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Review

The effects of exercise training on postprandial glycemia and insulinemia in adults with overweight or obesity and with cardiometabolic disorders: A systematic review and meta-analysis

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ABSTRACT

Background: We performed a systematic review and meta-analysis to investigate the effects of exercise training on postprandial glycemia (PPG) and insulinemia (PPI) in adults with overweight or obesity and with cardiometabolic disorders.

Methods: PubMed, Web of Science, and Scopus databases were searched until May 2022 using the key words "exercise," "postprandial," and "randomized control trial" to identify original studies investigating the effects of exercise training on PPG and/or PPI in adults with a body mass indexes (BMI) $\geq$ 25 kg.m^2. Standardized mean differences (SMD) and 95 % confidence intervals (CIs) were calculated using random effects models to calculate effect sizes for outcomes and to generate forest plots. Sub-group analyses and meta-regressions were performed for potential categorical and continuous moderators.

Results: Twenty-nine studies involving 41 intervention arms and 1,401 participants were included in the systematic review and meta-analysis. Overall, exercise training significantly decreased PPG [−0.36 (95 % CI −0.50 to −0.22), p = 0.001] and PPI [−0.37 (95 % CI −0.52 to −0.21), p = 0.001]. Subgroup analyses showed that PPG decreased following both aerobic and resistance training; whereas PPI was reduced following aerobic training, independent of age, BMI, and baseline glucose levels. Meta-regression analyses showed that frequency of exercise sessions, intervention durations, and duration of exercise time, did not moderate the effects of exercise training on PPI or PPG (p > 0.05).

Conclusion: In adults with overweight or obesity and with cardiometabolic disorders, exercise training is effective for reducing PPG and PPI, across ages and BMIs, irrespective of baseline glucose levels and exercise training characteristics.

1. Introduction

Obesity is characterized by the accumulation of excess adipose tissue, and increases the risk of cardiovascular diseases and comorbid conditions such as type 2 diabetes mellitus. Development of these obesity-related diseases increases the risk for premature death [1–3]. Impairment in the regulation of glucose levels following consumption of foods that challenge the system, may be detected prior to observing dysregulation in fasting levels, and therefore may be important to screen for in order to prevent disease and/or severe symptoms of diseases that have already been diagnosed [4]. Elevated postprandial glycemia (PPG), together with postprandial insulinemia (PPI) are important risk factors for obesity-related diseases [5] including cardiovascular diseases in otherwise healthy individuals with obesity, and those with type 2 diabetes mellitus [4,6–8]. Impaired postprandial metabolism is therefore an important therapeutic target for primary and secondary prevention of

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cardiometabolic diseases [4].

Lifestyle interventions designed to promote weight loss through increased energy expenditure and/or reduced energy intake, include exercise training, physical activity interventions, and dietary interventions, can effectively treat and/or prevent obesity and the onset of type 2 diabetes mellitus [9–14]. Exercise training without weight loss may also be effective [15,16] for improving a range of cardiometabolic health markers. These markers include indices such as body composition, visceral fat mass, lipid profiles, markers of inflammation, glucose regulation, and insulin resistance [11,17–25]. Evidence from systematic reviews and meta-analyses indicate significant effects following exercise training for improving markers of metabolic health including fasting glucose, fasting insulin, insulin resistance, basal hepatic insulin sensitivity, and adipose tissue insulin sensitivity [22,25–28]. However, limited meta-analytic data are available regarding the effects of exercise training on PPG and PPI. In adults who are insufficiently active or highly sedentary, increased acute physical activity and high-intensity interval exercise improve PPG and PPI [29–31], and the same is true for chronic exercise training [32]. In this regard, a previous meta-analysis from our research team showed that high intensity interval training (HIIT) is effective for reducing PPG and PPI [32]. However, Khalafi et al. only pooled data from HIIT and did not include other types of training i.e., aerobic, resistance and combined training and individuals with and without obesity [32]. Currently, no meta-analyses have comprehensively investigated the effects of exercise training on postprandial markers of metabolic health, including basal glucose, and insulin, area under the curve (AUC), and 2-hour glucose in overweight adults with cardiometabolic disorders. Furthermore, it is unclear from existing studies, whether there are different effects for aerobic, resistance, and combined training, and whether different exercise characteristics (frequency of exercise sessions per week, intervention durations, and duration of exercise time) may moderate the effects of exercise training on PPG and PPI. Therefore, we completed a systematic review and meta-analysis to determine whether exercise training has beneficial effects on PPG and PPI in adults with overweight or obesity, and with cardiometabolic disorders.

2. Methods

The current systematic review and meta-analysis followed the 27-item Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) (Supplementary Table 1) and the Cochrane Handbook of Systematic Reviews of Interventions guidelines, and was registered prospectively in the International Prospective Register of Systematic Reviews (PROSPERO), ID: CRD42022379050.

2.1. Search strategy

PubMed, Scopus, and Web of Science were searched from inception to May 2022 to identify original articles that compared the effects of exercise training versus a control group on postprandial metabolic markers. The following key words were used to search: (“exercise” or “physical activity” or “exercise training” or “aerobic training” or “resistance training” or “interval training” or “High-intensity interval training” or “concurrent training” or “combined training”) AND (“post*prandial*” or “post prandial” or “post*meal” or “postprandial” or “postprandial glucose” or “postprandial insulin” or “glucose tolerance test” or “glucose tolerance” or “oral glucose tolerance” or “insulin tolerance test” or “insulin tolerance” or “OGTT” or “ITT” or “AUC” or “postprandial lipemia” or “postprandial lipid” or “postprandial lipids” or “postprandial chylomicron” or “postprandial fat oxidation” or “postprandial triglyceridemia” or “postprandial triglyceride” or “postprandial triacylglycerol” or “oral triglyceride tolerance” or “triglyceride tolerance test” or “postprandial lipaemia”) AND (“randomized control trial” OR “randomized clinical trial” OR “randomized” OR “random*”). The synonyms were joined with the Boolean operator “OR”, and the operator “AND” was used to link terms. In PubMed, the limiters of Humans and English language, and in Scopus and Web of Science, the limiters of English language were used. In addition, reference lists of all included studies were manually screened, and an additional search in Google Scholar was performed to ensure no relevant articles were missed. Search term combinations used in all databases are summarized in Supplementary Table 2.

