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Original research

The impact of high intensity interval training on serum chemerin, tumor necrosis factor-alpha and insulin resistance in overweight women

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ABSTRACT

Background: Chemerin is an adipokine that may mediate the link between obesity, inflammation, insulin resistance, and cardiovascular disease. The aim of present study was to elucidate the impact of high-intensity interval training on serum levels of chemerin, tumor necrosis factor-alpha (TNF-α) and insulin resistance in overweight women.

Methods: Twenty-eight overweight, health and young women (mean age; 30.03 ± 3.13 years, mean Body Mass Index; 27.99 ± 2.91 kg/m^2, mean body fat percent; 36.07 ± 1.48%) were randomly assigned to either the control group (CG) or high intensity interval training group (HIIT). The participants exercised for three days per week for 8 weeks. Fasting blood samples were drawn before and after the exercise training program.

Results: The results indicated a significant reduction in serum chemerin and TNF-α concentration in HIIT group than CG. In addition, lipid profile improved following HIIT. Furthermore, weight, body mass index, body-fat percent and waist-to-hip ratio significantly reduced following HIIT. However, the results revealed no significant differences in insulin resistance and insulin levels between HIIT and CG group.

Conclusions: It's appeared that TNF-α and chemerin attenuations by intensive interval training are associated with improvements in body composition in overweight women. Therefore, performing this type of exercise is suggested for weight management and preventing from overweight and obesity complications.

1. Introduction

Obesity is associated with an increase in type 2 diabetes, hypertension and cardiovascular diseases (Boa et al., 2017). Adipose tissue acts as an endocrine organ that releases bioactive molecules known as adipokines (Boa et al., 2017). Despite the fact that excess of adipose tissue (AT) in obesity is generally viewed as harmful to individual's health, an adequate amount of AT and physiological levels of adipokines contribute to normal whole-body metabolic homeostasis (Boa et al., 2017; Mogharnasi et al., 2019a). In obesity, the balance between cardioprotective and pro-inflammatory adipokines shifts towards pro-inflammatory ones, most of which are risk factors for cardiovascular diseases (CVD) (Mogharnasi et al., 2019a). The interplay between adipocytes and components of the immune system changes following obesity (Boa et al., 2017).

Chemerin, a recently discovered adipocytokine, has been shown to regulate adipocyte differentiation and modulate the expression of adipocyte genes, such as glucose transporter-4, adiponectin and leptin, which are involved in glucose and lipid homeostasis (Goralski et al., 2007). It has been shown that chemerin is associated with body mass index (BMI), serum triglycerides, blood pressure, insulin resistance and inflammatory makers (Bozaoglu et al., 2009; Stejskal et al., 2008). In reality, chemerin might be a potential independent adipocytokine marker of metabolic syndrome (Dong et al., 2011). It is believed that pro-inflammatory cytokines TNF-α and IL-6 are involved in the pathogenesis of atherosclerosis (Pedersen et al., 2016). In a meta-analysis review IL-6, TNF-α and CRP attributed to increased cardiovascular risk independent of traditional risk factors in healthy populations (Kaptoge et al., 2013). Increased IL-6, CRP and TNF-α predicts a poor prognosis in stable and unstable CAD (Bozaoglu et al., 2009).

High intensity interval training (HIIT) has been proposed to be a time-efficient method to improve aspects related to body composition and disease (Madsen et al., 2015a, 2015b; Sijie et al., 2012) In this context, it has been shown that a modest improvement in anti-

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inflammatory cytokine after eight weeks of HIIT for subjects at risk for metabolic syndrome, while inflammatory cytokines did not change (Madsen et al., 2015a). In addition, two weeks HIIT did not alter the blood concentrations of IL-6, IL-10, and TNF-α in overweight/obese men (Leggate et al., 2012). Furthermore, no significant change in TNF-α has been demonstrated following six weeks HIIT in 30 overweight men (Ahmadizad et al., 2015). These differences in results may be due to differences in training intensity and volume, and training periods (Madsen et al., 2015a). Finally, it has been shown that exercise training decreases chemerin levels in overweight and obese subjects (Malin et al., 2014; Nam et al., 2016); but few studies has investigated the effects of HIIT on chemerin. Hence, the purpose of the present study was analyzed the effects of eight weeks of HIIT protocol on serum levels of chemerin, TNF-α and insulin resistance in overweight women.

