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REVIEW ARTICLE

The effect of exercise training on serum Omentin-1 levels, glycemic control and body composition in adults population: a systematic review and meta-analysis of randomized controlled trials

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Abstract

Background Omentin-1 has been acknowledged as an anti-inflammatory and insulin-sensitizing marker, which is mainly expressed in adipose tissue. Exercise training is a therapeutic intervention that can possibly improve and modify circulating Omentin-1 levels.

Objective To determine the effects of exercise training on circulating Omentin-1, glycemic control, and body composition in adult population.

Data sources Four electronic databases and reference lists of included articles were searched until February 5, 2023. The effect size of outcomes was summarized by calculating the mean difference (MD) with 95% confidence interval (CI).

Results Ten RCTs comprising 385 participants were included. The overall model revealed that exercise training increased Omentin-1 compared to the control (MD = 3.57 ng.ml; 95% CI, 1.80 to 5.34 ng.ml; $p < 0.001$). Subgroup analysis by exercise modalities revealed significant increases in Omentin-1 after isolated aerobic ($p = 0.002$) and resistance ($p < 0.001$) training but not after combined training. Subgroup analysis by sex indicated a significant improvement of Omentin-1 in women ($p = 0.015$) and men ($p = 0.007$). Furthermore, a significant increase was found in both healthy ($p = 0.035$) and non-healthy ($p = 0.002$) participants. Analysis of other outcomes indicated that exercise training significantly reduced glucose, insulin, insulin resistance, body weight, body mass index, and body fat, as well as improved lipid profiles.

Conclusion These findings reveal that isolated aerobic and resistance exercises resulted in an increase in serum levels of Omentin-1 in adults. More high-quality studies are required to clarify the mechanisms underlying the influence of exercise training on Omentin-1 concentrations.

Keywords Adipokines · Anti-inflammatory · Exerkine · Insulin resistance · Adipose tissue

Introduction

Metabolic disorders, such as type 2 diabetes, hypertension, and dyslipidemia, are becoming increasingly prevalent in the adult population, posing a significant challenge to healthcare systems worldwide [1–3]. Recent research has revealed that adipose tissue functions as an active endocrine tissue, releasing various bioactive molecules, including adipokines, that play a crucial role in energy homeostasis, blood pressure regulation, and glucose and lipid metabolism [4, 5]. Studies have reported a strong correlation between adipokines and the prevalence of metabolic disorders, suggesting that adipokines could potentially serve as therapeutic targets for the treatment of these conditions [6, 7]. Therefore, it is

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important to investigate the role of adipokines in the pathogenesis and treatment of metabolic disorders [8, 9].

Omentin-1 is a recently identified adipokine that has various isoforms, with Omentin-1 being the main form present in the bloodstream of humans [10]. Omentin-1 plays a crucial role in regulating insulin sensitivity, lipid metabolism, and lipolysis, and has been attributed with important functions such as anti-inflammatory and anti-atherosclerotic effects [11, 12], as well as regulation of high density lipoprotein production through stimulation of insulin receptor substrate [13, 14]. There is a growing body of evidence suggesting a strong association between Omentin-1 and dysmetabolic conditions such as insulin resistance, type 2 diabetes, and metabolic syndrome, as well as inflammatory response [15]. In this regard, a meta-analysis has revealed that omentin levels are significantly lower in individuals diagnosed with type 2 diabetes mellitus compared to controls, and reduced omentin levels are associated with an increased risk of complications in patients with diabetes [14, 16]. Omentin-1 has also been implicated in the development of obesity and obesity-related chronic disorders [17, 18]. Additionally, Omentin-1 has been demonstrated to improve energy balance, glucose metabolism, cardiovascular system function, and reduce oxidative stress [19, 20]. Hence, regulating Omentin-1 secretion and signaling may be important for both the management and mitigation of these adverse conditions [21].

Overall, Omentin-1 is a secretory factor that may act as both an endocrine factor, influencing systemic metabolism, including insulin action in subcutaneous adipocytes, and an autocrine/paracrine factor, regulating the biology of visceral adipose tissue locally [22, 23]. As such, investigating Omentin-1 has significant potential for identifying novel biomarkers and therapeutic targets for metabolic disorders.

Physical exercise is a critical non-pharmacological intervention for improving insulin sensitivity through a variety of metabolic and physiological alterations [24–26]. A growing body of evidence suggests that exercise training interventions may modify circulating Omentin-1 levels, which could have important implications for metabolic health. However, the literature on the effect of exercise on Omentin-1 levels has yielded inconsistent findings. Some studies have reported reductions in Omentin-1 levels after exercise training interventions [27], while others have observed increases [28–31]. Nevertheless, other investigations, such as that by Faramarzi et al. (2016), have reported no alterations in Omentin-1 levels following exercise training [32]. These discrepancies may be attributed to a wide range of factors, including differences in exercise modalities, duration, intensity, and frequency, as well as cohort features such as age, gender, and body composition.