2.2. Eligibility criteria and study selection

Original studies that met the following criteria were included in the meta-analysis: 1) full-text, peer-reviewed articles written in the English language; 2) studies with human subjects with cardiometabolic disorders and ages $\geq$ 18 years; 3) studies that included exercise training versus a control group, with parallel or crossover randomized designs; 4) studies with results providing measures of postprandial metabolic markers including glucose and/or insulin AUC; and 5) studies with intervention durations of $\geq$ 2 weeks. For participants, studies were included where interventions were conducted in adults with cardiometabolic disorders including overweight and obesity (body mass indexes (BMI) $\geq$ 25 kg.m^2 or a mean BMIs $\geq$ 27 kg.m^2), metabolic syndrome, polycystic ovary syndrome, pre-diabetes, and type 2 diabetes mellitus. Exercise training included any types of exercise: aerobic, resistance, and combined training were included and analyzed as subgroups. Other types of exercise such as Pilates, Tai chi, and sport-based activity were also included. There were no limitations regarding the type, intensity, time of each session, or frequency of exercise sessions (per week). Studies were included that measured total or incremental glucose and/or insulin AUC over a duration between 120 and 180 min, with at least three sampling time points following a meal challenge (standardized or mixed meal). Exclusion criteria were non-original studies such as reviews and meta-analyses, acute studies that performed a single session of exercise, and those using healthy participants, or studies including pregnant women with gestational diabetes. Study selection was done independently by two authors (F G and M H S) and any disagreements regarding study inclusion or exclusion were resolved by discussion with another author (M Kh). After removing duplicates, all remaining articles were screened based on titles and abstracts (stage 1), followed by full-text screening (stage 2).

2.3. Data extraction and synthesis

The following data were extracted from all eligible studies: 1) study name and publication year; 2) study design and sample size; 3) participant characteristics including age, BMI, and health statuses; 4) exercise intervention characteristics including type, intensity, time, intervention duration, and frequency of exercise; and 5) outcomes assessment characteristics including meal composition, AUC duration, sampling timing and frequency. In addition, for inclusion in the meta-analysis, means and standard deviations (SD) or mean changes and their SDs, were extracted for main outcomes including glucose and insulin AUC. For studies that provided more than one exercise arm or where exercise was performed in more than one group (such as men compared with women), all groups were included as separate arms, and the sample size for the control group was divided by the number of comparisons. For studies that provided more than one intervention, data from the exercise and control groups (or placebo) were included. For studies that reported both total and incremental AUC, the latter was included. If studies provided individual time-point data, incremental AUC was calculated. When required, data were extracted from published figures. In addition, when necessary, means and standard deviations were calculated from standard errors, confidence intervals, medians, ranges, and/or interquartile ranges. The corresponding authors of studies that were published in the last five years were contacted when data were missing or insufficient data were reported for the meta-analysis. Two authors (F G and M H S) independently extracted the data and any disagreements were resolved

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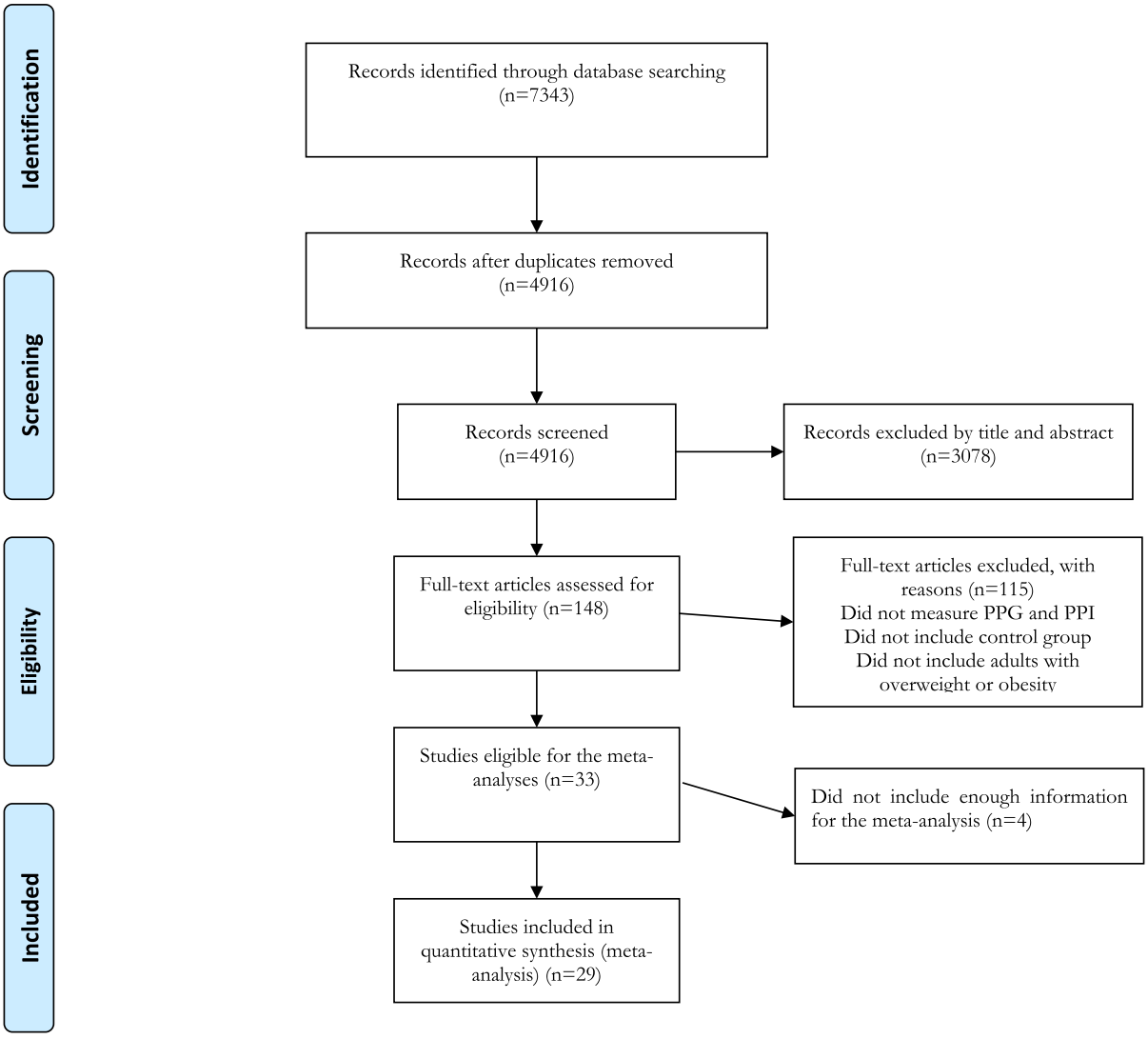

