Original Article

Effect of High-Intensity Interval Training on Plasma Omentin-1 Concentration in Overweight/Obese and Normal-Weight Youth

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Abstract

Objectives: Omentin-1 is a recently discovered adipokine, mainly produced by visceral adipose tissue, which is thought to improve insulin sensitivity. The study aimed to assess the association of plasma omentin-1 with cardiometabolic traits and physical performance and to test its response to high-intensity interval training (HIIT) in obese and normal-weight subjects. ***Methods:*** Nine overweight/obese (OG) and 9 normal-weight (NWG) young men performed an 8-week HIIT program. Body composition, physical performance, homeostasis model assessment index for insulin resistance (HOMA-IR) as well as plasma omentin-1and lipid levels were assessed before and after the HIIT program. ***Results:*** Baseline plasma omentin-1 was lower in OG than NWG men (359 ± 138 vs. 470 ± 114 ng/ml; p = 0.052). Plasma omentin-1 was related to body fat (r = −0.57; p = 0.03) and LDL-cholesterol (r = −0.49; p = 0.04). There was a trend towards significant association of omentin-1 with BMI (r = −0.47; p = 0.06) and VO2max (r = 0.41; p = 0.09). However, no association was observed with HOMA-IR. Following the HIIT program, omentin-1 concentrations have significantly (p < 0.01) increased in OG (359 ± 138 to 455 ± 126 ng/ml) and NWG men (470 ± 114 to 572 ± 115 ng/ml). In parallel, the cardiometabolic profile has improved with a significant decrease of HOMA-IR in OG. ***Conclusions:*** HIIT resulted in a plasma omentin-1 increase and an improvement with regard to cardiometabolic traits in the OG men, which may contribute to modulate insulin sensitivity.

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Introduction

Obesity and fat excess have become major health problems worldwide. These conditions are associated with increased risk of metabolic dysfunctions such as type 2 diabetes mellitus and cardiovascular diseases [1]. Adipose tissue is an active endocrine organ that secretes various bioactive mediators, the so-called adipokines. These factors signal to several organs including liver, skeletal muscle, brain, and the immune system, modulating lipid and glucose metabolism as well as inflammation [2, 3]. Most adipokines are pro-inflammatory, whereas a small number is anti-inflammatory, exerting beneficial actions upon obesity and obesity-associated morbidities [3]. Omentin-1 is one of the recently discovered adipokines. It is mainly secreted by visceral adipose tissue and expressed in adipose tissue stromal vascular cells [4, 5]. Omentin-1 was shown to enhance insulin-stimulated glucose transport and Akt phosphorylation in adipocytes, which suggest a role in improving insulin sensitivity [5]. Circulating omentin and omentin gene expression in adipose tissue are decreased in subjects with obesity, insulin resistance, or type 2 diabetes mellitus [6, 7]. Moreover, omentin levels were inversely related to BMI, waist circumference, insulin resistance and leptin, and positively related to adiponectin and HDL-cholesterol [6, 8]. These data suggest that omentin plays a beneficial role in obesity and its co-morbidities. However, some recent prospective studies have linked omentin with increased risk of cardiovascular disease and type 2 diabetes [9–11].

Physical activity is an efficient strategy for fighting against obesity and promoting cardiometabolic health [12, 13]. Physical exercise proved to modulate adipokine patterns [14, 15], which may contribute to its beneficial effect in obesity and obesity-related diseases. Data on the response of circulating omentin to physical training are rather scarce and inconsistent. Some studies showed increased levels [16, 17] while others showed no such variation [18, 19]. Almost all available data focused on omentin change following aerobic exercise training in middle-aged obese or diabetic individuals. The response of omentin to other types of training and among other groups of individuals remains unclear.

High-intensity interval training (HIIT) that alternates high-intensity periods of work and periods of rest has gained attention as time-efficient and effective method for improving cardiorespiratory fitness and reducing cardiometabolic risks [20, 21]. To date, only few studies examined the response of omentin to interval training in diabetic adults and individuals at high metabolic risk [22] as well as in middle-aged obese men [23]. This study aimed to test the response of plasma omentin to HIIT in overweight/obese and in normal-weight youth. It also examined the association of basal plasma omentin with cardiometabolic traits and physical performance indices. We hypothesized that HIIT modifies omentin level, along with improving cardiometabolic traits in young individuals, especially in those with weight excess.