Despite the growing interest in the potential effects of exercise training on circulating Omentin-1 levels, the general impact of exercise on serum Omentin-1 concentrations remains unclear due to inconsistent findings in the literature. To address this gap in knowledge, we conducted a systematic review and meta-analysis of randomized controlled trials (RCTs) in humans to investigate the impact of exercise training on Omentin-1 concentrations in adults. Our study aims to provide a comprehensive synthesis of the existing literature on this topic and to specifically investigate the effects of exercise training on Omentin-1 in adults, thus contributing to the broader understanding of the potential clinical implications of Omentin-1 as a biomarker and therapeutic target for metabolic disorders. In addition to our primary objective, we also assessed the effects of exercise training on insulin resistance, lipid profiles, and body composition in this population, providing valuable insights into the potential clinical benefits of exercise interventions for improving metabolic health.

Methods

We performed the current meta-analysis and systematic review based on Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [33]. This review was not registered.

Literature search

Four electronic databases, including PubMed, Medline, Web of Science, and Google Scholar were used for a detailed search by two investigators (AA and AN). The search utilized the following terms: ((((adult) AND (physical activity OR exercise)) AND Omentin/Omentin-1 AND human) NOT (child OR children OR adolescent OR rat OR mouse OR animal))). For additional eligible studies, reference lists of all relevant research, accompanied by reviews and book chapters, were also hand-searched. The search strategy covered the period from database inception until February 5, 2023. Two independent investigators read the titles and abstracts of the studies and filtered the related studies to include them in the text.

Article selection

To determine the study inclusion criteria, we considered the participants, intervention, comparison, outcome, and study design (PICOS) criteria. To qualify the studies in this review, three investigators (AA, RFM, and GhRMR) independently assessed the titles, abstracts, and full texts of the relevant articles to determine study eligibility. Qualified studies were required to meet the following criteria: (a) population: men and women (aged ≥ 18 years); (b) intervention: aerobic, resistance, or combined aerobic + resistance training, in which subjects did not take part in an exercise

training regimen within the prior 6 months; (c) comparison: non-exercise control group; (d) outcomes: Omentin-1, insulin resistance index, lipids, and body composition were used as outcome measures and measured at baseline and end of the intervention; (e) study design: RCTs published in the English language which comprised a comparison with non-exercise control groups.

Outcome measures

The primary outcome measure in the current study was serum Omentin-1 level at baseline and end of the intervention. The secondary outcome measures were glycemic factors (i.e., glucose, insulin, and HOMA-IR), lipid profiles, and body composition.

Data extraction

The data was extracted by two independent investigators (AA and RFM), and the discrepancies were resolved by the third reviewer (GhRMR). The information extracted included the following: details of publications (author, publication year, and country), characteristics of the study (the sample size for each group, health status, and exercise training modalities), characteristics of participants (mean or range age, gender), details of intervention (intervention period, frequency, intensity, duration, sets, and repetition), mean and standard deviation (SD) of the dependent variables at baseline, end of intervention, and/or changes between baseline and end of intervention. All values for Omentin-1 were recorded as ng.ml or converted to such if necessary.

Data synthesis

The effect size of each outcome was summarized by calculating the mean difference (MD) between the intervention and control groups from baseline and end of intervention for all included studies. Due to the similar methods of reporting techniques for outcomes, we utilized the MD with a 95% confidence interval (CI). The Comprehensive Meta-Analysis (CMA) software version 3.3.070 [34] was applied to conduct the analyses. Extracted outcome data was accomplished utilizing the change in the mean and SD values. The baseline mean was subtracted from the end of intervention mean, and the change SD was computed by applying study group subject numbers along with group p-values or 95% CI, where the change in mean and SD was not available. Where data were not revealed in text or tables and corresponding authors could not be contacted, data shown in figures was extracted or obtained where feasible by the GetData Graph Digitizer software. Where an article included a non-exercise control group and more than one exercise intervention group, we independently labeled each exercise intervention group and adjusted the sample size of the control group based on the number of exercise intervention groups. Because of the significant heterogeneity expected among studies, random-effects models were preferred.

The I^2 statistic [35] and Cochran's Q statistic [36] were used to establish heterogeneity. The I^2 ranges from 0 to 100%, where a value of 0% reflects no observed heterogeneity, and values of 25%, 50%, and 75% show low, moderate, and high heterogeneity, respectively [35]. Subgroup analyses comprising the effect of exercise training modalities, participants' gender, and health status of participants were considered to discover heterogeneity within main effects analyses. Meta-analysis was completed using Forest plots, and a 5% level of significance was considered to represent the significance of results.

Study quality

The Physiotherapy Evidence Database (PEDro) scale, which is an 11-item questionnaire considered to collect data on eligibility criteria, random allocation, concealed allocation, similarity of baseline values, blinding of participants, blinding of therapists and/or assessors, key outcomes, intention-to-treat analysis, between-group differences, point and variability measures, was applied to evaluate the methodological quality of the included studies [37]. This scale has been stated to be valid [38] and reliable [37]. Only those RCTs scoring ≥ 5 on the PEDro scale–a value considered to be of moderate to high quality [39] were considered for analysis. Two investigators (ANH and ShS) individually performed all quality assessments and any discrepancy was resolved by the third investigator (GhRMR).

Publication bias

To qualitatively assess publication bias, we used funnel plots of the effect size generated by Comprehensive Meta-Analysis software (version 2; Biostat Inc., Englewood, New Jersey, USA). Begg and Egger tests were used to assess funnel plot asymmetry, and a substantial publication bias was recognized if the P value was less than 0.10 [34]. To evaluate the effect of publication biases on the interpretation of the results, the trim and fill computation was assessed [34].