Fig. 1. Flow diagram of systematic literature search.

by discussion with another author (M Kh).

2.4. Quality assessment and sensitivity analyses

We used the TESTEX Scale to assess the quality of all included studies. This tool consists of a 15-point scale (5 points for study quality and 10 points for reporting), with scores ranging from 0 to 15. The study quality items include: eligibility criteria specified, randomization specified, allocation concealment, groups similar at baseline, and assessors blinded for at least one key outcome. The study reporting items include: outcome measures assessed in 85 % of participants, intention-to-treat analysis, reporting of between-group statistical comparisons, point measures and measures of variability reported, activity monitoring in control groups, relative exercise intensity remained constant, and exercise volume and energy expenditure [33].

Two authors (F G and M H S) independently assessed the quality of included studies and any disagreements were resolved through discussions with another author (M Kh). In addition, sensitivity analyses were conducted by removing individual studies to ensure that the direction and/or magnitude of the results were not affected by a single study.

2.5. Meta-analysis

To investigate the effects of exercise training on PPG and PPI, standardized mean differences (SMD) and 95 % confidence intervals (CIs) were calculated using random-effects models. The pre- and post-intervention means or change scores, and their standard deviations and sample sizes were used to determine SMD and 95 % CIs for each analysis. It was necessary to determine SMD because outcomes were reported using different units. Random-effects models were used because heterogeneity was expected given clinical and methodological variability, and this expected heterogeneity may have affected the results [34]. Several sub-group analyses were conducted based on baseline glucose levels using the categories of normal glucose: <5.6 mmol/L for fasting glucose, or <7.8 mmol/L for 2-h post-load glucose; and impaired glucose: ≥5.6 mmol/L for fasting glucose, or ≥7.8 mmol/L for 2-h post-load glucose. Sub groups for BMI were defined as: non-obese: <30 kg/m^2 or obese: ≥30 kg/m^2. For age, we created subgroups for ages <50yrs and ages ≥50yrs. Finally, we determined sub groups for exercise type as: aerobic, resistance, and combined training. Effect sizes were considered small when they ranged from 0.2 to 0.49, moderate for effects that were 0.50–0.79, and large for effects that were ≥0.80. Heterogeneity was assessed using the I^2 statistic, where I^2 was considered small when I^2 was ≤25 %, moderate when I^2 ranged from 25 to ≤50 %, high for I^2 values

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Table 1
Participants and test meal characteristics.

