High-Intensity Interval Training Improves Lipocalin-2 and Omentin-1 Levels in Men with Obesity

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Research Article

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Abstract

Objectives: We investigated the effects of 12 weeks of high-intensity interval training (HIIT) on selected circulating adipokines and other cardiovascular diseases (CVD) risks factors in males with obesity.

Methods: Thirty males with obesity were randomly assigned to HIIT and control groups. The HIIT group participated in a prescribed exercise program for 12 weeks, three times per week. Blood lipids, insulin resistance, and select serum adipokines were assessed before and after 12 weeks of the intervention period.

Results: HIIT improved body composition and lipid profiles ($p<0.05$) as well as decreased fasting insulin levels ($p=0.001$) and HOMA-IR ($p=0.002$) levels. Furthermore, HIIT increased levels of lipocalin-2 (lcn2) ($p=0.002$) while decreasing omentin-1 levels ($p=0.001$) in males with obesity. Changes in lcn2 and omentin-1 concentrations correlated with the changes in risk factors in the HIIT group ($p<0.05$).

Conclusions: The results indicate that 12 weeks of supervised HIIT exercise significantly improves both circulating concentrations of lcn2 and omentin-1, two recently described adipokines, and markers of CVD risk in males with obesity. Further research is necessary to understand the molecular mechanisms involved with these changes.

1. Introduction

Obesity is a complex and multifactorial disease [1] with an escalating global prevalence over the last three decades [2]. Obesity is also a risk factor for many chronic diseases such as metabolic syndrome, type 2 diabetes, and several cancers [3, 4] as well as many cardiovascular diseases [5]. The metabolic abnormalities associated with obesity are partly linked to an imbalance in the secretion of adipokines from adipose [2, 6].

Lipocalin-2 (lcn2), also known as neutrophil gelatinase-associated lipocalin, is a 25 kDa secretory glycoprotein [7]. Lcn2 is a pro-inflammatory molecule that has been implicated in metabolic and inflammatory disorders [8]. The expression and circulating levels of lcn2 are augmented in individuals with obesity and positively associated with the development of insulin resistance and obesity-related metabolic disorders [9]. To this end, obesity-induced increases of lcn2 levels are suggested to be novel and sensitive predictors of CVD [7, 10].

Additionally, omentin-1 (also known as intelectin-1) is another novel adipokine (34 kDa) that is mainly expressed in visceral fat [11, 12]. Omentin-1 has anti-inflammatory effects in obesity-related cardiometabolic disorders [13, 14], and plasma levels of omentin-1 and the adipose tissue gene expression levels are decreased in individuals with obesity [11, 15]. In fact, omentin-1 levels are negatively associated with obesity markers such as body mass index (BMI) and waist circumference (WC) [11, 16]. The decreased omentin-1 expression is implicated in several chronic inflammatory diseases [13] and also in obesity-induced insulin resistance development [17].

Pharmacological management of obesity is often associated with certain side-effects [18]. However, changing lifestyle such as engaging in an exercise training program is a more preferred strategy in primary prevention, as supported by the finding that [19] the beneficial effects of physical activity are partly mediated by changes in circulating adipokines [20, 21]. Although the benefits of traditional exercise formats (aerobic and resistance) on CVD risks are well described [17, 22, 23], there is a scarcity of data and inconsistent findings on the effects of the currently popular high-intensity exercise training formats [24] on circulating adipokines [14, 17, 25].

High-intensity interval training (HIIT) is an alternative to traditional exercise programs with higher adherence rates in adults with overweight/obesity [26]. A recent meta-analysis concluded that HIIT improves cardio-metabolic risk factors and insulin sensitivity in populations with overweight/obesity [19]. Additionally, HIIT provides superior benefits on glycemic control and cardiorespiratory fitness compared to moderate-intensity continuous aerobic training [27]. However, there is a dichotomy regarding the effects of HIIT on some adipokines in individuals with overweight/obesity [14, 28-30]. For instance, a study by Madsen et al. (2015) [28] reported that an eight week HIIT training program improved circulating omentin-1 levels, while a study by Nikseresht and colleagues (2016) failed to show improvements in omentin-1 levels after twelve weeks of moderate interval training in the males with obesity [25]. Likewise, Choi et al. (2009) reported unchanged levels of lcn2 levels in females with obesity after twelve weeks of combined exercise (aerobic and resistance) training [17].