Results

Literature search

Initially, 580 papers were identified through database searches, with an additional five papers recognized via reference list searching. After title and abstract screening, 440 papers were omitted, leading to a full text review of

29 eligible studies. Nineteen other articles were removed for the reasons presented in Fig. 1. Ten articles met all inclusion criteria. The PRISMA flow diagram outlining this process is depicted in Fig. 1.

Cohort characteristics

Table 1 reveals the details of included studies. Briefly, when combined, 385 individuals (177 males; 208 females) participated in the studies. Four studies exclusively recruited male participants [28–30, 40]; five studies exclusively recruited female participants [27, 32, 41–43]; and one study recruited both males and females [44]. In 10 studies, the mean age of subjects ranged from 24.5 to 60.3 years. According to Health status classification criteria, four studies had participants who were classified on average as obese [28, 30, 32, 40], two as postmenopausal [42, 43], two as healthy [27, 29], one as type 2 diabetes [41] and one with coronary heart disease participants [44]. All the included studies were RCTs published between 2010 and 2022.

Description of included studies

Our 10 included RCTs (16 intervention groups) had an aggregate of 385 participants, 223 intervention participants, and 162 controls.

The intervention period of the investigations ranged from eight to 16 weeks, with each session's length ranging from 15 to 80 min. Of the 10 included studies, five [29, 30, 32, 43, 44] used only an aerobic exercise program, one examined high-intensity interval training program [28], one [42] explored circuit resistance training, one [27] comprised resistance, endurance and combined resistance + endurance, one [41] explored all three modalities (aerobic, resistance and combined aerobic + resistance), and one [40] comprised nonlinear resistance and aerobic interval training.

The most common types of aerobic exercise included walking, jogging, and running. The intensity of workouts ranged from 55–65% of maximal heart rate, and 65–70% of the symptomatic limit heart rate. Nevertheless, one study [43] did not report the intensity of exercise. The duration of the study of one RCT that used a high-intensity interval training regimen [28] was 12 weeks, include cycling at an