Material and Methods

Participants

18 apparently healthy young males, selected among students of high class from two secondary schools in the city of Dahmani (Tunisia), volunteered for this study. Individuals with acute or chronic disease, those under medication, those consuming tobacco or alcohol, and trained athletes were excluded. A medical examination of each participant was done prior to inclusion revealing no contra-indications for physical exercise. Participants' habitual level of physical activity before/during the study consisted of 2 h of physical education lessons. Participants were divided based on BMI in a normal-weight group (NWG; BMI < 25 kg/m^2; age, 18.1 ± 0.93 years; n = 9) and an overweight/obese group (OG; BMI > 25 kg/m^2; age, 18.3 ± 1.22 years; n = 9). Participants followed a HIIT program, 3 times a week for 2 months. All participants conducted 24 training

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Table 1. Eight weeks of short HIIT program

Week of training	1–2	3–4	5–6	7–8
Number of series	2	2	2	2
Number of races per series	8	10	10	10
Run/active recuperation time, s	30/30	30/30	30/30	30/30
Percent of MAV (run/active recuperation)	(100/50)	(100/50)	(105/50)	(110/50)
Passive recovery time, min	5	5	5	5
Training load, ATU	600	750	775	800

ATU = Arbitrary training units.

Example: $(2 \times (8 \times 30 \text{ s} / 30 \text{ s})$; 100% / 50% MAV; passive recovery time = 5 min). It means that the subject had to run 2 series of 8 times 30 s: composed of 30 s running at 100% of MAV and 30 s active recovery at 50% of MAV. The subject recovers passively 5 min between each two series. Each session is repeated 3 times a week. Example of training load calculation for training sessions during the first week: $((100 + 50) / 2) \times 4 \times 2 = 600$ ATU.

sessions. Failure to attend one or more sessions was caught in the same or the following week. Participants were instructed to maintain their usual eating habits during the training program. The study was conducted in accordance with the guidelines of the Declaration of Helsinki and was approved by the Scientific and Ethics Committee of High Institute of Sports and Physical Education of Kef. Participants gave written informed consent to participate in the study.

Study Protocol

The study was conducted between February and April 2014. Temperature varied between 17 and 23 °C and humidity ranged from 70 to 75%. Participants followed the HIIT program during 8 weeks. Body composition, physical performance, and metabolic profile were assessed 1 day before the start and 2 days after the last training session of the program.

Anthropometric Measurements

Weight and height were measured with the subjects barefoot and lightly clothed, allowing calculation of BMI (kg/m^2) = weight / height^2. Skinfold thickness at four sites (biceps, triceps, sub-scapular and supra-iliac sites) were monitored with Harperden's skinfold calipers (Baty International, West Sussex, UK). Percentage of body fat was assessed considering the skin-fold thickness at the four sites according to Durnin and Wormersley [24].

Training Program

The training program was designed for 8 weeks, 3 sessions per week (Monday, Wednesday and Friday). Each training session consisted of a 15-min warm-up, a main stage, and 10-min cool-down stage. Warm-up consisted of 10-min continuous jogging at moderate intensity (50% of maximal aerobic velocity (MAV)), followed by 5-min dynamic stretching exercises and 5 short bursts of 20-meter accelerations. The main stage consisted of two series of 30-second runs at 100-110% of MAV interspersed by periods of active recovery of 30-second runs at 50% of MAV. Training progression was carried out by increasing the number of repetitions from 8 to 10 repetitions from the 3rd week, and increasing the intensity of work from the 5th week (5% increase of the MAV every 2 weeks) (table 1). Finally, participants cooled down by running at low intensity and performing static stretching during 10 min.

Physical Performance Tests

MAV and maximal oxygen uptake (VO2max) were measured via a continuous incremental field test, the Vameval Test [25]. The test was performed on a 400-meter outdoor running track. It starts at a running speed of 8 km/h and increases by 0.5 km/h every minute until exhaustion. Participants adjusted their running speed to cones placed at 20-meter intervals. The test ended when the subject could no longer maintain the required running speed dictated by an audio beep for 2 consecutive occasions. Heart rate was recorded during the test using a Polar heart rate monitor (Polar™ S810, Kempele, Finland). The highest value recorded

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during the Vameval test was considered to be the maximal heart rate (HRmax). To verify the accuracy of the MAV measurement, the HRmax was ensured to be within the interval theoretical HRmax ± 10.