2. Materials & methods

2.1. Participants

Twenty-eight young overweight and healthy women (mean age: 30.03 ± 3.13 years; mean Body Mass Index: 27.99 ± 2.91 kg/m^2; mean body-fat percentage: 36.07 ± 1.48%) from the Islamic Azad University (Iran) participated in this study. They were randomly assigned into equal groups of control (CG) and HIIT. The study conforms to the principles outlined in the Declaration of Helsinki. Their physical activity was controlled by Baecke Physical Activity questionnaire (Milton et al., 2014). All participants provided written informed consent after awareness of benefits and dangers. Individuals with a history of regular exercise in the last year and cardiovascular, liver, kidney and lung diseases and diabetes as well as serious physical trauma were excluded from the study. In addition, dietary intakes of participants were controlled by dietary recall questionnaire (Bogdanis et al., 2013; Mogharnasi et al., 2019b). Before and after the 8-week HIIT period, weight, body mass index (BMI) and body fat percentage were measured with bioelectrical impedance analysis (Boca, South Korea). The characteristics of each group are presented in Table 1.

2.2. Exercise training protocols

The participants in the HIIT group ran a 20-m path that was specified by three cones within 30 s with maximum speed and then walked for 30 s. The duration of training protocol in each session was 2 min running with maximum speed and walking for 2 min. Training progression was implemented by increasing one repetition every 2 weeks and in the 6th week, it reached to 6 repetitions. The training protocol was done in 8 weeks and 3 sessions per week. It should be noted that prior to initiation of the training protocol in each session, the participants spent 10 and 5 min to warm up and cool down, respectively. It is important to note that the protocol is comprised of repeated bouts of maximal 40 m shuttle run test that is a valid test for evaluating anaerobic performance.

2.3. Biochemistry assay

Twenty-four hours before the first training session and 48 h after the protocol, fasting venous blood samples were obtained by venipuncture. Blood samples were centrifuged for 15 min (3000 rpm, 4 °C) and serum samples were stored at −80 °C for subsequent analyses. Serum chemerin concentrations were determined using the ELISA kit (CK-E11406, EastBioPharm, China). The Intra-assay coefficient of variation and sensitivity of the method were <4.0% and 4.99 pg/ml, respectively. Serum TNF-α concentrations were determined using the ELISA kit (CK-E10110, EastBioPharm, China). The Intra-assay coefficient of variation and sensitivity of the method were <5.0% and 1.52 ng/L, respectively. Insulin was analyzed using the TOSOH AIA-600 II Automated Immunoassay Analyzer (TOSOH Bioscience, South San Francisco, CA, USA). Minimum assay sensitivity was 0.5uU/ml, mean intra-assay CV was 4.69%, and inter-assay CV was 6.0%. Homeostasis Model Assessment (HOMA) index was calculated using the insulin resistance formula as the product of fasting plasma glucose (mM) and insulin (μU/mL) divided by the constant 22.5. Triglycerides (TG) were measured with the glyceryl phosphate method. HDL was analyzed using a two-reagent system involving stabilization of LDL-C, very low-density lipoprotein cholesterol (VLDL), and chylomicrons using cyclodextrin and dextrin sulfate, and subsequent enzymatic-colorimetric detection of HDL-C. LDLC was calculated using the Friedewald equation.

2.4. Statistical analysis

The collected data were analyzed in SPSS software (version 16.0) and presented as means ± standard deviations. Initially, normal distribution of data was determined by Kolmogorov–Smirnov test. Due to the normality of data distribution baseline and endpoint continuous values were compared within groups by paired samples *t*-test. Comparison between HIIT and CG was performed by Student's independent *t*-test. $P < 0.05$ was considered to be statistically significant.

Table 1
Changes of anthropometric, lipid profile and insulin resistance measurements of participants in HIIT and CG.

Variables	CG		HIIT	
	Pre	Post	Pre	Post
Weight (kg)	70.84 ± 5.92	71.2 ± 6.12	74.18 ± 4.49	72.55 ± 4.58*
BMI (kg/m^2)	27.84 ± 0.88	27.99 ± 1.06	28.14 ± 1.12	27.57 ± 1.39*
Body fat (%)	35.54 ± 1.54	35.63 ± 1.76	36.6 ± 1.42	35.8 ± 1.68*
WHR	0.84 ± 0.02	0.84 ± 0.01	0.85 ± 0.02	0.84 ± 0.02*
HDL (mg/dl)	44.57 ± 6.33	43.85 ± 6.2	45.21 ± 10.56	49.85 ± 8.6*#
LDL (mg/dl)	105.21 ± 18.32	106.07 ± 18.63	97.5 ± 30.78	88.57 ± 21.93*#
TG (mg/dl)	103.92 ± 37.85	107 ± 39.38	96.71 ± 33.88	87.42 ± 27.99*#
TC (mmol/l)	185.42 ± 24.17	190.42 ± 26.3	173.35 ± 30.13	159.71 ± 24.06*#
glucose (mmol/l)	85.28 ± 5.39	85.28 ± 10.28	86.28 ± 9.24	82.85 ± 7.84
Insulin (μIU/mL)	4.2 ± 0.25	4.47 ± 0.48	4.57 ± 0.41	4.30 ± 0.24
HOMA-IR	0.85 ± 0.15	0.95 ± 0.2	0.95 ± 0.19	0.86 ± 0.11

