exerts protective effects on endothelial cell function by inhibiting apoptosis (2, 19). Moreover, LDL-C oxidation is known as a key step for initiation of atherosclerosis and HDL-C inhibits LDL-C oxidation through its antioxidant activity (2).

In our study, both exercise protocols caused a significant decrease in triglyceride level, which is in line with findings of previous studies (25-27). Changes in triglyceride levels can be attributed to the LPL’s response to exercise. It has been established that LPL regulates lipoprotein levels and breakdown of triglyceride-rich lipoproteins. Studies have shown that regular aerobic exercise reduces hepatic lipase, thereby lowering triglyceride production in VLDL-C and LDL-C (3). LDL-C elevation is an independent risk factor for coronary artery disease, while lowering LDL-C to 60 mg/dL reduces the incidence of coronary heart disease by 50% over two years (2). It is well-demonstrated that physical activity increases LDL-C concentration, thereby reducing its oxidation rate and ultimately lowering the risk of cardiovascular disease. In other words, increased LDL-C diameter limits its permeability in arteries, and reduces the risk of atheroma plaques formation (21). It has been suggested that exercise intensity may be correlated with the decrease in LDL-C concentrations (28). In a previous study, six weeks of HIIT increased lipids and carbohydrates metabolism, improved the content of several mitochondrial proteins by 18 to 29% (citrate synthase, beta-hydroxyacyl-CoA dehydrogenase and pyruvate dehydrogenase) and increased fatty acid transporters (FABP pm, FAT/CD36) (6). Two studies reported an increase in oxidative capacity and beta-hydroxyacyl coenzyme A dehydrogenase enzyme after two weeks of HIIT. The molecular mechanisms involved in post-HIIT skeletal muscle adaptation have been investigated to some extent. Exercise intensity is a key factor in increasing PGC1-α, the key enzyme in mitochondrial biogenesis. Gibala (29) and Little et al. (30) reported a significant increase in PGC1-α three hours after HIIT. Although upstream signals that stimulate PGC1-α and mitochondrial biogenesis are not well understood, they might be related to strong changes in phosphorylation potential (ATP/ADP/PAMP) during physical activity. Simultaneous activation of AMPK (29) and p38/MAPK via increased reactive oxygen species production might enhance PGC1-α and mitochondrial biogenesis (31). In addition, two weeks of HIIT (10 × 1min with one minute rest interval) increased PGC1-α by 25% (29-31). Yasari et al. showed that six weeks of treadmill exercise biosynthesis of monounsaturated fatty acids,
at 60-70% VO₂max could lower stearoyl-CoA desaturase-1 (SCD-1), which can limit the main constituents of VLDL-C and triglycerides in 2-week-old rats, inhibition of SCD-1 increased lipid oxidation (32). Eight weeks of moderate exercise on treadmill (60% VO₂max) also increased the carnitine palmitoyltransferase in the liver and prevented hepatic steatosis in trained rats. Thus, exercise is capable of inducing hepatic fat oxidation (33, 34).

Opposed to HIIT, walking exercise did not cause a significant decrease in BMI and blood pressure. Similar to our findings, Kanan et al. found that HIIT can significantly reduce BMI, LDL-c and diastolic blood pressure in sedentary obese individuals (35). Important physiological factors including cardiac output, total peripheral resistance, blood volume and the autonomic nervous system regulate blood pressure (36). Other factors such as age, sex and circadian rhythm are also effective in blood pressure regulation (37).

Another risk factor for cardiovascular disease is body weight or BMI. Physical activity decreases with age, which can result in weight gain and fat accumulation. Abdominal obesity is one of the most important risk factors for acute myocardial infarction. Studies show that both genetic and environmental factors can contribute to abdominal obesity and body weight. Lifestyle and dietary habits modification can lower the risk of these conditions (5, 7).

CONCLUSION

As an effective alternative to traditional aerobic exercise, HIIT can be utilized to induce favorable physiological, functional and health-related changes. This finding is of great importance from a public health perspective since this type of training can cause more favorable physiological adjustments in a shorter time compared to moderate-intensity interval training.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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