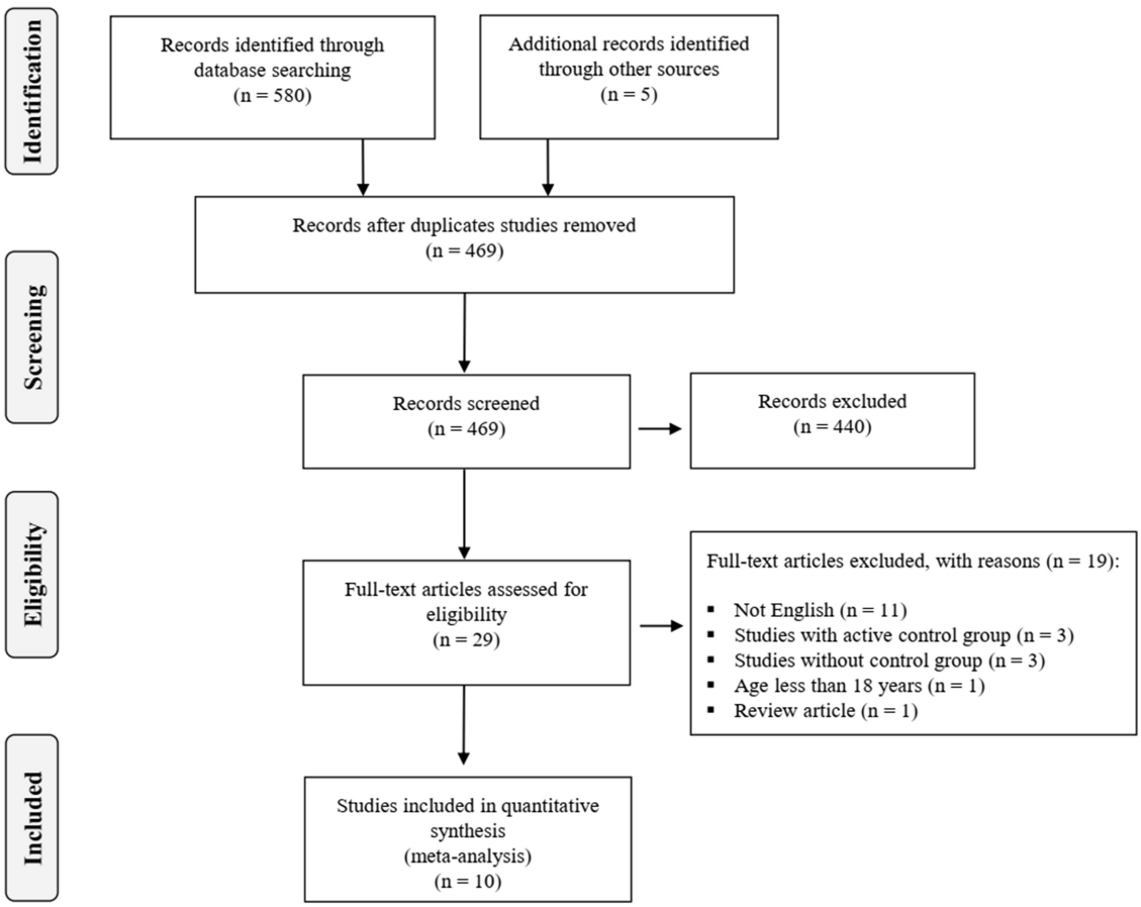

Fig. 1 Flowchart showing the process for the inclusion of studies

Table 1 Meta-analysis of exercise training included studies

Study; country	n INT (CON)	Age (yrs) Mean (SD)	Gender	Health status	Modes of exercise	INT group: frequency and duration	Omentin-1 measurement
AminiLari 2017; Iran	37 (15)	Ranged 45 to 60	Female	T2D	AT, RT, or CT	**AT group:** The warm-up phase comprised of 20 min of stretching and jogging. The main phase of the study was consisted of 25 min exercise in order to achieve 50% to 55% of maximum heart rate as measured by cycle ergometer. Running, exercise and stretching made up the cooling-down phase **RT group**: The warm-up involved 20 min of stretching exercises and jogging on the spot. The main phase consisted of three sets × eight repetitions of weight training including leg extension, prone leg curl, abdominal crunch, biceps, triceps, and seated calf. The exercise intensity was 50% to 55% of one-repetition maximum (1RM). The cooling-down also consisted of running, free exercises and stretching **CT group**: The main phase consisted of aerobic training integrated with RT, with half the execution time and the same intensity of resistance and aerobic groups. The trainings programs were performed within three sessions per week for 12 weeks. Every 2 weeks, in all exercise groups training was increased by 5 min and the intensity by 5%. The average intensity of main stage in every exercise group was 5.5 metabolic equivalent of task (MET) in first week and increased to 7.1 MET at the end of the study The training programs were performed within three sessions per week for 12 weeks	ELISA
Atashak 2022; Iran	15 (15)	INT: 24.5 (3.2) CON: 25.3 (3)	Male	Obesity	HIIT	Each of the prescribed sessions began with a 5 min warm-up cycling at a moderate intensity corresponding to 40–50% of each participant's HRmax cycling exercise (each lasting 2 min) at an intensity of 85–95% HRmax, followed by 1 min of passive recovery between each bout HIIT started with 85% of HRmax during the first four weeks with 1 min passive 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 12th week At the end of each training session, there was a 5-min cool-down period involving slow cycling and gentle stretching	ELISA
Banitalebi 2016; Iran	31 (9)	Total 60.3	Female	Healthy	E + S S + E CI	The exercise intervention was eight weeks of combined (resistance plus endurance) training. Experimental groups underwent training three times per week. Each session consisted of 10 min of general warm up, 50 min of exercise training, and 10 min for cooling-down processes	ELISA

Table 1 (continued)

Study; country	n INT (CON)	Age (yrs) Mean (SD)	Gender	Health status	Modes of exercise	INT group: frequency and duration	Omentin-1 measurement
Faramarzi 2015; Iran	19 (16)	Ranged 25 to 45	Female	Obesity	AT and Core stability	The exercise intervention was 12 weeks of rhythmic aerobic exercise plus core stability training. 3 times per week-each session 10 min general warm-up 30 min rhythmic aerobic exercise and 30 min core stability training and 10 min for cooling processes	ELISA
Mousavi 2022; Iran	21 (18)	INT: Nonsmoker 28 (2.7) Smoker 30.5 (1.7) CON: Nonsmoker 27.6 (2.5) Smoker 29.3 (2.5)	Male	Healthy	AT	The aerobic training program was performed 20–35 min a day, 3 days a week for eight consecutive weeks. Each session was executed in three continuous stages as follows: warm-up by exercises including marching, walking briskly, and jogging for 10 min; main activity (aerobic training program); and cooldown by walking, slow jogging and typical post-running stretches for 10 min The exercise sessions lasted 20 min and the initial intensity of training was set at 55–65% of an individual's maximal heart rate (HRmax) for the first four weeks and was progressively increased to 70% of HRmax for 35 min in the 8th week of the protocol	ELISA
Nikseresht 2016; Iran	22 (11)	NRT grp 40.4 (5.2) AIT grp 39.6 (3.7) CON 38.9 (4.1)	Male	Obesity	NRT, AIT	NRT: The NRT program involved different intensities with flexible periodization and consisted of 55 min of weight training per session and 3 sessions per week for 12 week AIT: AIT included running on a treadmill (4 × 4-min intervals at 80%–90% of maximal heart rate, with each interval separated by 3 min at 65%). The intensity of the training program was controlled by using a heart rate monitor	ELISA
Saeidi 2019; Iran	12 (12)	INT: 58 (5) CON: 56 (5)	Female	Postmenopausal	CRT	Participants in the EG group performed movements at 55% of 1-RM for 8 weeks (3 sessions per week) Each exercise session included a 5 min warm-up and then followed by the 12 prescribed exercises, with duration of approximately 30 s at each exercise station. The number of repetitions at each station was recorded for the participants. In each session, two sets (turns) of 12 exercises were carried out such that between each set, there was a 3 min active rest period	ELISA
Saremi 2010; Iran	9 (9)	43.1 (4.7)	Male	Obesity	AT	It was performed 50–60 min a day, 5 days a week for 12 weeks. The training program began at 60–65% of maximal heart rate and gradually increased to 80–85% of maximal heart rate by week 12. Aerobic training included treadmill walking/running	ELISA