The benefits of HIIT on some of the recently discovered adipokines (such as lcn2 and omentin-1) are poorly described. Furthermore, there are numerous limitations in methodological issues in previous studies, including low participant numbers, varying physical activity abilities of participants, and a wide range of BMI levels, likely accounting for inconsistent findings on the benefits of exercise training (particularly HIIT) in improving adipokine levels in individuals with obesity. To improve these shortcomings, our study examines the influence of HIIT on selected adipokines and other CVD risk factors in a relatively larger group of individuals with obesity. We investigated the effects of 12-weeks of HIIT on lcn2 and omentine-1 concentrations in inactive males with obesity and examined the hypothesis that HIIT leads to beneficial changes in these adipokines concentrations as well as other related CVD risk factors.

2. Methods

2.1, Participants

Thirty healthy but inactive males with obesity volunteered to participate in this study. All subjects were screened thorough medical history and personal health questionnaires. Individuals with histories of acute/chronic health conditions such as cardiovascular or metabolic disease, cerebrovascular disease, cancer, and those who smoked tobacco products. The inclusion criteria were as follows: to be healthy, sedentary (performing < 2 hours of physical activity per week), and between 20–30 years of age, with a BMI ≥ 30 kg/m^2. Prior to being enrolled, all participants underwent a physical examination by a licensed physician to ensure that they were sufficiently healthy to participate in the study. Participants were informed about the experimental procedures and possible risks, and informed consent was obtained before starting the study. The subjects were then randomly allocated to either HIIT training (n= 15) or control (sedentary; n= 15) groups. The study was conducted in accordance with the Declaration of Helsinki, and all procedures and the experimental protocols were approved by the Committee on the Use of Human Research Subjects at the Regional Research Ethics Committee of the Islamic Azad University. Also, all subjects were fully informed about the aims and experimental procedures and provided written informed consent. Subjects were asked to maintain their usual eating habits during the training program. All subjects completed a validated food intake questionnaire and recorded a 24-hour food intake before and at the end of the study so that we could determine if both groups had similar diets.

2.2, Anthropometry and body composition assessments

Anthropometric and body composition variables (body weight, height, BMI, WC, hip circumference (HC), and waist to hip ratio (WHR)) were evaluated using standard techniques before and at the end of the training protocol. Body

weights and heights were measured with light clothing and no footwear after an overnight fast using a digital scale, and the height of the subjects was recorded to the nearest 0.1 cm and weight (nearest 0.1 kg) using a stadiometer. The BMI of each participant was calculated by dividing body weight (kg) by the square of their height (m^2). Waist circumference was measured at the narrowest part of the trunk between the bottom of the rib cage and top of the pelvis, and HC was measured at the largest laterally projecting prominence of the pelvis or pelvic region from the waist to the thigh using a flexible tape measure at the end of a normal expiration. The WHR was calculated as the waist circumference divided by the hip measurement. Fat density (fat mass) was predicted from skin-fold measurements taken on the right side of the body using a specialized skinfold caliper (Baseline Economy 'Slim-Guide', USA) at the triceps, abdominal, and super iliac sites. The percentage of body fat was then estimated through the use of the regression equations described by Brozek et al. [31]. All of the noted measurements were taken by the same trained technician to minimize methodological variations.

2.3, High-intensity interval training protocol

All participants in the training group underwent a 12-week, supervised HIIT exercise program on three non-consecutive days of the week, while the control group maintained their usual lifestyles and did not perform any unusual strenuous activity. All training sessions were performed on an electronic cycle ergometer (Monark Ergomedic 839E electronic test cycle, Sweden) under the supervision of an experienced physical trainer at the University Athletic and Fitness Center. Each of the prescribed sessions began with a 5 min warm-up period consisting of stretching exercises and continuous cycling at a moderate intensity corresponding to 40–50% of each participant's maximal heart rate (HRmax). This was followed by five intervals bouts of cycling exercise (each lasting 2 min) at an intensity of 85-95% HRmax [30], followed by 2 minutes of passive recovery between each bout. The HIIT training started with 85% of HRmax during the first four weeks with 1 min recovery periods between each exercise bout and increased by 5% in each subsequent 4-week period so that the intensity of training reached 95% HRmax with 1 min passive recovery between each exercise bout at the end of the 12^{th} week. At the end of each training session, there was a 5-minute cool-down period involving slow cycling and gentle stretching. Heart rate was monitored during training sessions using a Polar heart rate monitor (Kempele, Finland) to maintain the correct training intensity. The adherence rate to the exercise training program was 100%, with all participants completing each of the three training days every week. The participants were advised to consume the same foods two days before the pretest and post-test blood samplings. The first blood sampling (pretest) was obtained 48 hours before the start of training, and the second blood sample was taken at the end of the program (post-test). The nutritional intake of individuals in all the groups was computed by the method proposed by McCance and Widdowson [32].