Source, year	Sample size	Biological sex (female/male)	Health status	Mean age (years)	Mean BMI (kg/m^2)	Test meal	Postprandial time (min)	Outcomes
Blumenthal et al., 2000 [64]	78	F and M	Overweight and mild hypertension	Ex:46.6 ± 1.2 CON:47.2 ± 1.8	Ex:32.8 ± 4.0 CON:32.6 ± 5.1	75 g glucose	180	PPG PPI
Bouchonville et al., 2014 [65]	55	F and M	Older adults with obesity	Ex:70.0 ± 4.0 CON:69.0 ± 4.0	Ex:36.9 ± 5.4 CON:37.3 ± 4.7	75 g glucose	120	PPG PPI
Brown et al., 2009 [66]	37	F	Adults with polycystic ovary syndrome	Ex:36.5 ± 5.0 CON:28.0 ± 11.0	Ex:37.9 ± 9.6 CON:31.3 ± 14.9	75 g glucose	–	PPG PPI
Cassidy et al., 2016 [67]	28	F and M	Adults with Type 2 diabetes mellitus	Ex:61.0 ± 9.0 CON:59.0 ± 9.0	Ex:31.0 ± 5.0 CON:32.0 ± 6.0	75 g OGTT	120	PPG
Chen et al., 2021 [68]	46	F and M	Middle-aged adults with obesity	Ex:58.3 ± 4.2 CON:58.3 ± 4.2	Ex:28.1 ± 3.1 CON:27.6 ± 3.2	75 g glucose	120	PPG PPI
Connolly et al., 2016 [35]	62	F	Adults with mild hypertension and overweight	Ex_1:44.0 ± 5.0 Ex_2:46.0 ± 4.0 CON:45.0 ± 4.0	–	–	120	PPG PPI
Cox et al., 2004 [69]	30	M	Adults with obesity	Ex:43.0 ± 4.2 CON:43.9 ± 4.5	Ex:30.5 ± 3.9 CON:29.7 ± 3.5	75 g glucose	120	PPG PPI
DiPietro et al., 2008 [70]	20	F	Older adults with overweight	Ex:72.0 ± 3.0 CON:77.0 ± 6.0	Ex:27.6 ± 3.5 CON:28.6 ± 5.1	75 g glucose	180	PPG PPI
Donges et al., 2013 [36]	47	M	Adults with obesity	Ex_1:45.4 ± 6.1 Ex_2:51.7 ± 7.6 Ex_3:46.2 ± 5.0 CON:49.5 ± 7.4	Ex_1:32.0 ± 4.7 Ex_2:29.7 ± 3.2 Ex_3:30.2 ± 2.5 CON:29.6 ± 5.9	75 g glucose	120	PPG PPI
Dunstan et al., 1998 [71]	27	F and M	Adults with non-insulin-dependent diabetes mellitus	Ex:50.3 ± 6.6 CON:51.1 ± 7.0	Ex:28.3 ± 3.0 CON:30.1 ± 3.3	75 g glucose	120	PPG PPI
Hallsworth et al., 2011 [72]	21	F and M	Adults with non-alcoholic fatty liver disease	Ex:52.0 ± 13.3 CON:62.0 ± 7.4	Ex:32.3 ± 4.9 CON:32.3 ± 4.8	75 g glucose	120	PPG
Hallsworth et al., 2015 [73]	29	F and M	Adults with non-alcoholic fatty liver disease	Ex:54.0 ± 10.0 CON:52.0 ± 12.0	Ex:31.0 ± 4.0 CON:31.0 ± 5.0	75 g glucose	120	PPG
Houghton et al., 2017 [74]	26	F and M	Adults with non-alcoholic steatohepatitis	Ex:54.0 ± 12.0 CON:51.0 ± 16.0	Ex:33.0 ± 7.0 CON:33.0 ± 5.0	75 g glucose	120	PPG
Karstoft et al., 2013 [37]	32	F and M	Adults with Type 2 diabetes mellitus	Ex_1:60.8 ± 7.6 Ex_2:57.5 ± 8.3 CON:57.1 ± 8.5	Ex_1:29.9 ± 5.5 Ex_2:29.0 ± 4.5 CON:29.7 ± 5.4	75 g glucose	120	PPG
Keadle et al., 2014 [75]	30	F and M	Adults with overweight and obesity	Ex:43.9 ± 9.7 CON:42.7 ± 10.1	Ex:35.2 ± 5.3 CON:35.3 ± 5.2	75 g glucose	120	PPG PPI
Lehrskov et al., 2019 [76]	34	F and M	Adults with abdominal obesity	Ex:39.0 ± 13.0 CON:48.0 ± 12.0	–	Mixed meal (15 % fat, 20 % protein, and 65 % carbohydrat)	180	PPG PPI

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Table 1 (*continued*)

Source, year	Sample size	Biological sex (female/male)	Health status	Mean age (years)	Mean BMI (kg/m^2)	Test meal	Postprandial time (min)	Outcomes
Mendham et al., 2015[38]	33	M	Adults with obesity	Ex_1:46.8 ± 6.6 Ex_2:49.5 ± 6.6 CON:49.2 ± 7.0	Ex_1:27.6 ± 2.9 Ex_2:29.1 ± 3.8 CON:29.5 ± 3.2	75 g glucose	120	PPG PPI
Mendham et al., 2015 [77]	33	M	Adults with obesity	Ex:39.5 ± 10.6 CON:36.1 ± 16.1	Ex:31.6 ± 3.1 CON:34.5 ± 6.6	75 g glucose	120	PPG PPI
Nygaard et al., 2017 [78]	52	F and M	Individuals with high-risk for type 2 diabetes and with obesity	Ex:53.0 ± 9.0 CON:46.0 ± 8.0	Ex:26.3 ± 2.4 CON:27.4 ± 4.5	75 g glucose	120	PPG PPI
Potteiger et al., 2002 [79]	27	–	Adults with overweight	–	–	75 g glucose	180	PPG PPI
Potteiger et al., 2003 [43]	66	F and M	Young adults with overweight	17.0–35.0	25.0 ± 34.9	75 g glucose	180	PPG PPI
Reichkendler et al., 2014 [39]	61	M	Young adults with overweight	Ex_1:30.0 ± 6.0 Ex_2:28.0 ± 4.2 CON:31.0 ± 4.1	Ex_1:28.6 ± 2.7 Ex_2:27.6 ± 2.3 CON:28.0 ± 3.2	75 g glucose	120	PPG PPI
Roberts et al., 2013 [80]	49	M	Adults with overweight and obesity	Ex:21.5 ± 2.3 CON:21.9 ± 1.8	Ex:31.1 ± 2.3 CON:33.2 ± 3.2	75 g glucose	120	PPG PPI
Ross et al., 2000 [40]	52	M	Adults with obesity	Ex_1:45.0 ± 7.5 Ex_2:44.7 ± 7.6 CON:46.0 ± 10.9	Ex_1:32.3 ± 1.9 Ex_2:31.3 ± 2.3 CON:30.7 ± 1.6	75 g glucose	120	PPG PPI
Ross et al., 2015 [41]	300	F and M	Adults with obesity	Ex_1:52.1 ± 7.4 Ex_2:50.9 ± 8.6 Ex_3:50.3 ± 8.1 CON:52.2 ± 8.2	Ex_1:33.7 ± 4.4 Ex_2:33.5 ± 4.9 Ex_3:33.4 ± 4.3 CON:33.1 ± 4.6	75 g glucose	120	PPG PPI
Ross et al., 2004 [15]	54	F	Adults with obesity	Ex_1:43.2 ± 5.1 Ex_2:41.3 ± 7.2 CON:43.7 ± 6.4	Ex_1:32.8 ± 3.9 Ex_2:32.9 ± 3.1 CON:32.4 ± 2.8	75 g glucose	120	PPG PPI
Suntisawee et al., 2021 [81]	16	F and M	Adults with obesity with impaired glucose tolerance	Ex:50.4 ± 1.6 CON:50.4 ± 2.3	Ex:27.7 ± 0.5 CON:27.7 ± 0.8	75 g glucose	120	PPG
Tessier et al., 2000 [82]	39	F and M	Adults with Type 2 diabetes mellitus	Ex:69.3 ± 4.2 CON:69.5 ± 5.1	Ex:30.7 ± 5.4 CON:29.4 ± 3.7	75 g glucose	180	PPG PPI
Winding et al., 2018 [42]	29	F and M	Adults with Type 2 diabetes mellitus	Ex_1:58.0 ± 8.0 Ex_2:54.0 ± 6.0 CON:57.0 ± 7.0	Ex_1:27.4 ± 3.1 Ex_2:28.1 ± 3.5 CON:28.0 ± 3.5	75 g glucose	120	PPG