Table 1 (continued)

Study; country	n INT (CON)	Age (yrs) Mean (SD)	Gender	Health status	Modes of exercise	INT group: frequency and duration	Omentin-1 measurement
Wang 2019; China	50 (50)	INT: ranged 42 to 59 CON: ranged 41 to 56	Both	Coronary heart disease	AT	walking and walking up and down stairs slowly, 5–10 min at a time, once in the morning, once in the middle and once in the evening; (2) they gradually increased the intensity of exercise after discharge, the form of exercise was plain walking, the target rate was controlled at 65%-70% of the symptomatic limit heart rate and the exercise was conducted once in the morning and once in the evening	ELISA
Yates 2018; USA	7 (7)	INT: 57.5 (4.4) CON: 55.8 (5.2)	Female	Postmenopausal	AT	The lifestyle intervention was based on the 16-week Diabetes Prevention Program and was delivered as 16 weekly in-person sessions. Participants were provided with individualized calorie goals based on activity level, age, height, and weight, along with a dietary fat intake goal of 25% calories from fat. Participants were instructed to record dietary intake daily, including calculation of total calories and grams of dietary fat consumed, and weigh themselves weekly. Additionally, participants were provided a pedometer to monitor their daily step counts, and worked with study personnel to set up daily step goals. They were also offered the opportunity to participate in two supervised exercise sessions per week	ELISA

Abbreviations: *n*, Number; *INT*, Intervention; *CON*, Control; *SD*, Standard deviation; *T2D*, Type 2 diabetes; *AT*, Aerobic training; *RT*, Resistance training; *CT*, Combined training; *1RM*, One repetition maximum; *BMI*, Body mass index; *HIIT*, High-intensity interval training; *HRmax*, Maximum heart rate; *TG*, Triglyceride; *TC*, Total cholesterol; *HDL*, High-density lipoprotein; *LDL*, Low-density lipoprotein; *E + S*, Endurance + strength; *NRT*, Nonlinear resistance training; *AIT*, Aerobic interval training; *CRT*, Circuit resistance training; *ELISA*, Enzyme-linked immunosorbent assay

intensity of 85–95% maximum heart rate, followed by one minute of passive recovery between each bout.

Effect of exercise on Omentin-1

Ten studies (16 arms) were analyzed for Omentin-1 as revealed in Fig. 2. Overall, exercise interventions resulted in an increase in the Omentin-1 level (MD = 3.57 ng.ml; 95% CI [1.80 to 5.34 ng.ml]; $p < 0.001$; $I^2 = 84\%$; p for heterogeneity < 0.00001). Subgroup analyses are revealed in Supplemental Table 1. There was a significant increase in the Omentin-1 level for the 9 arms reporting the level of Omentin-1 for aerobic exercise interventions (MD = 3.54 ng.ml; 95% CI [1.25 to 5.83 ng.ml]; $p = 0.002$). Moreover, the 3 arms aimed at resistance training evidenced a significant increase in the Omentin-1 level of 2.83 ng.ml (95% CI [1.36 to 4.30 ng.ml]; $p < 0.001$). However, the 4 arms applying a combined aerobic and resistance training protocol discovered a non-significant change in Omentin-1 levels of 4.61 ng.ml (95% CI [-2.39 to 11.62 ng.ml]; $p = 0.197$). Subgroup analysis by sex indicates a significant improvement of Omentin-1 in women (MD = 3.56 ng.ml, $p = 0.015$) and men (MD = 5.43 ng.ml, $p = 0.007$). Furthermore, a statistically significant increase was found in both studies with healthy participants (MD = 2.97 ng.ml, $p = 0.035$) and non-healthy participants (MD = 4.41 ng.ml, $p = 0.002$).

Effect of exercise on glucose, insulin, and HOMA-IR

Seven studies (11 arms) providing a total of 221 participants reported fasting glucose as an outcome measure. Pooled results demonstrated that exercise training significantly reduced glucose levels (MD = -5.92 mg/dl; 95% CI, -10.03 to -1.81 mg/dl; $p < 0.001$; $I^2 = 63\%$; p for heterogeneity = 0.002; Table 2). A comparison of glucose levels based on the mode of exercise training revealed that glucose levels decreased significantly when isolated aerobic (MD = -5.24, $p = 0.30$) and resistance training (MD = -14.03, $p = 0.018$) were used. Another subgroup analyses revealed that glucose levels reduced in studies with male participants (MD = -8.83, $p < 0.001$), as well as in studies with healthy participants (MD = -9.43, $p < 0.001$; Supplemental Table 1).

There was a significant pooled MD for the effectiveness of exercise training programs on insulin levels (-1.52; 95% CI, -2.56 to 0.49; $p = 0.004$, $I^2 = 40\%$; nine intervention arms; Table 2). A comparison of insulin levels based on sex revealed that insulin concentrations reduced significantly only in men (MD = -1.77 [-3.08 to -0.46], $p = 0.008$) but not in women (MD = -0.92 [-2.90 to -0.91], $p = 0.362$). Moreover, subgroup analyses based on health status revealed significant reductions only in healthy subjects (MD = -2.44 [-3.32 to -1.56], $p < 0.001$; Supplemental Table 1).