2.4, Blood sampling and laboratory measurements

Blood samples were obtained from the participants' antecubital vein at baseline and 48-72 hours after the last training session at the same time (between 7:00 and 9:00 a.m.) after an overnight fast of ten hours. Samples were allowed to clot at room temperature for 10 min and then centrifuged at 3000 (4° C) rpm for 15 min. Fasting levels of blood glucose, insulin, and lipids were measured immediately. In contrast, serum specimens to measure of lcn-2 and omentin-1concentarions were aliquoted into sterile microtubes and frozen at -80^{o} C. Serum concentrations of triglycerides (TG), total cholesterol (TC), and high-density lipoprotein cholesterol (HDL-C) were measured at baseline and end of 12 weeks using an automated clinical chemistry analyzer (Dimension RxL Max, Siemens Healthcare Diagnostics, Germany). Low-density lipoprotein cholesterol (LDL-C) levels were calculated according to

Friedewald et al. [33], where LDL-C = TC - [HDL-C + (TG/5)]. Fasting blood glucose (Glu) concentrations were measured with a modified hexokinase enzymatic method (7020 Clinical Analyzer, Hitachi, Tokyo, Japan), and serum insulin levels were measured using an enzyme-linked immunosorbent assay (ELISA) method (Monobid, California, USA) (intra-Assay CV: 2.1%, inter- Assay CV: 2.4%). Insulin resistance was calculated via the homeostasis model assessment index (HOMA-IR) method: Glu (mmol/L) x fasting insulin (mU/L) / 22.5.

Concentrations of lcn2 were measured with an ELISA in duplicate using commercially available kits (Boster Biological Technology Ltd, China). The intra and inter-assay coefficients of variation for lcn2 were <5.3%, and the sensitivity of the measurements was 0.02 ng/ml. Levels of omentin-1 were determined in duplicate via ELISA kits (Biovendor, Germany). The intra-assay coefficient of variation was 2.8%, and the inter-assay CV was 6.1%, with a sensitivity of 0.12 ng/ml.

2.5, Statistical analysis

Data are presented as mean ± standard deviation (SD). A normal distribution of the data was confirmed using the Kolmogorov-Smirnov test, and homogeneity of variance was verified using Levene's test before statistical analyses were applied. One-way analysis of variance ANOVA was used to evaluate homogeneous groups for anthropometric or physiological parameters at baseline. Paired t-tests were used to compare pre-training and post-training variables in each group (Control or Training). Two-way (group × time) repeated-measures ANOVA was used to evaluate the effects of exercise training on the dependent variables. Effects sizes (ES) were calculated from ANOVA output by converting partial eta-squared to Cohen's *d [34]*. Moreover, within-group ES were computed using the equation ES = (mean post - mean pre) / SD. ES of 0.20-0.60, 0.61-1.19 and ≥ 1.20 were considered as small, moderate, and large, respectively [35]. Pearson's correlation method was used to calculate correlations between changes in the variables in response to training. All statistical analyses were performed using SPSS statistical software (SPSS version 20.0 for Windows, SPSS Inc., Chicago, IL, USA). The significance level was set at $P \leq 0.05$.

3. Results

The physiological, body composition, and anthropometrical characteristics of the participants before and after the study period are presented in **Table 2**. There were no differences in the age, body weight, BMI, and body fat percentage between the training and control groups at the beginning of the study (p>0.05). Twelve weeks of HIIT decreased body morphology and body composition indices such as weight (p=0.001; ES: 0.431), BMI (p=0.001; ES: 0.428), waist circumference (p=0.0001; ES: 0.293), WHR (p=0.007; ES: 0.302), and body fat percent (p=0.001; ES: 0.458), while these parameters remained unchanged in the control group (p>0.05).

