

Circulating Omentin-1, Insulin Resistance and Lipid Profile Responses Following 8-weeks Aerobic Training Intervention Among Smokers Vs. Non-smokers

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Research

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Abstract

Background: Omentin-1 is a recently circulating adipokine that plays a crucial role in modulating insulin resistance and diabetes. We investigated the effect of eight weeks aerobic exercise training on serum omentin-1, insulin resistance and lipid profile in the smoker and non-smokers with normal-weight.

Methods: Nineteen healthy men and twenty smoker men were randomly assigned into healthy control group (C), healthy exercise group (E), control smoker group (CS) and exercise smoker group (ES). Exercise groups participated in an 8-weeks aerobic exercise training program (three times a week, 45 min per session at 65%-80% of maximum heart rate). Serum omentin-1 and insulin values were determined by ELISA and HOMA-IR, glucose and lipid profile were measured at pre and post of the intervention. Paired Sample t-test, one-way analysis of variance (One-way ANOVA) and post-hoc Tukey test were applied to analyze the data ($p < 0.05$).

Results: Aerobic exercise improved both serum omentin-1 and high lipoprotein cholesterol (HDL-C) in the exercise groups ($p < 0.05$). Also, Exercise training reduced insulin, blood sugar, HOMA-IR, total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) levels ($p < 0.05$). Omentin-1 were significantly associated with insulin ($r = -0.40$, $P = 0.01$), HOMA-IR ($r = -0.38$, $P = 0.04$), TG ($r = -0.40$, $P = 0.01$), TC ($r = -0.49$, $P = 0.02$), LDL-C ($r = -0.70$, $P = 0.02$) and HDL-C ($r = 0.55$, $P = 0.03$).

Conclusion: The findings suggest that aerobic exercise-induced changes in omentin-1 in exercise trained smokers may be associated with the beneficial effects of exercise on reduced insulin and lipid profile.

Introduction

Cigarette smoking is linked to higher risk for developing many respiratory infections disease and cardiovascular diseases in smoker population [1]. The data showed that smokers (aged under 65 years), being a normal weight and current smoker, had an increased risk of death from those being a very obese and never smoker [2]. The evidence confirmed that cigarette smoking were associated with altered normal status of the lipid profile. So, there were significant increment in the levels of total cholesterol (TC), triglyceride (TG), low density lipoprotein (LDL-C), very low density lipoprotein (VLDL) and reduced level of HDL-C among smokers [3]. In fact, smoking leads to accumulation of central fat and thereby insulin resistance in these individuals. Consequently, smoking increases the risk for metabolic syndrome and incidence of diabetes, and these changes increase risk of developing cardiovascular disease [4].

Meanwhile, the adipose tissue, as an active endocrine organ, produces bioactive peptides and proteins that are called "adipokines". Some of these adipokines lead to the prevalence of insulin resistance and cardiovascular complications linked to obesity [5]. Omentin-1, a novel adipokine, is the main isoform of omentins in circulating plasma levels of human that is mostly secreted from visceral adipose tissue [6–7]. Recent evidence have implicated that the possible role of omentin-1 in the pathophysiology of obesity and insulin resistance [8]. The omentin-1 circulating levels are inversely related to insulin resistance and are decreased in obesity, as well as type 2 diabetes [9]. Ansari et al. [10] newly reported decrease in omentin-1 serum level among smokers as a metabolic risk factor and as a major prognostic agent of lung cancer in these individuals. Low omentin-1 levels in response to cigarette smoking can cause the immunomodulatory effect on the immune system that appear to raise the risk of exposure to infections in smokers [11]. However, the exact mechanism underlying the decreased omentin level observed in cigarette smokers is little understood.