HOMA-IR was significantly reduced, with an MD of − 0.62 (95% CI, − 0.82 to − 0.42; $p < 0.001$) (Table 2). Both healthy and non-healthy participants revealed a significant reduction in HOMA-IR, with an MD of -0.72 (95% CI, -0.90 to -0.53; $p < 0.001$) and -0.45 (95% CI, -0.82 to -0.07; $p = 0.019$), respectively. Moreover, subgroup analyses based on sex revealed that HOMA-IR decreased significantly in both men (MD = -0.60 [-0.87 to -0.33], $p < 0.001$) and women participants (MD = -0.62 [-1.08 to -0.16], $p = 0.008$) (Supplemental Table 1).

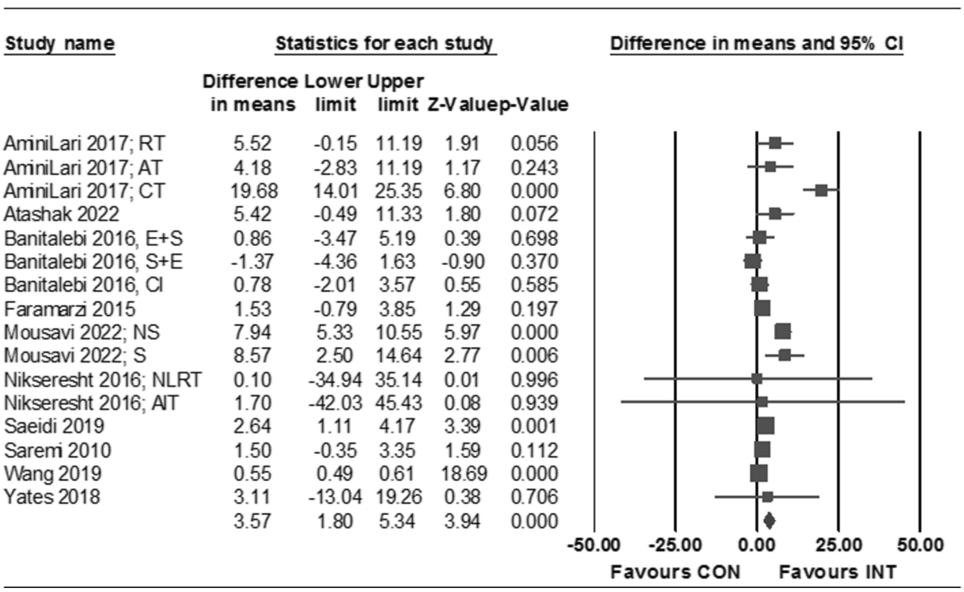

Fig. 2 Forest plot for the Omentin-1. RT, resistance training; AT, aerobic training; CT, combined training; E + S, endurance + strength; S + E, strength + endurance; CI, combined intervention; NS, non-smoker; s, smoker; NLRT, non-linear RT; AIT, aerobic interval training

Table 2 Full results of secondary outcomes

Variable	N. arms	MD (95% CI)	*p*-value	p-heterogeneity	I^2
Glucose (mg.dl)	11	-5.92 (-10.03, -1.81)	< 0.001	0.002	63%
Insulin	9	-1.52 (-2.56, -0.49)	0.004	0.100	40%
HOMA-IR	8	-0.62 (-0.82, 0.42)	< 0.001	0.306	15%
TG (mg.dl)	7	-16.48 (-21.91, -11.05)	< 0.001	0.521	0%
TC (mg.dl)	6	-12.63 (-18.23, -7.04)	< 0.001	0.901	0%
HDL (mg.dl)	7	3.11 (0.34, 5.88)	0.028	0.042	54%
LDL (mg.dl)	7	-7.64 (-14.49, -0.78)	0.029	0.031	56%
Body mass (kg)	14	-2.18 (-3.16, -1.19)	0.003	1.000	0%
BMI ($kg.m^2$)	12	-0.82 (−1.17, −0.47)	< 0.001	1.000	0%
Body fat (%)	12	-2.12 (-2.66, -1.57)	< 0.001	0.926	0%

MD, mean difference; *CI*, confidence interval; *TG*, triglyceride; *TC*, total cholesterol; *HDL*, high-density lipoprotein; *LDL*, low-density lipoprotein; *BMI*, body mass index

Effect of exercise on lipids

Table 2 illustrates the results for lipids. Statistically significant reductions were found for triglyceride after exercise interventions (-16.48 mg/dl [-21.91 to -11.05], $p < 0.001$). Total cholesterol and low-density lipoprotein were also significantly reduced after exercise regimens (-12.63 mg/dl [-18.23 to -7.04], $p < 0.001$ and -7.64 mg/dl [-14.49 to -0.78], $p = 0.029$, respectively). Inversely, high-density lipoprotein was significantly increased after exercise training regimens (3.11 mg/dl [0.34 to 5.88], $p = 0.028$).

Effect of exercise on body composition

Body weight was significantly reduced, with an MD of − 2.18 kg (95% CI, − 3.16 to − 1.19 kg; $p < 0.001$) (Table 2). Non-healthy participants had a larger and significant reduction in body weight, with an MD of -2.38 kg (95% CI, -3.53 to -1.23 kg; $p < 0.001$) than healthy participants (MD = -1.62 kg; 95% CI, -3.53 to 0.30 kg; $p = 0.10$). Moreover, studies with male participants revealed a larger reduction in body weight, with an MD of -3.40 kg (95% CI, -5.66 to -1.14 kg; $p = 0.003$) than those with female participants (MD = -1.89 kg; 95% CI, -2.99 to -0.80 kg; $p = 0.001$) (Supplemental Table 1).