*S*quat-jump (SJ) and counter movement jump (CMJ) were carried out as described by Bosco et al. [26], using an Optojump system (Globus; Microgate Ltd., Bolzano, Italy). The two tests differ by starting position, which is standing position for CMJ and 90° of flexion of the knee joints for SJ. Participants are instructed to jump as high as possible while keeping their hands on their hips. Performance in SJ and CMJ was expressed in flight height (cm). For each test, participants performed three trials with 1 min of recovery in between. The best performance was retained.

The five jump test (FJT) was carried out as described by Chamari et al. [27]. It consists of five successive horizontal jumps. The subject begins with joined feet and ends in the same position. Starting at right station, the subject performs five strides. He jumps on one leg (right or left) into raising the knee and the arms in front. During the 5th stride, the subject brings back both legs together to go back to the starting position. Performance was expressed as total distance (m).

Sprint tests were carried out as described by Chamari et al. [28]. Each subject was asked to run a distance of 30 m in a straight line as fast as possible with a free standing start. Three pairs of photocells were disposed in a straight line; the first one on the starting line, the second on the line of 10 m and the third on the finish line (30 m). The sprint time was registered with photoelectric cells (Microgate Ltd.) that were placed at 1 m height above ground. Each subject performed three trials in total with 3-min of recovery between efforts. The best performance was retained for the analysis.

Blood Sampling and Methods of Analysis

Fasting blood was sampled in morning (around 8 a.m.) from an antecubital vein into EDTA-containing tubes. The blood was drawn 1 day before the start and 2 days after the last training session. The samples were centrifuged at 2,000 × *g* for 25 min, and plasma was frozen at – 40 °C until analysis (within 3 months). Plasma omentin-1 was assessed using an enzymze-linked immunosorbent assay (ELISA) kit (BioVendor, Brno, Czech Republic) with an intra-assay CV below 5%. Plasma insulin concentrations were measured by chemiluminescence immunoassay using a Liaison analyzer and the respective reagents kit (DiaSorin Inc., Stillwater, MN, USA). Plasma glucose, total cholesterol, HDL-cholesterol, and triglyceride were assessed by enzymatic colorimetric methods, and C-reactive protein by immuno-turbidimetric method using an Architect C8000 auto analyzer and the respective reagents kits (Abbott Laboratories, Abbott Park, IL, USA). LDL-cholesterol was calculated using the Friedwald formula [29]. Insulin resistance was estimated by the homeostasis model assessment (HOMA-IR) index as HOMA-IR = (fasting insulin (mU/l) × fasting glucose (mmol/l) / 22.5)) [30].

Statistical Analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences version 18.0 software (SPSS Inc., Chicago, IL, USA). Normality assumption of the data was confirmed using the Kolmogorov-Smirnov test and homogeneity of variance was verified using Levene's test. Independent-samples t-test was used to compare basal variables between groups (OG vs. NWG). Paired-samples t-test was used to compare pre-training and post-training variables in each group (OG or NWG). Changes in dependent variables resulting from the training program were assessed by two-way (time × group) repeated measures analysis of variance. Multiple regression models adjusting for group (OG/NWG) were applied to test correlations of baseline omentin with cardiometabolic traits and indices of physical performance. A p value < 0.05 based on two-sided calculation was considered significant.

Results

At inclusion, total cholesterol, LDL-cholesterol, triglycerides, C-reactive protein and insulin concentrations as well as HOMA-IR were significantly higher in the OG compared to the NWG, whereas aerobic and anaerobic parameters of physical performance were significantly better in NWG (table 2). There was a trend toward significant lower plasma omentin-1 concentrations in the OG compared to the NWG (359 ± 138 ng/ml vs. 470 ± 114 ng/ml; p = 0.052). In regression models combining all participants and adjusting on groups, baseline