Exercise training is an important non-pharmacological strategy to improve insulin sensitivity via several metabolic/physiological changes [9]. Since, regular exercise training diminishes the risk factors for metabolic diseases such as obesity, type 2 diabetes, and cardiovascular disease [12], may modify circulating omentin-1 level. In this regard, Saremi et al. [6] have been reported increment in omentin-1 concentration following 12 weeks of aerobic training in obese participants but Faramarzi et al. [13] observed no statistically significant change in its level after 12 weeks of exercise program in overweight women. However, until now, the effect of exercise training interventions on omentin-1 level has not been explored in smoker. Therefore, the changes of omentin-1 and the possible association between its serum level and metabolic parameters following exercise training can provide new insight into the mechanism underlying the benefits of exercise training in smokers. To our knowledge, this is the first study to determine the effect of an exercise intervention on circulating omentin-1 level and its associations with some metabolic parameters in apparently healthy smoker with normal weight.

Materials And Methods

Participants

For this study, nineteen healthy and twenty smoker young men were randomly categorized into healthy control; C group ($n = 9$; age 27.6 ± 2.5 years, BMI $22.61 \pm 1.94 \text{ kg.m}^{-2}$), healthy exercise; E group ($n = 10$; age 28 ± 2.73 years, BMI $22.77 \pm 1.67 \text{ kg.m}^{-2}$), control smoker; CS group ($n = 9$; age 29.3 ± 2.51 years, BMI $23.59 \pm 2.23 \text{ kg.m}^{-2}$) and exercise smoker; ES group ($n = 11$; age 30.5 ± 1.7 years, BMI $22.72 \pm 1.69 \text{ kg.m}^{-2}$). The smokers had consumed approximately 11–16 cigarettes per day for the past five years. We eliminated subjects who had cardiovascular disease or any other major illness that could have affected the results. The exercise groups participated in an 8-weeks aerobic training program, while the control groups maintained their usual lifestyle. The physical training activity of the participants was determined during one of the experimental sessions in interview. All experimental procedures approved by the ethics committee of Bu-Ali Sina University (Hamedan, Iran). All participants provided written informed consent forms after comprehend all the necessary research-related detail.

Aerobic exercise training program

The aerobic training program was performed 20–35 min a day, 3 days a week for 8 weeks. All exercises training was supervised by an expert exercise physiologist. Each session was executed in three continuous stages as follows: warm-up, main activity and cool-down. The initial intensity of training was

set at 55–65% of an individual's maximal heart rate (MHR) and progressively increased to 65–70% of MHR at the 8th week of protocol [14]. We used an age-based prediction Eq. (220 – age) for calculating the predicted MHR. Heart rate was checked continuously during all exercise sessions by using a Beurer's PM 100 beltless Heart rate monitor (made in Germany).

Experimental Measurements

All baseline measurements were performed prior to the beginning of the protocol, and post testing evaluations were done 48 h after the last session of the training program. Omentin-1 serum level was analyzed by a commercial ELISA kit (Eastbiopharm, Hangzhou, China). The intra-assay and inter-assay coefficients of variation were less than 12%, and sensitivity of the assay was 1.03 ng/mL. The serum values of HDL-C, TG, and TC concentrations were measured by an enzymatic colorimetric method using biochemical Auto-analyzer Prestige 24i (Made in Japan). LDL-C level was estimated using the Friedewald formula when serum TG was < 400 mg/dL. The glucose level was determined using the glucose oxidase method kit (Pars Azmoon, Tehran, Iran). Insulin concentration was assayed with the chemiluminescence method (LIAISON®, Germany). The Homeostasis model assessment of insulin resistance (HOMA-IR) was used to calculate insulin resistance, and it was estimated from fasting glucose and insulin, according the following equation: [Fasting glucose (mg/dl) × Fasting insulin (U/l)/405] (15).

Statistical analysis

The results are expressed as the mean ± standard deviation. All the statistical analyses were conducted with SPSS (version 22) software at significance of 0.05. Assumption of normality of the data distribution and homogeneity of variances were verified by the Shapiro-Wilk test and Levene test. Paired sample t-test was performed to examine within group differences pre and post 8-week exercise intervention in each group. One-way analysis of variance (ANOVA) followed by Tukey's post hoc test was applied to find differences among the groups. The correlations between the variables were estimated by Pearson's correlation test.