BMI was significantly reduced, with an MD of − 0.82 kg.m^2 (95% CI, − 1.17 to − 0.47 kg.m^2; $p < 0.001$) (Table 2). Non-healthy participants had a larger reduction in BMI, with an MD of -0.86 kg.m^2 (95% CI, -1.27 to -0.45 kg.m^2; $p < 0.001$) than healthy participants (MD = − 0.71 kg.m^2; 95% CI, − 1.38 to -0.05 kg.m^2; $p = 0.036$). Also, studies with male participants had a larger reduction in BMI, with an MD of -0.95 kg.m^2 (95% CI, -1.59 to -0.32 kg.m^2; $p = 0.003$) than those with female participants (MD = -0.76 kg.m^2; 95% CI, -1.18 to -0.34 kg.m^2; $p < 0.001$) (Supplemental Table 1).

Body fat was significantly reduced, with an MD of -2.12% (95% CI, -2.66 to -1.57%; $p < 0.001$). Non-healthy participants had a larger reduction in body fat, with an MD of -2.31% (95% CI, -3.18 to -1.45%; $p < 0.001$) than healthy participants (MD = -1.99%; 95% CI, -2.68 to -1.30%; $p < 0.001$). Furthermore, studies with male participants had a larger reduction in body fat, with an MD of -2.59% (95% CI, -3.62 to -1.55%; $p < 0.001$) than those with female participants (MD = -1.94%; 95% CI, -2.57 to -1.30%; $p < 0.001$) (Supplemental Table 1).

Quality assessment

Supplemental Table 2 shows the quality assessment of the included studies. Overall, the median PEDro score was 6/11 points (ranging from 5 to 8).

Publication bias

The funnel plot, including the Egger regression test for the Omentin-1 analyses, did not suggest publication bias, nor did Duval and Tweedie's trim and fill computation change the results. Moreover, funnel plots, including Egger regression tests for other analyses, did not suggest publication bias, nor did Duval and Tweedie's trim and fill computation change the results (see Supplemental Figs. 1–11).

Discussion

The primary purpose of the present study was to undertake a systematic review and meta-analysis of RCTs considering the effects of exercise training regimens on Omentin-1 levels in adults. The second purpose was to estimate the impact of exercise training protocols on insulin resistance, lipid profiles, and body composition in this population. The meta-analysis found a statistically significant increase in serum Omentin-1 levels among participants who underwent exercise training interventions. In addition, exercise training

resulted in significant improvements in HOMA-IR, glucose and insulin levels, lipids, and all body composition parameters, including body weight, BMI, and body fat. Meanwhile, subgroup analyses according to the type of exercise interventions (aerobic, resistance, and mixed of them), the health status (healthy and non-healthy), and sex (men, women, and both) revealed that exercise training had statistically similar results across these subgroups (Supplemental Table 1).

Recent evidence [6, 45, 46] has confirmed that an improvement in serum adipokine concentrations can lower the risk of various diseases such as obesity, diabetes, and cardiovascular disease, suggesting the potential role of adipokines as therapeutic targets in this regard. Among adipokines, Omentin-1 can disclose the pathophysiology of obesity and insulin resistance [47] by reflecting anti-inflammatory effects in obesity-related cardiometabolic disorders [48]. It has been revealed that Omentin-1 increases insulin action by activating protein kinase B to improve insulin signaling and glucose uptake by adipocytes [22, 49].

Exercise training interventions have been suggested to improve the circulating levels of several adipokines [50]. Previous research has shown, for example, that different exercise training modalities might have therapeutic targets in various populations by increasing plasma or serum adiponectin and decreasing leptin concentrations [46, 51, 52]. Nevertheless, the effects of exercise regimens on Omentin-1, an anti-inflammatory adipokine secreted by visceral adipose tissue [22, 30], still remain unclear in different populations. The findings of the current study revealed that exercise training increased serum Omentin-1 levels in adults.

In adipose tissue, Omentin-1 has been identified as one of the first molecules to exhibit a significant difference in gene expression between visceral and subcutaneous fat depots [22]. Additionally, Omentin-1 has been shown to enhance the effect of insulin action on glucose metabolism, suggesting a potential role in insulin sensitivity [49]. As a secretory factor, Omentin-1 may also act as both an endocrine factor to modulate systemic metabolism and an autocrine and paracrine factor to regulate adipose tissue biology locally [22]. Since Omentin-1 circulates in the blood, it may have effects on distant tissues such as muscle, liver, and subcutaneous fat to enhance insulin sensitivity and glucose metabolism, suggesting a wider role in nutrient storage and partitioning. As subcutaneous fat involves more than 80% of the adipose tissue in the human body, the fact that Omentin-1 circulates systemically and potentiates insulin action in subcutaneous fat may be of physiological and pathophysiological importance [22]. While the clinical applications of Omentin-1 are not yet fully established, our findings are consistent with the findings of Jung et al. (2021) and suggest that Omentin-1 may have significant potential as a newly identified hormone with important roles in adipose tissue and systemic metabolism. Furthermore, our study suggests that Omentin-1 could be a promising therapeutic target for metabolic disorders and a valuable biomarker for assessing metabolic health [53].