Abbreviations: F female, M male, PPG postprandial glucose, PPI postprandial insulin, Ex exercise, CON control, OGTT oral glucose tolerance test

ranging from 50 to ≥75 %, and considerable when I^2 was >75 %). In addition, meta-regression analyses were performed for frequency of exercise sessions per week, intervention durations, and duration of exercise time (min/week). Publication bias was assessed using visual interpretation of funnel plots and Egger's tests. Significance for effect size and heterogeneity were considered at $p < 0.05$, and for Egger's tests at $p < 0.1$. The trim and fill method was used when publication bias was identified by visual interpretation of funnel plots.

3. Results

3.1. Search results

The initial database searches identified 7,343 records, of which 4,916 remained after removing duplicates. Subsequently, 148 studies remained after title and abstract screening, and of those, 29 met all inclusion criteria. Reasons for removal of 108 articles are presented in Fig. 1. Nine studies [15,35–42] included more than one exercise arm,

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Table 2
Intervention Characterisistics.

Source, year	Exercise Mode	Exercise protocol	Control protocol	Session per week	Intervention duration
Blumenthal et al., 2000 [64]	Aerobic	35-min at 70–85 % of initial HRR by cycle ergometry and walking	Maintain usual dietary and exercise habits	3–4	6 months
Bouchonville et al., 2014 [65]	Combined	AT: 30-min at 65–85 % of HR_{peak} by treadmill, stationary cycling and stair climbing RT: Whole body exercises; 1–8 sets with 8–12 reps at 65–85 % of 1RM	Received general information regarding a healthy diet, and were prohibited from participating in diet or exercise programs	3	12 months
Brown et al., 2009 [66]	Aerobic	60-min at 40–60 % peak VO2 by treadmill, elliptical machine or stationary cycle	No change in lifestyle	–	16–24 weeks
Cassidy et al., 2016 [67]	Aerobic	HIIT: 5 sets of 120–230 s at a rating of 16–17 BRPE by 3-min recovery	Standard care	3	12 weeks
Chen et al., 2021 [68]	Aerobic	HIIT: 5 set of 3-min at 90 % of HR_{peak} by 3-min recovery at 70 % of HR_{peak}	Maintain lifestyle	–	12 weeks
Connolly et al., 2016 [35]	Aerobic	HIIT: 6 to 10 sets of 30-s by 2-min passive recovery by free-style swimming AT: 60-min at low intensity consisted of continuous front-crawl swimming	No training or lifestyle changes in the same period	3	15 weeks
Cox et al., 2004 [69]	Aerobic	30-min at 60–70 % of maximum workload by stationary cycling	The light exercise protocol consisted of a series of slow flexibility exercises once per week and stationary cycling (against zero resistance) twice a week. Every second week, subjects substituted one cycling session for a session where they walked slowly, at a rate of $\approx \leq$ 2 km in 30 min.	3	16 weeks
DiPietro et al., 2008 [70]	Aerobic & Resistance	AT: 60-min at 65–75 % VO2peak by treadmills	RT: 45-min at 45–50 % VO2peak by Thera-Bands, Thera Balls, and hand weights	4	6 months
Donges et al., 2013 [36]	Aerobic & Resistance & Combined	AT: 40–60 min at 75–80 % HR_{max} by cycling RT: Whole body exercises; 10 exercises, 3–4 sets at 8–10 reps at 75–80 % of 1RM CT: 1.5–2 sets at 8–10 reps at 75–80 % of 1RM followed by 20–30 min at 75–80 % of HR_{max}	Nonexercising, maintained diet and physical activity patterns	3	12 weeks
Dunstan et al., 1998 [71]	Resistance	2–3 sets of 60-min at 50–55 % of 1RM	Non-exercise	3	8 weeks
Hallsworth et al., 2011 [72]	Resistance	Whole body exercises; 8 exercises, 45–60 min at 50–70 % of 1RM by resistance exercise	Continued normal treatment	3	8 weeks
Hallsworth et al., 2015 [73]	Aerobic	HIIT: 5 sets of 120–230 s at 16–17 BRPE by 3-min recovery with cycle ergometry	Standard care continuing any prescription medication	3	12 weeks
Houghton et al., 2017 [74]	Combined	45–60 min, aerobic 3 intervals at 16–18 BRPE followed by a resistance exercise including 5 exercises at 14–16 BRPE by a fixed bike	Standard care	3	12 weeks
Karstoft et al., 2013 [37]	Aerobic	60-min at 55 % of the peak energy-expenditure rate by continuous-walking 60-min at 70 % of the peak energy-expenditure rate, alternated 3-min repetitions at low and high intensity, consisting of cycles of 3-min of fast walking (above the target) and 3-min of slow walking (below the target)	Continue their habitual lifestyle	5	4 months
Keadle et al., 2014 [75]	Aerobic	40-min at 40–65 % of HRR by Treadmill or stationary cycle ergometer or Arctrainer.	Maintain behavior and their current level of activity	5	12 weeks
Lehrskov et al., 2019 [76]	Aerobic	HIIT: 45-min high-intensity interval training by an ergometer bicycle	No exercise	3	12 weeks
Mendham et al., 2015 [38]	Aerobic	SSG: 46–56 min in four quarters at 80–85 % of HR_{max} by 2-min passive recovery by small-sided games HIIT: four quarters at 80–85 % of HR_{max} by 2-min passive recovery by continuous stationary cycling	Maintained normal activity and dietary patterns	3	8 weeks
Mendham et al., 2015 [77]	Aerobic	45–60 min at 70–85 % of HR_{max} by sports-based exercise	Maintained normal activity and dietary patterns	2–3	12 weeks
Nygaard et al., 2017 [78]	Aerobic	A minimum 30-min of daily light physical activity	Maintained their usual lifestyle	7	12 weeks
Potteiger et al., 2002 [79]	Aerobic	30–45 min at 65 %-75 % of HRR	Continued with normal daily activities	3–5	9 months
Potteiger et al., 2003 [88]	Aerobic	20–45-min at 60–75 % of HRR	Non-exercise	3–5	16 months
Reichkendler et al., 2014 [39]	Aerobic	aerobic exercise at 50–70 % of $VO2_{max}$ until 300–700 kcal/day	Maintain their sedentary lifestyle	3	11 weeks
Roberts et al., 2013 [80]	Resistance	Whole body exercises; 2–3 sets with 6–15 reps at 100 % of approximated 6–15 RM.	No training	3	12 weeks
Ross et al., 2000 [40] AUC	Aerobic	asked to maintain the isocaloric diet for the duration of the treatment period and to perform exercise that expended 700 kcal/d at at an intensity not>80 % of HRmax by brisk walking or light jogging an or on a motorized treadmill	to maintain body weight throughout the 12-week treatment period	–	12 weeks