Blood chemistry data pre- and post-intervention are presented in **Table 3**. Plasma levels of TC (p=0.007; ES: 0.321) and LDL-C (p= 0.007; ES: 0.288) were reduced, and HDL-C (p=0.008; ES: 0.302) increased. Likewise, fasting insulin levels (p= 0.001; ES: 0.377) and the insulin resistance index (assessed by HOMA- IR) were deceased (p=0.002; ES: 0.334) in males with obesity after 12-week of the HIIT program. There were no modifications in TG (p=0.074) and Glu (p=0.117) concentration after the training program. Notably, serum lcn2 concentration significantly decreased (p=0.002; ES: 0.382) in the 12-week HIIT group, while there were no alterations in the control group (p =0.095). Plasma levels of serum omentin-1 level increased after the 12-week of HIIT (p=0.001; ES: 0.558), while there were no changes in the control group (p =0.416).

We examined the association between changes in adipokine levels (lcn2 and omentin) and changes in cardiovascular risk factors following HIIT. As shown in **Table 4**, changes in omentin-1 correlated negatively with CVD risk factors, while changes in lcn2 levels were positively associated with changes in other CVD risk factors ($p<0.05$).

In addition, we found 12 weeks of HIIT significantly decrease resting heart rate (P=0.001, ES: 0.58) and systolic blood pressure (P=0.0001, ES: 1.02) as well as diastolic blood pressure (P=0.0001, ES: 0.95) compared to pretests values as well as control group **Table 5.**

4. Discussion

Lifestyle interventions such as exercise training can help to control weight and attenuate many cardiometabolic risk factors in individuals with obesity. To that end, we examined the effects of HIIT on serum levels of two novel CVD biomarkers, lcn2 and omentine-1, and other cardiovascular risk factors in inactive males with obesity, and we found that 12 weeks of HIIT significantly improves the concentrations of both these adipokines.

Our data shows improvements in body composition parameters following a 12-week of HIIT in males with obesity. These findings are in line with the previous studies showing the beneficial effects of HIIT on body composition in individuals with overweight/obesity [36, 37]. Moreover, a recent meta-analysis concluded that HIIT is a time-efficient alternative that induces greater reductions in body fat percentage compared to traditional exercise programs in adults with [37]. This could partly be attributed to the higher concentration of the catecholamines (which increase lipolysis in adipose tissues through β-adrenoceptor stimulation) and also with a greater increase in metabolic rate and fat expenditure resulting from HIIT compared to moderate-intensity exercise training [38]. Nevertheless, it should also be noted that some studies report no changes in body composition after the HIIT program in sedentary people [29, 39, 40]. Differences in the participants' age, health/physical activity status, exercise intensity (i.e., parameters of the HIIT program), and particularly the duration of intervention may underlie the discrepancy between our results and findings of other studies. For example, a 4-week HIIT training program may not be sufficient to cause significant changes in body composition in males with obesity in the studies by Alkahtani et al. (2013) [39] and Sasaki et al. (2014) [29].

As noted, we found increased circulating omentin-1 levels in males with obesity after a 12-week HIIT training program. Insulin resistance (measured by HOMA-IR), fasting insulin levels and body markers of obesity was inversely related to the plasma levels of omentin-1. Consistent with our results is a recent study by [14] on males with overweight/obesity showing that an 8-week HIIT program increased plasma omentin-1 levels and that the cardiometabolic profile improved (i.e., decreases of HOMA-IR). Similarly, studies by Madsen and colleagues (2015) reported that eight weeks of a HIIT program increased circulating omentin-1 concentrations and caused a reduction in abdominal fat mass as well as improving glycemic control in patients with type 2 diabetes [28]. Furthermore, it has also been reported that 16-weeks of concurrent exercise training (resistance + aerobic) increased serum omentin-1 concentrations in children with obesity, associated with improved insulin sensitivity and weight loss [41].