The exact mechanism for exercise-induced increases in the level of Omentin-1 has not been acknowledged yet. Nonetheless, some main possibilities could be suggested for the association between serum Omentin-1 concentrations and exercise training in our study. Firstly, Omentin-1 concentrations have been reported to correlate with improved plasma lipids due to weight loss [54] and/or physical exercise [31]. In this context, our results reveal that exercise training regimens were associated with significant improvements in lipid profiles. Omentin has been demonstrated to enhance the phosphorylation of 5-AMP-activated protein kinase, which inhibits the synthesis of endogenous cholesterol [55]; therefore, it seems that Omentin-1 has a remarkable role in regulating lipid metabolism and also acts against diabetic dyslipidemia as a compensatory mechanism [56]. Moreover, Omentin-1 was suggested to have an anti-atherogenic behavior; as a result, it can affect the level of high-density lipoprotein by modulating insulin action [19, 57]. Secondly, it has been stated that an improvement in body composition parameters is one other of the probable mechanisms to explain the Omentin-1 increase following exercise training [30, 56], which was observed in our study. In this regard, studies have established that weight management or loss through lifestyle interventions (exercise training along with dietary modification) meaningfully resulted in increased basal Omentin-1 concentrations in a range of populations, accompanied by additional improvements in body composition and metabolic profile [30, 31, 56]. Nevertheless, Wilms and colleagues (2015) observed an increase in Omentin-1 concentrations without a profound change in body weight [31]. On the other hand, the results of Faramarzi et al. (2015), one of the included studies in our meta-analysis, demonstrated significant decreases in BMI and body fat without profound alterations in plasma levels of Omentin-1, glucose, insulin, and HOMA-IR [32]. These controversial findings may reveal that Omentin-1 secretion is very sensitive to exercise and may rise even without the induction of a negative energy balance. In addition, other explanations for the inconsistency in these findings might derive from dissimilarities in the applied exercise training protocols (i.e., intensity, duration, and modality). Undoubtedly, these ideas need additional examination.

Another explanation is that weight loss improves insulin sensitivity, which leads to an increase in Omentin-1 as it has been evidenced that hyperinsulinemia is an inhibitor of Omentin production [58]. A number of studies have verified the function of Omentin-1 in facilitating insulin signaling through the activation of kinase B protein/Akt and enhancing glucose uptake stimulated by insulin into adipose tissue [59]. It has been shown that Omentin-1 has the potential to enhance glucose metabolism and insulin sensitivity through

the facilitation of glucose transport into the muscles subsequent to exercise training [13]. The study conducted by Castro et al. (2019) revealed a relationship between omentin-1 and skeletal muscle as well as adipocytes [13]. According to Alizadeh et al. (2017), exercise training results in an elevation in omentin gene expression in adipose tissue and thus enhances insulin sensitivity [60]. Moreover, Omentin secretion by adipocytes is related to the physiological adaptation of skeletal muscle to exercise training [31]. Therefore, independently of the insulin effect, exercise training alters circulating Omentin-1 levels and impacts skeletal muscle glucose metabolism via protein kinase B. Nevertheless, additional studies are required to examine Omentin-1 concentrations during chronic exercise training regimens.

The current review has some strengths. First, we adopted a three-step search strategy for four databases, reference lists, specialized journals, and gray literature. Second, we chose only the RCT design due to its credibility. Nevertheless, we believe that there are also some limitations in our study. First, only 10 RCTs with a total of 385 participants were studied. According to this, further trials with large sample sizes are needed to deliver more definitive findings, and the findings should be interpreted with caution. Second, in this review, statistical heterogeneity is evident. Despite the fact that subgroup analyses were undertaken to identify possible sources of heterogeneity, the high levels of heterogeneity cannot be adequately and rationally explained. Third, one of the most essential limitations of our meta-analysis is that we did not register our protocol with PROSPERO, which is the major registration platform for systematic reviews and meta-analyses. Finally, while all the included studies in our meta-analysis used enzyme-linked immunosorbent assay (ELISA) kits to measure serum Omentin-1 concentrations, not all the studies provided detailed information on the features of the ELISA kits used, such as assay range, assay sensitivity, or minimal detectable concentration for Omentin-1. This limitation means that it was not possible to provide a comprehensive comparison of different assay methods used for estimating Omentin-1 in different RCTs, which may affect the comparability of the results across the studies.

Conclusion

The overall conclusion of this meta-analysis was that exercise training interventions raised serum Omentin-1, reduced glucose, insulin, body weight, BMI, and body fat, as well as improved lipids and insulin resistance. Nevertheless, isolated aerobic and resistance exercise interventions significantly affected the serum levels of Omentin-1, but not combined aerobic plus resistance exercises. Further high-quality investigations are required to clarify the mechanisms underlying the influence of exercise training on Omentin-1 concentrations.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s13410-023-01229-w.

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Data availability As this is a systematic review and meta-analysis, all relevant data are included in the paper.

Declarations

Ethics approval and consent to participate Not applicable.

Conflict of interest The authors report no declarations of interest.

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