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Table 2 (*continued*)

Source, year	Exercise Mode	Exercise protocol	Control protocol	Session per week	Intervention duration
		asked to maintain body weight and they consumed enough calories to compensate for the energy expended during the daily exercise sessions (approximately 700 kcal).			
Ross et al., 2015 [41]	Aerobic	180–300 kcalat 50 % of $VO2_{peak}$ by walking or light jogging 360–600 kcal at 50 % of $VO2_{peak}$, walking or light jogging 360–600 kcal at 75 % of $VO2_{peak}$, walking or light jogging	No exercise and maintain their level of physical activity	5	24 weeks
Ross et al., 2004 [15] AUC	Aerobic	asked to maintain the isocaloric diet for the duration of the treatment and daily exercise wherein the energy expended was 500 kcal/d at 80 % of HRmax by brisk walking or light jogging an or on a motorized treadmill asked to maintain body weight and they consumed the calories required to compensate for the energy expended during the daily exercise sessions (500 kcal) at 80 % of HRmax by brisk walking or light jogging an or on a motorized treadmill	asked to maintain body weight	–	14 weeks
Suntisawee et al., 2021 [81]	Resistance	50-min functional exercise training in a circuit manner, A circuit consisted of 12 exercises, to be performed consecutively with 60 s of rest in between each exercise	Carried out normal daily physical activity, including walking.	5	12 weeks
Tessier et al., 2000 [82]	Combined	40-min at 30–74 % of $VO2_{max}$ by rapid walk and, whole major muscle exercises 2 sets with 20 reps	Continue with their usual activity regimen	3	16 weeks
Winding et al., 2018 [42]	Aerobic	AT: 40-min at 50 % of W_{peak} by cycling HIIT: 10 × 1-min at 95 % of W_{peak} and 1-min recovery at 20 % of W_{peak}	No training	3	11 weeks

Abbreviations: W_{peak} peak workload, HIIT high intensity interval training, $VO2_{max}$ maximal oxygen consumption, $VO2_{peak}$ peak oxygen uptake, HR_{max} maximum heart rate, AT aerobic training, RT resistance training, CT combined training, Reps repetitions, 1RM one repetition maximum, SSG small-sided games, HRR heart rate reserve, BRPE borg rating of perceived exertion

and one study [43] included male and females in separate study arms. Finally, 29 studies (randomized control trials), involving 41 intervention arms, were included the meta-analysis.

3.2. Participants, interventions and outcome characteristics

A total 1,413 participants were included in the meta-analysis, with ages and BMIs ranging from ~22 to 72 years and 27 to 38 $kg.m^2$, respectively. A majority of studies included both female and male participants, four included only females, and seven included only males. One study did not clearly report the biological sex of participants. All included studies recruited individuals with BMIs > 25 $kg.m^2$ and with co-morbidities including hypertension, polycystic ovary syndrome, type 2 diabetes mellitus, and fatty liver diseases. For exercise training, the intervention durations ranged from 2 to 16 months, and the exercise frequency ranged from 3 to 5 sessions per week. A majority of studies used aerobic exercise as either continuous or interval training such as walking, running or cycling. Other studies used resistance or combined training. Glucose and insulin AUC outcomes were calculated following an oral glucose tolerance test (OGTT) using 75 g glucose, with one study using a mixed meal challenge instead of OGTT. The assessment period durations were between 120 and 180 min. More details regarding participants, exercise interventions, and outcomes assessments are presented in Tables 1 and 2.