Values are means ± standard deviations. Abbreviations: BMI; Body Mass Index, CG; Control Group, HDL; High-Density Lipoprotein, HIIT; High Intensity Interval Training, HOMA-IR; Homeostasis Model Assessment-estimated Insulin Resistance, LDL; Low-Density Lipoprotein, TC; Total Cholesterol, TG; Triglyceride, WHR; Waist-Hip Ratio. The asterisk (*) indicates a significant difference from the baseline value in the same group. The hash sign (#) indicates a significant difference from CG at Post stage.

3. Results

In the context of anthropometric measurements, the results indicated no significant differences between the baseline values of weight (P = 0.186), BMI (P = 0.198), WHR (P = 0.976), and body fat (%) (P = 0.988) in CG and HIIT groups. In response to the HIIT program, weight (P = 0.002), BMI (P = 0.003), body fat (%) (P = 0.001) and WHR (P = 0.04) significantly reduce compared with pre-test. However, no significant differences were observed in weight (P = 0.29), BMI (P = 0.13), WHR (P = 0.81) and body fat (%) (P = 0.72) in CG and HIIT groups. The differences in anthropometric measurements are shown in Table 1.

In terms of lipid profile, our results showed no significant differences in baseline levels of HDL (P = 0.578), LDL (P = 0.234), TG (P = 0.302), and TC (P = 0.270) between CG and HIIT groups. However, within-group comparisons revealed that HIIT regimen only resulted in a significant increase in HDL (P = 0.001). In contrast, within-group comparisons showed that HIIT protocol significantly reduced TG (P = 0.021), TC (P = 0.011), and LDL (P = 0.027) levels. At the end of protocol, HDL level was significantly greater in HIIT than CG (P = 0.04). In addition, TC (P = 0.003), TG (P = 0.04), and LDL (P = 0.03) levels were significantly lower in HIIT than CG. The changes in lipid profile measurements are shown in Table 1.

Baseline levels of glucose (P = 0.231), insulin (P = 0.432), and HOMA-IR (P = 0.177), did not differ between CG and HIIT groups. In addition, no significant differences were observed in glucose (P = 0.312), insulin (P = 0.078), and HOMA-IR (P = 0.161) between the two groups after intervention. The results of the changes in insulin resistance markers are shown in Table 1.

In the context of adipokine, the exercise group showed a significant reduction in serum chemerin (1824.64 ± 118.54 vs. 1633.71 ± 137.19 ng/ml for pre and post, respectively) (P = 0.001) and TNF-α (255.56 ± 27.39 vs. 241.33 ± 40.41 pg/ml for pre and post, respectively) (P = 0.017), after 8 weeks. However, the control group did not show a significant difference in chemerin (1952.64 ± 116.56 vs. 2007 ± 112.52 ng/ml for pre and post, respectively) (P = 0.543) and TNF-α levels (270.6 ± 17.05 vs. 273.33 ± 15.93 pg/ml for pre and post, respectively) (P = 0.876). Finally, statistically significant differences were found in chemerin (P = 0.04) and TNF-α (P = 0.01) levels between the two groups after intervention. Chemerin and TNF-α changes are depicted in Fig. 1.