Our study indicates that changes in serum omentin-1 are accompanied by changes in body composition and insulin resistance profiles, but we cannot determine if this represents a causal relationship. A potential mechanism for exercise training-induced increases in omentin-1 is likely related to decreases in adipose tissue [28] and weight loss-induced improvements in insulin sensitivity [15], that could, in turn, lead to increased omentin-1 gene

expression [15]. In addition, omentin-1 levels may increase in response to the physiological adaptation of skeletal muscle due to the release of myokines in response to exercise [42]. In contrast to our observations and those described above, a study by Nikseresht et al. (2016) observed no changes in serum omentin-1 concentrations in middle-aged males with obesity after 12 weeks of moderate interval training [25]. Likewise, omentin-1 concentrations were unaltered after three months of aerobic exercise in individuals with obesity [43]. The discrepancies between our findings and those reported by others could be explained, in part, by important differences in the intensity of the training program (moderate vs. high intensity), protocol implementation difference (duration and type of training methods), gender, and in the extent of overall body composition changes.

Our findings also show that the serum lcn2 concentrations are reduced after twelve weeks of HIIT in inactive males with obesity. Recent investigations have demonstrated that serum lcn2 levels are higher in subjects with obesity [9, 44]. The beneficial influences of exercise training on lcn2 levels could lead to mediating obesity-related metabolic disorders and CVD risks by improving the profile of adipokines secretion [45]. A study conducted by Moghadasi and Mohammadi (2014) also reported that lcn2 levels decreased after eight weeks of resistance or endurance training in sedentary young men, likely due to the anti-inflammatory effects of exercise-related adaptation [22]. In addition, plasma levels of lcn2 decreased after eight weeks of endurance training in males with overweight/obesity [22]. Our results also support those, demonstrating a direct association between lcn2 levels and body composition change [23]. This suggests that exercise-induced decreases in lcn2 levels could be attributed to body fat percentage changes and other body composition parameters. In addition, exercise training on its own has been proposed to reduce levels of nuclear factor- kappa B (NF-κB) that activates adipokine and chemokines [46]. Nonetheless, our results are in contrast with those of Choi et al. (2009), who indicated no changes in serum lcn2 concentrations in females with obesity after 12 weeks of aerobic exercise (45 min/session, 300 kcal/day) and muscle strength training (20 min/session, 100 kcal/day) five times a week [17]. The differences in the characteristics of subjects (women vs. men) and, in particular, the intensity of exercise training (moderate vs. high intensity) may explain the discrepancy in exercise-induced changes in plasma adipokine levels [27, 37]. More comprehensive studies are required to understand the exact relationship between fitness, body composition, and lcn2 levels.

There are several limitations to our study to consider. The first is the lack of stringent dietary and energy expenditure controls, which could affect a number of the physiologic variables that can modulate adipokine release. Furthermore, we estimated body fat percentage by using the skinfold thickness method, which is certainly less accurate than a more direct assessment utilizing a dual-energy X-ray absorptiometry (DEXA) or magnetic resonance imaging (MRI) scan [47]. Finally, the current study involved only men and not women.

5. Conclusions

We report that 12-weeks of HIIT induced improvements in body composition, insulin resistance, lipid profiles, and the serum levels of two critical adipokines (lcn2 and omentin-1) associated with CVD and inflammation indicators in inactive males with obesity. Our results support the view that HIIT may be an effective non-medical therapeutic strategy to reduce CVD risk factors and obesity-induced disorders. Further studies are needed to understand the exact mechanisms of adipokine changes in response to exercise training in different populations and also to determine whether measurement of serum lcn2 and omentin-1 levels can serve as predictive biomarkers of CVD risk status.

Abbreviations

BMI: body mass index

CVD: cardiovascular diseases

ELISA: enzyme-linked immunosorbent assay

Glu: blood glucose

HC: hip circumference

HDL-C: high-density lipoprotein cholesterol

HIIT: high-intensity interval training

HR_{max}: maximal heart rate

lcn2: lipocalin-2

NF-κB: nuclear factor- kappa B

TC: total cholesterol

TG: triglycerides

WC: waist circumference

WHR: waist to hip ratio

Declarations

Contribution statement

Sirvan Atashak: Conceptualization, Data Collection, Formal analysis. Stephen R. Stannard: Statistical Analysis and Conceptualization. Ali Daraei: Data Interpretation, Manuscript Preparation and Literature Search. Mohammad Soltani: Data Interpretation, Manuscript Preparation and Literature Search. Ayoub Saeidi: Investigation, Data Collection and Conceptualization. Fatah Moradi: Investigation and Conceptualization. Ismail Laher: Resources and Data Interpretation. Anthony C. Hackney: Resources and Data Interpretation, Supervision. Hassane Zouhal: Supervision, Writing - original draft.