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Table 2. Anthropometric, physical performance and metabolic parameters at baseline (pre HIIT) and after (post HIIT) high-intensity interval training in overweight/obese and normal-weight groups

| | NWG (n = 9) | | OG (n = 9) | | Interaction (time × group)[a] | |
	pre HIIT	post HIIT	pre HIIT	post HIIT	F	P
BMI, kg/m^2	20.5 ± 1.51	20.5 ± 1.67	30.8 ± 4.56[***]	30.3 ± 4.25[†]	6.24	0.024
Body fat, %	12.0 ± 3.28	11.9 ± 3.10	22.5 ± 1.87[***]	22.1 ± 1.82[†]	2.17	0.160
MAV, km/h	14.9 ± 0.53	15.4 ± 0.74[††]	11.5 ± 1.15[***]	12.1 ± 0.96[††]	0.64	0.434
VO2max, ml/kg/min	54.1 ± 1.84	55.6 ± 2.58[††]	42.0 ± 4.03[***]	44.2 ± 3.37[††]	0.58	0.459
Heart rate max, beat/min	190 ± 10.0	189 ± 10.2	193 ± 8.65	192 ± 7.58	0.03	0.859
10-meter sprint time, s	2.01 ± 0.11	2.01 ± 0.14	2.40 ± 0.17[***]	2.37 ± 0.15[†]	1.13	0.304
30-meter sprint time, s	4.62 ± 0.19	4.59 ± 0.20[†]	5.63 ± 0.47[***]	5.58 ± 0.43[†]	0.30	0.591
Squat jump height, cm	28.2 ± 2.77	30.0 ± 2.71[††]	19.8 ± 4.18[***]	20.8 ± 4.21[††]	2.31	0.148
CMJ height, cm	30.0 ± 3.24	32.0 ± 3.43[††]	21.6 ± 4.92[***]	23.0 ± 5.11[††]	0.74	0.404
Five jump test length, m	11.1 ± 0.53	11.4 ± 0.69[††]	8.99 ± 1.18[***]	9.30 ± 1.15[††]	0.01	0.910
Cholesterol, mg/dl	136 ± 20.3	127 ± 20.2	175 ± 26.0[**]	150 ± 15.8[†]	2.39	0.142
Triglycerides, mg/dl	82.5 ± 31.1	68.4 ± 16.5	122 ± 39.0[*]	90.0 ± 21.2[†]	1.25	0.280
LDL-cholesterol, mg/dl	85.2 ± 19.2	79.2 ± 15.4	113 ± 30.0[*]	96.2 ± 13.1[†]	1.62	0.221
HDL-cholesterol, mg/dl	36.3 ± 6.15	36.4 ± 7.23	37.0 ± 2.12	37.2 ± 3.21	0.09	0.764
Fasting glucose, mg/dl	89.8 ± 10.5	91.1 ± 9.98	100 ± 13.9	93.9 ± 7.83	1.10	0.309
Fasting insulin, µIU/ml	8.18 ± 5.04	5.69 ± 3.39	21.3 ± 14.22[**]	13.4 ± 5.88	1.19	0.292
HOMA-IR	1.87 ± 1.43	1.26 ± 0.51	4.99 ± 2.62[**]	3.12 ± 1.47[†]	1.96	0.181
C-reactive protein, mg/l	0.85 ± 0.67	1.26 ± 1.42	2.78 ± 1.58[**]	2.97 ± 1.78	0.10	0.759

Data are expressed as mean ± SD.