4. Meta-analysis

4.1. PPG

Thirty-nine intervention arms demonstrated that overall, exercise training decreases PPG [SMD: −0.36 (95 % CI −0.50 to −0.22), p = 0.001] (Fig. 2). There was no heterogeneity amongst studies (I^2 = 14.90 %, p = 0.21), but visual interpretation of funnel plots suggested publication bias, which was confirmed by the Egger's test (p = 0.005). The trim and fill method identified nine missing studies from the right side of the plots. When the missing studies were accounted for, the SMD was −0.24 (95 % CI −0.41 to −0.07). Subgroup analyses (Table 3) showed that PPG decreased in adults < 50yrs. (SMD: −0.39, p = 0.001), and in adults ≥ 50yrs (SMD: −0.39, p = 0.001); for participants with normal glucose at baseline (SMD: −0.30, p = 0.001), and impaired glucose (SMD: −0.52, p = 0.002); with BMIs indicating overweight (SMD:-0.48, p = 0.001), and obesity (SMD:-0.31, p = 0.001); and for aerobic training (SMD:-0.32, p = 0.001) and resistance training (SMD:-0.95, p = 0.006). In addition, meta-regression analyses showed that the frequency of exercise sessions per week, intervention durations, and duration of exercise time, did not moderate the effects of exercise training on PPG (p > 0.05) (Supplementary Table 4).

4.2. PPI

Thirty-one intervention arms demonstrated that exercise training decreased PPI [SMD: −0.37 (95 % CI −0.52 to −0.21), p = 0.001] (Fig. 3). There was no significant heterogeneity amongst studies (I^2 = 23.98 %, p = 0.11), and visual interpretation of funnel plots suggested publication bias, which was confirmed by the Egger's test (p = 0.03). The trim and fill method identified 12 missing studies from the right side of the plots that when accounted for, changed the SMD to −0.13 (95 % CI −0.31 to −0.04). Subgroup analyses (Table 3) showed that PPI decreased in adults < 50 yrs. (SMD: −0.38, p = 0.001); for participants with normal glucose at baseline (SMD: −0.30, p = 0.001); for participants with BMIs either indicating overweight (SMD:-0.38, p = 0.002), or obesity (SMD:-0.38, p = 0.001); and following aerobic training (SMD:-0.39, p = 0.001). In adults ≥ 50yrs, decreases in PPI approached significance (SMD: −0.34, p = 0.05), and with the same is true for participants with impaired glucose at baseline (SMD: −0.33, p = 0.06). In addition, meta-regression analyses showed that the frequency of exercise sessions per week, intervention durations, and duration of exercise time, did not moderate the effects of exercise training on PPI (p > 0.05) (Supplementary Table 4).

4.3. Quality assessment and sensitivity analyses

The overall quality assessment scoring is provided in Supplementary Table 3. Briefly, scores ranged from seven to 13 out of a maximum score of 15. In addition, sensitivity analysis showed that omitting individual studies did not change the direction or significance of the results.

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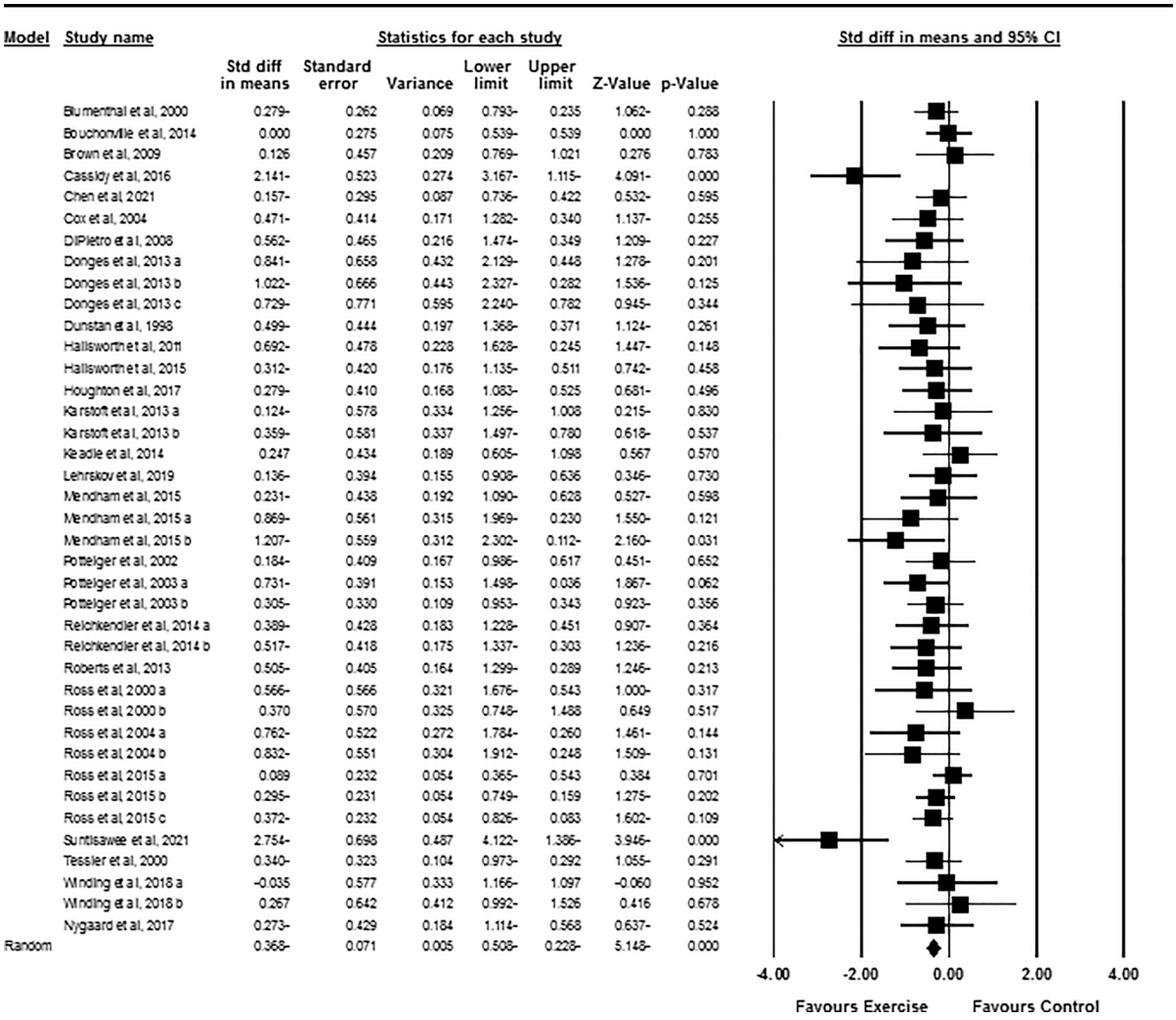