Results

Anthropometrical and metabolic characteristics (mean ± SD) of study participants at baseline and after the eight-week exercise intervention are demonstrated in Table 1. After the 8 weeks aerobic training, the ES group experienced weight loss, which was accompanied by significant reductions in BMI, glucose, insulin resistance index, TG, TC, and LDL-C levels ($P < 0.05$). In addition, omentin-1 and HDL-C serum concentrations increased significantly in the exercise groups ($P < 0.05$) (see Table 1 and Fig. 1).

Table 1 Participant characteristics before and after 8 weeks of aerobic training (mean ± SD).

	C group (n=9)		E group (n=10)		CS group (n=9)		ES group (n=11)	
	Before	After	Before	After	Before	After	Before	After
Anthropometry								
age (years)	27.6±2.5	-	28±2.73	-	29.3±2.51	-	30.5±1.7	-
height (cm)	174.83±4.75	-	171.7±6.61	-	173±4.15	-	178.16±8.58	-
Weight (kg)	69.3±8.7	69±8.5	66.8±9.7	65.2±9.3 [#]	70.8±4.8	70.6±3.5	72.3±9.6	69±9.2 [*]
BMI (kg .m ⁻²)	22.61±1.94	22.51±2	22.77±1.76	22.25±1.64	23.59±2.23	23.54±1.95	22.72±1.69	21.67±1.69 ^{*#}
Insulin resistance								
Glucose (mg .dl ⁻²)	86.06±3.47	86.43±3.49	85.95±5.31	76.41±4.72 ^{*#}	86.11±5.47	86.16±6.49	86±1.41	75.16±5.7 ^{*#}
Insulin (μU .ml ⁻²)	5.37±0.66	5.39±1.19	5.39±2.79	2.99±2.1 [*]	5.36±0.29	5.37±0.99	5.37±1.55	3±0.75 [*]
HOMA-IR	1.1±0.12	1.13±0.12	1.21±0.48	0.57±0.31 ^{*#}	1.16±0.2	1.41±0.34	1.17±0.23	0.67±0.26 ^{*#}
Lipid profile								
TG (mg.dl ⁻²)	116.8±6.69	116.69±5.74	116.83±10.18	99.33±6.62 [*]	131.16±18.14	131.5±12.77	131.66±24.88	103±27.8 ^{*#}
TC (mg.dl ⁻²)	149.4±7.5	149.16±3.73	149.6±7.12	137±9.03 [*]	158.4±10.28	158.16±7.7	158±19.18	142.2±24.18 ^{*#}
HDL-C (mg.dl ⁻²)	37.96±10.5	38±8.04	38.25±2.21	42±2.7 [*]	37.8±5.48	38±7.02	38±4.96	42.5±5.44 [*]
LDL-C (mg.dl ⁻²)	80.3±9.57	80.27±6.32	80.48±14.8	70.83±8.72 ^{*#}	83.36±10.6	84.5±7.67	81.24±13.52	67.36±15.54 ^{*#}

* $P < 0.05$, significant difference between pre- and post-8 weeks. [#] $P < 0.05$, significant difference between control and exercise groups after 8 week

Note: HOMA-IR=homeostasis model assessment for insulin resistance; LDL-C=low-density lipoprotein cholesterol; HDL-C=high-density lipoprotein cholesterol.

After the 12 weeks of aerobic exercise training, inverse relationships were identified between omentin-1 and glucose ($r=-0.37$, $P = 0.05$), insulin ($r=-0.40$, $P = 0.01$), HOMA-IR ($r=-0.38$, $P = 0.04$), TG ($r=-0.40$, $P = 0.01$), TC ($r=-0.49$, $P = 0.02$), and LDL-C ($r=-0.70$, $P = 0.02$). Also, a positive correlation was found between omentin-1 and HDL-C ($r = 0.55$, $P = 0.03$) (see Table 2).