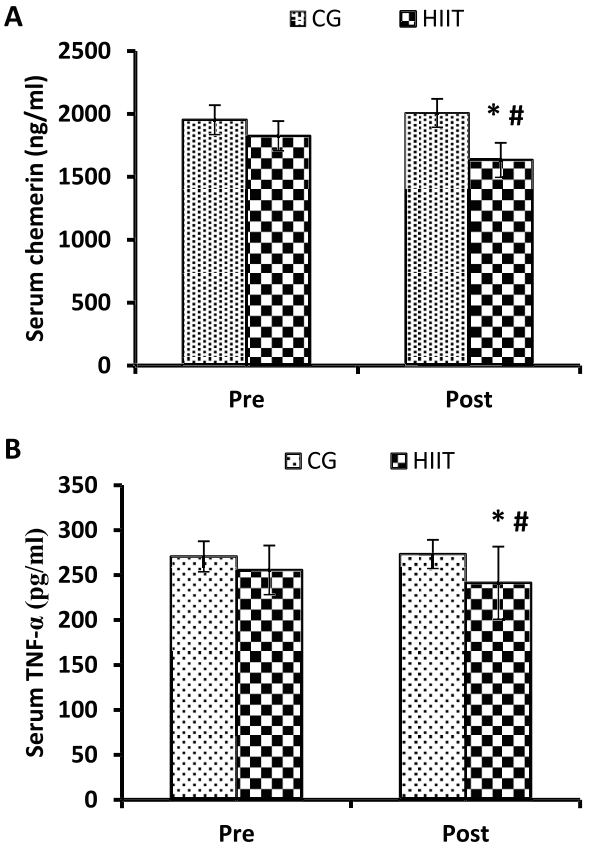

Fig. 1. Effect of HIIT regimen on chemerin (A) and TNF-α (B) level in overweight women. The asterisk (*) indicates a significant difference from the baseline value in the same group. The hash sign (#) indicates a significant difference between groups.

4. Discussion

Following the 8-week intervention, the participants in the HIIT group attained more than 10% reductions in the serum chemerin. The results also revealed that there was no significant difference between HIIT group and the CG in insulin resistance and insulin levels after eight weeks of CG. Also TG, LDL and cholesterol levels reductions after 8 weeks of HIIT were 9.6%, 9.15% and 7.86%, respectively, while HDL increased 10.26%. In response to the training program, weight, BMI, percentage of body fat and WHR tended to decrease (2.19%, 2.02%, 2.18% and 1.17% respectively) compared with pre-test. However, there was no significant difference between HIIT and control groups.

Our findings concerning exercise-induced decreases in chemerin are consistent with previous researches showing declines in chemerin concentrations following chronic exercise interventions (Chakaroun et al., 2012; Kim et al., 2014; Lee et al., 2013; Lloyd et al., 2016). The mechanism by which exercise training reduces chemerin levels has not yet been fully elucidated. Physical activity may be important for sustaining low chemerin levels where physically active individuals typically have more favorable adipokine profiles than less active people (You and Nicklas, 2008). Also, it has been shown that chemerin concentrations increase with adiposity (Bozaoglu et al., 2009; Wang et al., 2009). In accordance with these studies, our results showed that serum chemerin levels significantly reduced after weight, body-fat and WHR reductions in overweight women. Chemerin has been proven to have a direct positive correlation with adiposity parameters i.e. BMI and body fat percentage (Chakaroun et al., 2012; Habib et al., 2017). In vitro, accumulating evidence has shown that the exposing of adipose cells by high concentration of chemerin resulted in the inhibition of cAMP formation, which in turn prevents lipolysis (Carmen and Víctor, 2006). It has been reported that obesity surgery-induced weight loss causes a significant reduction in both omental and subcutaneous adipose tissue chemerin expression (Chakaroun et al., 2012). Both chemerin secretion and its circulatory levels were positively correlated with fat cell size (Andersson et al., 2016). Hence, it is likely that one of the reasons for the decrease of chemerin in the present study is the reduced body fat percentage of overweight subjects. Furthermore, the reduction in chemerin levels indicated that changes in body-fat percentage and WHR after 8 weeks of HIIT may play an important role in the regulation of macrophage infiltration into adipose tissue and serum inflammatory markers. Exercise training can be useful in enhancing insulin sensitivity (Amador et al., 2017). Chemerin has been shown to decrease glucose uptake via insulin signaling in adipocyte and primary human skeletal muscle cells (Robinson et al., 2015). Thus it appears that improvement in peripheral and/or hepatic insulin sensitivity would be associated with reductions in chemerin serum levels after HIIT in overweight subjects. Our results indicate, however, that 8 weeks of HIIT reduced serum levels of chemerin but could not reduce insulin resistance. A possible explanation for this observation may be related to the disparate effects of chemerin on glucose metabolism in adipocytes. While some have shown chemerin to impair insulin stimulated glucose uptake (Kralisch et al., 2009), others have reported that chemerin promoted glucose uptake in adipocytes (Takahashi et al., 2008). Thus, these opposing in vitro data in adipocytes may help explain why in vivo

correlations are observed between chemerin and insulin sensitivity in some (Saremi et al., 2010; Stefanov et al., 2014) but not all human studies (Bozaoglu et al., 2009). Additionally, two recent studies observed associations between changes in chemerin and improvements in insulin sensitivity following chronic exercise interventions. Stefanov et al. (Golbidi and Laher, 2014) found decreases in serum chemerin following a six-month endurance and resistance training regimen to be associated with concomitant decreases in HOMA-IR. Kim et al. (2014) observed a correlation between decreased serum chemerin and improved insulin sensitivity index following a 12-week lifestyle modification program which also included both aerobic and resistance exercise.