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Tables

Table 1. Food intake (mean ± SD) of study groups for 2 days before the pretest and after post-test blood sampling.

	Training group (n=15)		Control group (n=15)	
Variable	**Pre**	**Post**	**Pre**	**Post**
Total Energy (kcal/d)	2225±190	2306±103	2254±216	2274±136
Total protein (g/d)	111±16	112±17	110±20	114±18
Protein (g/kg BW/d)	1.05±0.45	1.2±0.4	1.1±0.5	1.2±0.3
Total protein (% energy)	18.3±4.1	19.6±3.5	18.1±4.0	19.5±3.0
Total carbohydrate (g/d)	295±19	304±23	305±21	316±20
Total carbohydrate (% energy)	48.5±6.9	50.8±7.1	50.5±6.3	52.6±7.8
Total fat (g/d)	81.5±21	77.3±27	80±22	76±28
Total fat (% energy)	30.9±7.6	28.8±6.9	31.2±8.8	29.5±7.5

Table 2. Body composition of training and sedentary groups measured pre and post-training.

Variable	Training group (n=15)			Control group (n=15)			
	Pre	Post	p_1	Pre	Post	p_1	p_2
Age (years)	24.55±3.21	-	-	25.37±3.01	-	-	0.480
Weight (kg)	93.20±5.13	89.98±3.77*	<0.001	90.75±5.50	91.06±6.23	0.466	0.120
BMI ($kg \cdot m^{-2}$)	31.11±1.17	30.07±1.49*	<0.001	30.74±0.92	30.84±1.10	0.497	0.119
Body fat (%)	29.72±5.44	27.14±5.09*	<0.001	27.61±5.14	27.76±5.17	0.327	0.744
WC (cm)	102.71±7.31	100.19±5.87*	0.000	100.70±5.95	101.10±6.21	0.243	0.684
HC (cm)	100.94±8.21	100.66±8.03	0.450	101.99±4.35	101.81±4.21	0.314	0.626
WHR	1.01±0.03	0.99±0.04*	0.007	0.98±0.03	0.99±0.04	0.148	0.757

Values are mean ± standard deviation. BMI: Body Mass Index; WHR: Waist-to-hip ratio; WC: Waist circumference; HC: Hip circumference. * indicated a significant difference between pre and post-training ($p<0.05$). P_1: p-value within the group; P_2: p-value between groups.

Table 3. Blood biochemical parameters of training and sedentary groups measured pre and post-training

Variable	Training group (n=15)			Control group (n=15)			
	Pre	Post	p_1	Pre	Post	p_1	p_2
Lipid profile							
TC (mg/dl)	202.18±34.04	186.96±23.10*	0.005	190.05±36.95	192.41±39.60	0.097	0.001
TG (mg/dl)	156.21±24.59	152.87±26.29	0.074	149.53±27.37	151.69±27.00	0.0113	0.588
LDL-C (mg/dl)	132.77±34.40	115.26±22.73*	0.007	120.00±38.76	122.36±41.85	0.095	0.001
HDL-C (mg/dl)	38.16±6.66	41.12±5.27*	0.008	40.16±7.42	39.71±7.58	0.204	0.001
Insulin resistance							
Insulin (µg/l)	18.80±5.18	15.97±4.01*	0.001	17.71±5.64	17.83±5.88	0.285	0.001
Glu (mg/dl)	99.68±16.73	98.33±15.94	0.117	96.21±14.45	95.90±16.25	0.536	0.090
HOMA-IR	4.79±1.94	3.98±1.50*	0.002	4.40±2.00	4.43±2.12	0.483	0.001

TC: Total Cholesterol; TG: Triglyceride; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; Glu: glucose; Lcn2: lipocalin-2; * indicated significant difference between pre and post-training (P<0.05). p_1: p-value within the group; p_2: p-value between groups.

Table 4. Pearson correlation analyses of changes of adipokine levels with changes in other CVD risk factors in the training group.