[*]$p < 0.05$; [**]$p < 0.01$; [***]$p < 0.001$ (compared to baseline in normal-weight group); [†]$p < 0.05$; [††]$p < 0.01$ (compared to baseline in the same group).
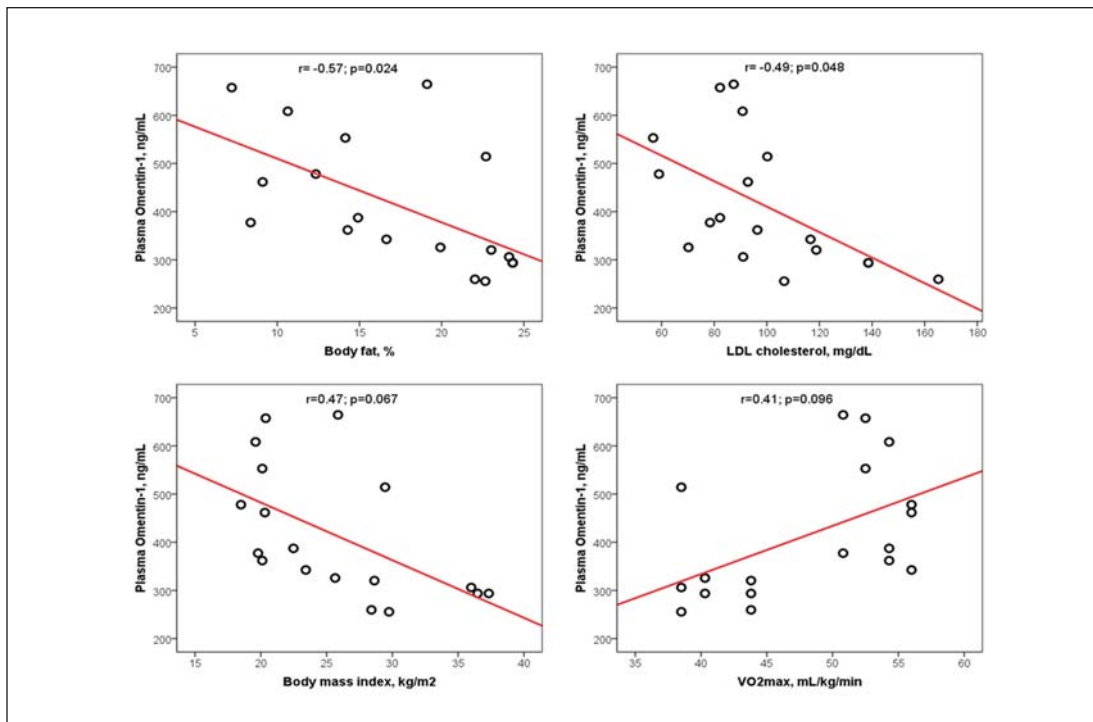
[a]Comparison was performed using two-way repeated measures ANOVA.

plasma omentin-1 was inversely related to body fat and LDL-cholesterol. There was trend towards significant correlations of omentin-1 with BMI, total cholesterol, and VO2max (fig. 1). No significant correlations were observed with HOMA-IR, C-reactive protein, or the indices of anaerobic physical performance.

Eight weeks of HIIT program resulted in a significant increase in plasma omentin-1 in both the OG and NWG (fig. 2). BMI and body fat have decreased in the OG, but not in the NWG. Following the training program, the indices of physical performance (MAV, VO2max, FJT, SJ, CMJ, and 30-meter sprint time) have significantly improved in both groups while the 10-meter sprint time has improved in OG only. Finally, total cholesterol, LDL-cholesterol, triglycerides, and HOMA-IR have significantly decreased in OG, only. Repeated measures detected a significant difference between OG and NWG for BMI, only. No significant differences were detected for all other variables (table 2).

Discussion

The main finding of the study was an increase in plasma omentin levels following HIIT in both overweight/obese and normal-weight untrained young men. Baseline plasma omentin tended towards being significantly lower in the OG compared to the NWG. The last finding agrees with literature data of decreased omentin levels in overweight, obese and diabetic subjects [6, 7, 18]. Plasma omentin was negatively related to body fat and LDL-cholesterol. These findings agree with data from cross-sectional analyses [6, 8, 16] suggesting a beneficial

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Fig. 1. Correlation of baseline omentin-1 with selected cardiometabolic traits and indices of physical performance.