TNF-α is a proinflammatory cytokine produced by a great variety of cells, that contributes to insulin resistance and increased risk of myocardial infarction. Up to now, controversial data have been made available about the effects of exercise on TNF-α levels in healthy or diabetic individuals (Lloyd et al., 2016). Our findings show that 8 weeks of HIIT did not significantly change serum levels of TNF-α. TNF-α is a cytokine produced primarily by monocytes and macrophages infiltration in adipose tissue. However, other immune cells, such as lymphocytes and natural killer cells, may also produce it. A high concentration of TNF-α is usually associated with cell death, CVD, inflammation and other acute phase proteins (Golbidi and Laher, 2014). This cytokine also activates certain intracellular kinases, which inhibit the signaling of insulin and hence impair glucose uptake (Gerosa-Neto et al., 2016). Circulating and monocyte-derived TNF-α is a well-established cytokine that induces insulin resistance and generally responds to lifestyle interventions (You and Nicklas, 2008; Kelly et al., 2011). Because resident adipose macrophages secrete additional cytokines that contribute to both local and systemic inflammation, it remains possible that paracrine or autocrine inflammatory secretion contribute to the change in chemerin levels seen in this study. In fact, a study on patients with rheumatoid arthritis demonstrated that anti-TNF therapy reduced chemerin concentrations (Herenius et al., 2013). Leggate et al. (2012) studied 12 overweight and obese males where the participants undertook 6 HIIT sessions over 2 weeks. They did not find any alterations in the blood concentrations of IL-6, IL-10, and TNF-α. A discrepancy in the results could be due to short training periods. Also, our results showed that 8 weeks of HIIT reduced body-fat percentage. Perhaps the weight loss attained at the end of the training program was adequate to mediate TNF-α alteration (Duzova et al., 2018). In line with our findings, it has been demonstrated that weight lose induced by both of step-aerobic and jogging-walking exercises is associated with a decrease in serum TNF-α concentration in overweight sedentary females (Duzova et al., 2018). Moreover, research data have suggested that TNF-α receptor levels are more stable proteins and better indicators of TNF-α activity than serum TNF-α concentrations (Straczkowski et al., 2001). A future study evaluating the influence of exercise on them would thus be more precise.

We found in the present study a decrease in cholesterol, TG, LDL and an increase in HDL following HIIT intervention with no between-group difference. Therefore, the greater reduction in fat mass is likely to contribute to the larger improvement in the adipokine profile and insulin resistance in the training group. The associations between post-intervention reductions in chemerin and fat mass are consistent with previous combined weight loss interventions (Kim et al., 2014; Lee et al., 2013; Khoo et al., 2015), underscoring the links between adiposity and chemerin production. Chemerin messenger RNA (mRNA) expression increases with adipocyte size (Sell et al., 2010) and is lower in lean compared with obese and diabetic individuals (Chakaroun et al., 2012), while changes in the production of other adipokines, particularly adiponectin, may also influence chemerin production (Khoo et al., 2015).

Amount of consumed calories and type of nutrition can affect the circulating chemerin, TNF-α and insulin resistance (Lloyd et al., 2015). One of the limitations of this study was the lack of a controlled diet. In addition, the present study was conducted in a relatively small sample. Therefore, future studies may include more participants and a detailed record of food intake throughout the study.

5. Conclusion

In conclusion, 8 weeks of high intensity interval training reduced chemerin and TNF-α in overweight females. Also, this training program reduced BMI, body-fat percentage, cholesterol, TG, LDL and increased HDL in these subjects. It is suggested that these exercises can be used to prevent complications of obesity and overweight.

Author contributions statement

All authors conceived the study and its design and coordination. SC, HT, FA and SG were involved in the data collection, data analysis, and drafting of the manuscript. Finally, all authors read and approved the final version of the manuscript, and agreed with the order of presentation of the authors.

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Conflicts of interest

There are no conflicts of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.obmed.2019.100101.

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