Table 2
Pearson correlations of serum omentin-1 with anthropometric and metabolic variables after exercise in smoker exercise group (n = 11).

Variables	p	Correlation
Weight (kg)	0.76	-0.38
BMI (kg.m^{-2})	0.71	-0.48
Glucose (mg.dl^{-2})	0.05	-0.37
Insulin ($\mu\text{U.ml}^{-2}$)	0.01	-0.40
HOMA-IR	0.04	-0.38
TG (mg.dl^{-2})	0.01	-0.40
TC (mg.dl^{-2})	0.02	-0.49
LDL-C (mg.dl^{-2})	0.02	-0.70
HDL-C (mg.dl^{-2})	0.03	0.55
Note: HOMA-IR = homeostasis model assessment for insulin resistance; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol		

Discussion

In this study, we investigated the effects of 8 weeks aerobic exercise training in male smoker with normal weight. Body variables (i.e. body weight and BMI), insulin resistance (i.e. fasting glucose, insulin, and HOMA-IR), and blood lipid profile (i.e. TG, TC, LDL-C) decreased in the ES group compared with the CS group after the 8-week exercise intervention. Our result are in line with previous observations and reinforce the beneficial effect of exercise training on these important metabolic factor in confronting many diseases such as metabolic syndrome, type 2diabetes, and cardiovascular disease [6, 16–17].

Many studies have indicated lower concentration levels of omentin-1 in obesity, impaired glucose tolerance and type 2diabetes [18–20]. Also, lower levels of omentin in response to cigarette smoking may contribute to increased susceptibility to infections in smokers [11]. In order to investigating the changes of omentin-1 in smoker, we explored the effect of 8-weeks aerobic exercise training on circulating omentin-1 in smoker and examined the omentin-1 response in relation to insulin resistance [21]. The findings showed that 8-weeks of aerobic exercise training at 55–70% of MHR led to marked rise of omentin-1 concentration in the participants of E and ES groups. Unfortunately, to the best of our knowledge, no report to date has examined the effect of exercise on circulating omentin-1 level in smoker. Nevertheless, some studies have been done about the effect of exercise training on omentin-1 [21]. Our result was consistent with that of the studies by Wilms et al. [22], Ouerghi et al. [23], and Saremi et al. [6] which illustrated an increase in omentin level following exercise, and is inconsistent with Faramarzi et al. study [13]. It seems that an intensity threshold is essential to improve plasma omentin-1 level, and so it can be the cause of the contradictions with some reports [24]. We found that increase in concentrations of serum omentin-1 was in along with decreasing of blood glucose, fasting insulin level, HOMA-IR and some lipid profiles after 8-week exercise training program [21]. In the present study, 8-weeks of aerobic exercise training effectively lowered insulin resistance in both trained smokers and non-smokers groups, which is in line with previous investigation [17].

In accordance with our results, prior studies have reported that omentin has a negative correlation with fasting insulin, and HOMA-IR [18, 25]. Therefore, mechanism for increase of omentin is directly related to weight loss and a decrease in BMI after exercise training. In this regard, Moreno-Navarrete et al. [26] reported that the concentration of circulating omentin-1 rises after weight reduction, which is consistent with our observation.

The results of current study showed that the levels of insulin, glucose and insulin resistance were reduced in two training groups. Not much research has been done on the mechanism of omentin-1 and its association with glucose and insulin levels, but few studies has shown the role of omentin-1 in transmitting insulin signaling via kinase B protein/Akt activation and increasing the insulin-stimulated glucose uptake into adipose tissue [18]. Also, omentin-1 can improve glucose metabolism and insulin sensitivity by increase glucose transport into the muscles following exercise training. According to the results of Castro et al. [27], there is an association between skeletal muscle and adipose tissue in relation to omentin-1. In fact, exercise training cause an increase in omentin gene expression in adipocytes tissue and improve insulin sensitivity [28]. Nonetheless, there is inconsistency in relationship between omentin level and insulin resistance with the results of Hossein-nezhad study [8] which might be due to various subject populations or other undefined components that may influence on omentin-1 level.