Fig. 2. Plasma omentin-1 concentration in overweight/obese and normal-weight young men at baseline and after HIIT program. **p < 0.01 (compared to baseline in the same group); †p = 0.052 (compared to NWG).
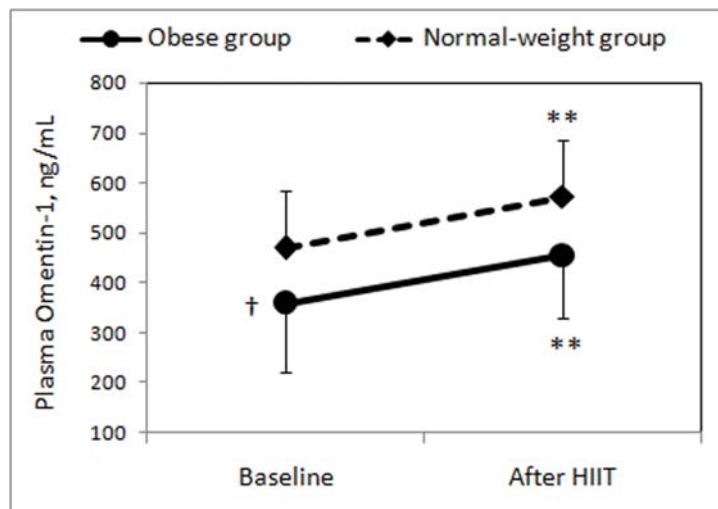
role of omentin in cardiometabolic health. However, there is prospective evidence linking omentin with increased risk of type 2 diabetes and cardiovascular disease [9–11]. The present study found no associations between circulating omentin and HOMA-IR or C-reactive protein. Such data could be part of the controversy over the role of this adipokine in cardiometabolic health. Further work is necessary to examine the role of omentin in cardiometabolic health and to elucidate the underlying mechanisms.

In accordance with previous reports [16, 17], this study showed circulating omentin to be related to VO2max. Such an association is understandable as both circulating omentin and

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physical fitness are actually low in overweight/obese subjects compared to normal-weight subjects. The study detected no significant relationship between circulating omentin and indices of anaerobic performance. To date, no data are available on the association between omentin and anaerobic performance.

Following HIIT, plasma omentin had increased in both overweight/obese and healthy normal-weight youth. In accordance, Madsen et al. [21, 22] reported that an 8-week HIIT training (strenuous bicycle exercise) had induced improvement in circulating omentin, together with abdominal fat mass loss and enhanced glycemic control in middle-aged type 2 diabetics. However, Nikseresht et al. [23] showed no change in omentin after 12 weeks of moderate interval training in middle-aged obese men. The response to aerobic training also showed mixed results, with studies showing omentin increase [16, 17] and others showing no change [18, 19]. The present research is the first to document omentin increase after interval training in youth as well as in normal-weight individuals.

In parallel with omentin increase, the training resulted in improvement in fatness, plasma lipids, and insulin sensitivity in the obese. Previous studies showed concomitant improvement of omentin and cardiometabolic traits following endurance training [16] or dietary intervention [31]. Hence, it could be supposed that omentin increases as a consequence of weight loss or improvement of obesity-related factors. However, circulating omentin had also increased following physical training in obese women with no parallel change in body weight or metabolic traits [17]. Conversely, omentin remained unchanged after exercise training despite significant reductions in weight, insulin, and HOMA-IR [18]. Thus, exercise-associated omentin change could not fully be attributed to weight loss. It may increase in response to physiological adaptation of skeletal muscle to exercise. Skeletal muscle releases myokines into the systemic circulation in response to exercise [32]. These myokines act on various tissues including the adipose tissue, influencing glucose and lipid metabolisms, and energy balance [32]. For instance, circulating irisin had increased following high-intensity interval exercise [33]. Wilms et al. [17] hypothesized that exercise-induced release of myokines from the muscle fibers may modulate omentin secretion by adipose tissue. However, circulating omentin did not change after physical training [18, 19], and increased following dietary intervention [31]. So, it is not clear whether omentin increases as a result of the training itself or as a consequence of the improvement of body composition.

The present study has some limitations. The group sizes are small, and a control group without training is lacking. This may make the finding less consistent and especially makes it difficult to establish causal relationship between plasma omentin increase and improvement of cardiometabolic traits. The study did not control for dietary intake and energy expenditure, which could affect adipose tissue homeostasis. Finally, body fat was estimated based on the skinfold thickness method, which is not as accurate as DEXA scan or MRI for this purpose.

In conclusion, the study showed that omentin is associated with body composition and cholesterol, but not with indices of insulin sensitivity and inflammation. Interval training resulted in an increase in circulating omentin, which was concomitant with an improvement of cardiometabolic traits in the obese. The role of omentin in cardiometabolic health is still not well understood. Further research is needed to clarify the mechanisms of omentin change upon physical training, as well as its impact on physical performance and cardiometabolic health.

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Disclosure Statement

The authors declare that there are no conflicts of interest.

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