Variables	Δomentin-1	P	ΔLcn-2	P
Δ weight	-0.699	0.004*	0.635	0.004*
Δ BMI	-0.720	0.002*	0.653	0.008*
Δ body fat percent	-0.701	0.004*	0.714	0.003*
Δ WC	-0.575	0.025*	0.466	0.080
Δ HC	-0.330	0.230	0.123	0.663
Δ WHR	-0.475	0.074	0.457	0.087
Δ TG	-0.091	0.748	0.088	0.762
Δ TC	-0.589	0.021*	0.842	0<0001*
Δ HDL	0.562	0.029*	-0.826	0<0001*
Δ insulin	-0.526	0.013*	0.788	0<0001*
Δ LDL	-0.583	0.022*	0.840	0<0001*
Δ Glu	-0.507	0.054	0.507	0.054
Δ HOMA-IR	-0.654	0.008*	0.808	0.000*

Δ: changes from before to after exercise; *: correlation is significant at the $p \leq 0.05$ level.

Table 5. Resting hemodynamic variables (mean ± SD) of training and sedentary groups measured pre and post-training.

Variable	Training group (n=15)		Control group (n=15)	
	Pre	Post	Pre	Post
Resting Heart rate	68.34±6.2	65.23±4.3*#	67.89±5.2	68.32±4.7
SBP	127.2±3.2	121.3±4.3*#	126.9±3.1	127.1±4.1
DBP	79.2.±2.3	77.1±2.1*#	78.5 ±3.5	78.6±3.8

SBP: systolic blood pressure; DBP: diastolic blood pressure; * indicated significant difference between pre and post-training ($p<0.05$). # indicated significant difference between two groups ($p<0.05$).

Figures

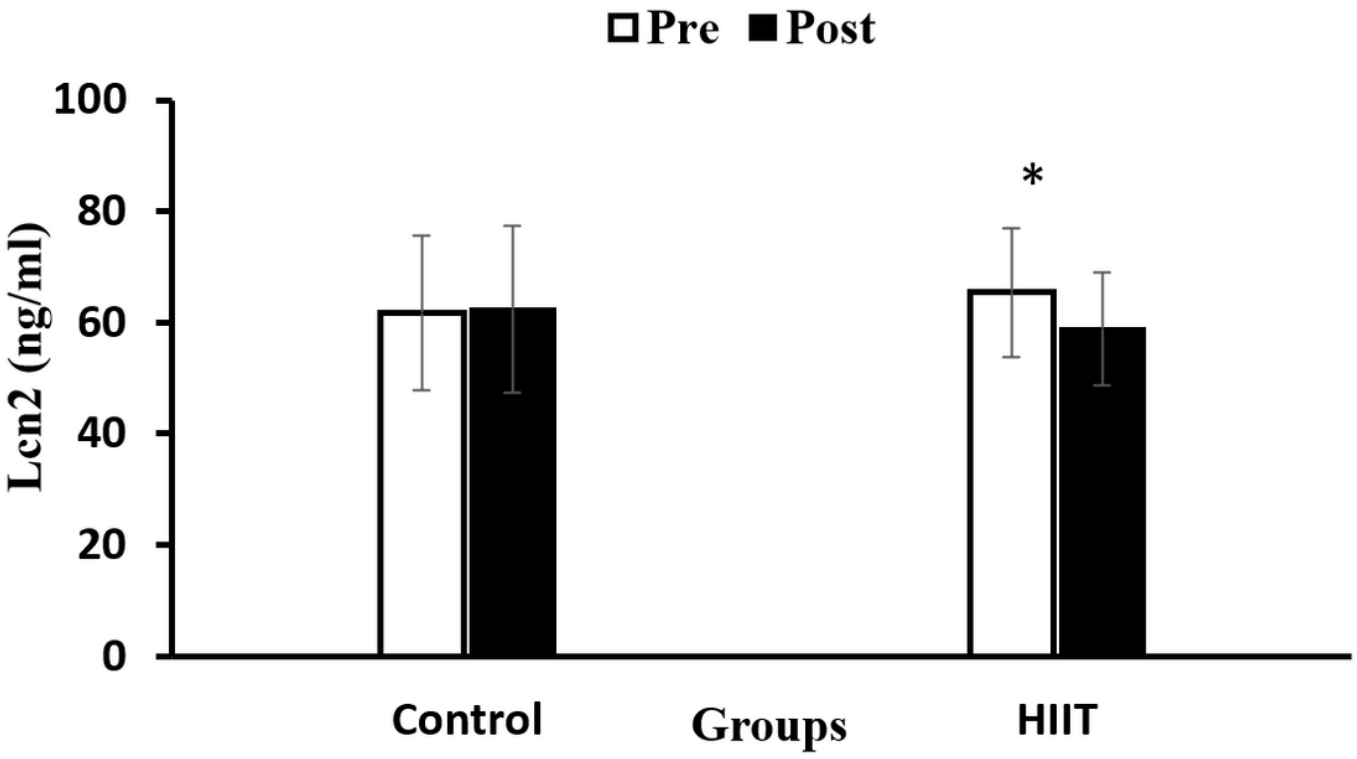

Figure 1

Pre and post-training values (mean±SD) for Lcn2 (lipocalin-2), in HIIT (High intensity interval training), and control groups. * Indicates significant differences from the control group ($P<0.05$).