In this study, we obtained inverse correlations between serum omentin-1 level and TG, LDL-C, TC, and also a direct correlation was identified between omentin-1 and HDL-C levels. Contrary to our results Hossein-Nezhad et al. [8] and Elsaid et al. [29] didn't observe any significant relation between serum omentin-1 and lipid levels. Moreno-Navarrete et al. [26] reported that omentin level was correlated with some lipid metabolic parameters such as TC, LDL-C, and TG. Also Abd-Elbaky et al. [30] found an inverse relationship between omentin-1 and TC levels in adults' population that concurs with our findings. It is

proposed that the differences in body fat distribution in the subjects studied may impact on production or secretion of adipokines and the results of investigations [31]. It seems that omentin play an important role in lipid metabolism regulation and so against diabetic dyslipidemia as a compensatory mechanism [26]. Since, it has been shown that omentin-1 promotes 5-AMP-activated protein kinase phosphorylation which acts as an inhibitor of endogenous cholesterol synthesis [21].

Moreover, in agreement with earlier findings [7, 19], we found out serum level of omentin-1 was positively associated with HDL-C level. It was suggested that omentin has anti-atherogenic behavior, thus it can effect on HDL-C level through modulating insulin action [32, 33]. A potential mechanism of increase in HDL-C level following aerobic exercise training in the smoker may be linked to modifications in the activities of some enzymes such as lipoprotein lipase and lecithin-cholesterol acyltransferase and hepatic triglyceride lipase [34].

Conclusion

Our results suggest that 8-weeks aerobic exercise training enhance glucose/lipid metabolism and thereby can reduce complications of smoking through omentin-1 among smokers with normal weight. Omentin-1 had a significant relationship with BMI, insulin resistance and lipid profile. However, the physiological and regulatory role of omentin-1 on energy metabolism following different exercise training protocols (and also detraining period) in smokers is not very well understood. Further experimental studies are recommended.

Abbreviations

Body mass index	BMI
High lipoprotein cholesterol	HDL-C
Homeostasis model assessment of insulin resistance	HOMA-IR
Low-density lipoprotein cholesterol	LDL-C
Maximal heart rate	MHR
Total cholesterol	TC
Triglyceride	TG
Very low density lipoprotein	VLDL

Declarations

Ethical approval

The experimental protocol was in accordance with institutional guidelines and approved by the Ethics Committee of the University. All participants signed voluntary consent forms prior to research commencement.

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Competing interests

"The authors declare that they have no competing interests".

Authors Contributions

Dr. Heidarianpour designed the study. Mr. Mousavi performed the exercise protocol, collected the blood samples, and analyzed the data. Dr. Heidarianpour and Dr. Tavassoli contributed to write the discussion and the manuscript. All authors have approved the final version of the manuscript.

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Availability of data and materials

All data generated or analysed during this study are included in this published article.

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Figures

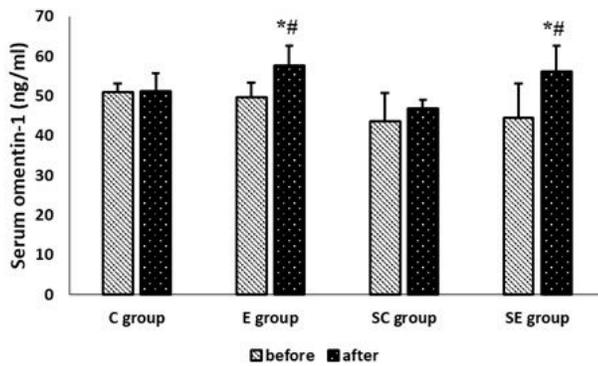


Figure 1

Serum omentin-1 concentration in all participants before and after 8 weeks of aerobic training (mean+SD) * $p < 0.05$; significant difference between before and after the exercise training, and # $p < 0.05$; significant difference between exercise group and control group