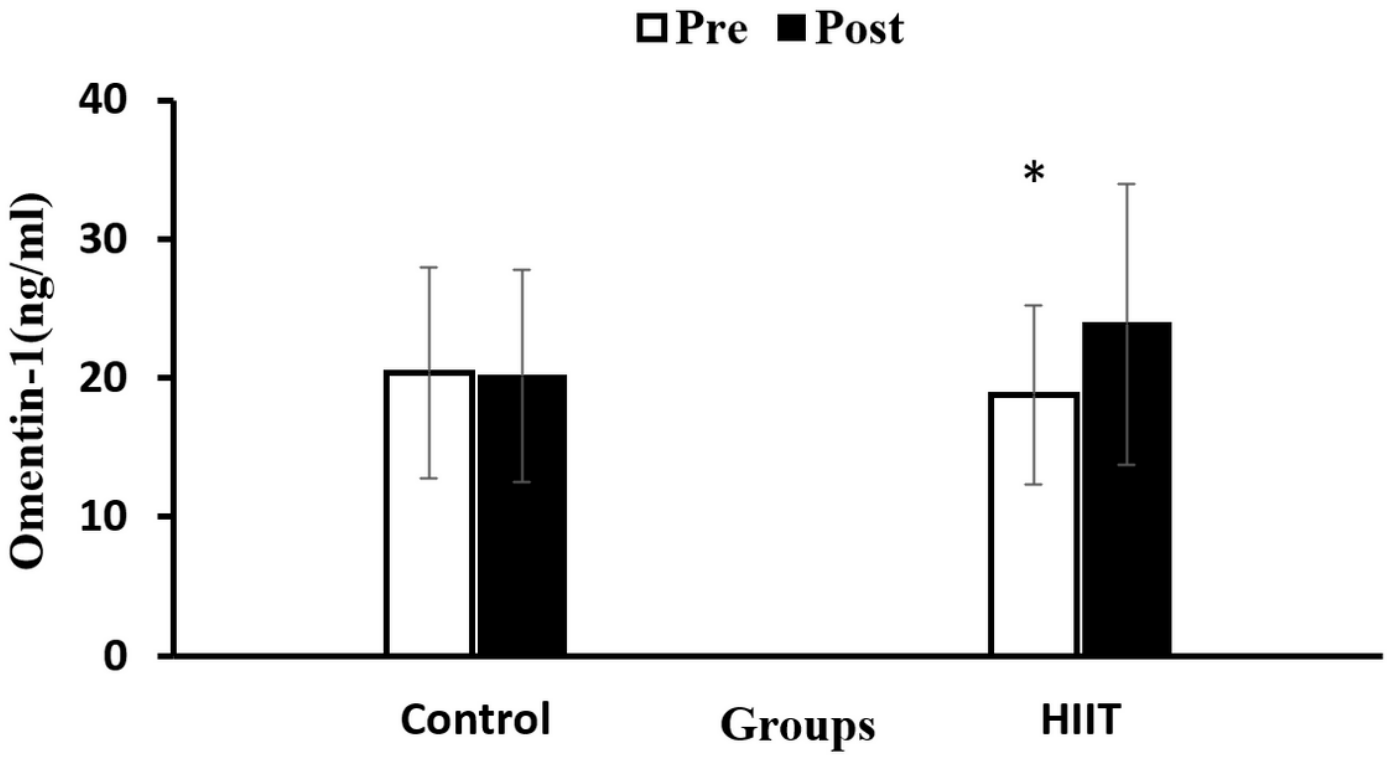

Figure 2

Pre and post-training values (mean±SD) for Omentin-1, in HIIT (High intensity interval training), and control groups.
* Indicates significant differences from the control group (P<0.05).