Fig. 2. Forest plot of the effects of exercise training compared with control on PPG. Data are reported as SMD (95 % confidence limits). SMD: standardized mean difference, PPG: postprandial glycemia.

5. Discussion

We demonstrate that exercise training is effective for reducing both PPG and PPI. In addition, results suggested that aerobic-based training is effective for reducing PPG and PPI, and resistance training reduced PPG. The current results demonstrated that the exercise training benefits on PPG and PPI may occur in adults with overweight or obesity, and in those with normal and impaired glucose levels, regardless of age.

Previously published meta-analyses have suggested that exercise training is effective for improving cardiometabolic health. These meta-analyses have mainly focusing on lipid profiles, blood pressure, inflammatory markers, and fasting glycemic markers such glucose, insulin, HbA1c, or insulin resistance index [44-47]. However, limited studies have focused on postprandial glycemic and insulinemic outcomes. Current meta-analyses have also suggested that a single bout of exercise may be effective for improving PPG and PPI [48], and the beneficial effect of chronic exercise has only been reported following high-intensity interval training [32]. While exercise training has been shown to improve 2-h OGTT, any effects in individuals with prediabetes are unclear [49]. This research gap is important since previous research has indicated that postprandial responses are more import than after fasting [4] and increasing evidence suggest it is an independent risk factor for CVD [4,6,8] and better predicts mortality [50]. We therefore provide clinically important findings indicating that exercise training is effective for reducing PPG and PPI in adults with overweight and obesity, and with cardiometabolic disorders. These results are in agreement our pervious meta-analyses that included participants with and without metabolic disorders, and indicated that HIIT is effective for reducing PPG and PPI, particularly if glucose is abnormal [32]. This reduction in PPG and PPI is at least a partial reflection of the beneficial effects of exercise on body composition, including reductions in visceral fat mass, increases in skeletal muscle glucose uptake, and controlling hepatic glucose production. Adipose tissue, particularly visceral fat mass, is associated with increased release of free fatty acids, and also secretes several adipokines which are important regulators of insulin resistance in the liver and muscle [51,52]. In addition, skeletal muscle is the main site for glucose uptake during postprandial periods [52,53] meaning exercise can increase insulin sensitivity, plus glucose transporter translocation and activity. Furthermore, skeletal muscle morphological changes, including increased capillary density, may also be associated with improving glycemia [54]. Exercise training may also improve postprandial glycemia via improving insulin sensitivity in the liver, and through regulation of gluconeogenesis [55].

We investigated whether exercise training is effective in adults with overweight or obesity who had normal or impaired glucose levels. Previous reviews have suggested that exercise training is associated with a greater reduction in PPG in adults with impaired fasting glucose [32]; however, we suggested that exercise training is effective for reducing PPG and PPI in individuals with cardiometabolic disorders regardless of baseline glucose levels. These different results may be due to the health

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Table 3
Summary of subgroup analyses.

Marker	Moderators	Trials	SMD (95 % CI)	P-value	P-heterogeneity
PPG					
Mean age	≥50 years	17	−0.39 (−0.64 to −0.13)	0.003*	0.01
	<50 years	21	−0.39 (−0.58 to −0.20)	0.001*	0.89
Mean BMI	≥30 kg/m^2	23	−0.31 (−0.48 to −0.14)	0.001*	0.26
	<30 kg/m^2	15	−0.48 (−0.74 to −0.22)	0.001*	0.23
Type of training	Aerobic	31	−0.32 (−0.45 to −0.18)	0.001*	0.52
	Resistance	5	−0.95 (−1.64 to −0.27)	0.006*	0.06
	Combined	3	−0.13 (−0.56 to 0.29)	0.52	0.61
Glucose levels	Fasting < 5.6 or 2-h < 7.8 mmol/L	24	−0.30 (−0.45 to −0.15)	0.001*	0.76
	Fasting ≥ 5.6 or 2-h ≥ 7.8 mmol/L	14	−0.52 (−0.86 to −0.18)	0.002*	0.02
PPI					
Mean age	≥50 years	8	−0.34 (−0.68 to −0.001)	0.05	0.007
	<50 years	22	−0.38 (−0.57 to −0.20)	0.001*	0.56
Mean BMI	≥30 kg/m^2	19	−0.38 (−0.60 to −0.15)	0.001*	0.03
	<30 kg/m^2	11	−0.38 (−0.61 to −0.14)	0.002*	0.54
Type of training	Aerobic	26	−0.39 (−0.57 to −0.20)	0.001*	0.04
	Resistance	3	−0.31 (−0.84 to 0.22)	0.25	0.67
	Combined	2	−0.24 (−0.75 to 0.25)	0.33	0.48
Glucose levels	Fasting < 5.6 or 2-h < 7.8 mmol/L	26	−0.39 (−0.57 to −0.20)	0.001*	0.06
	Fasting ≥ 5.6 or 2-h ≥ 7.8 mmol/L	4	−0.33 (−0.68 to 0.02)	0.06	0.49

status of participants as all our participants were overweight disorder despite normal glucose, where previous meta-analysis included healthy people. Obesity is related to increased circulating insulin during the postprandial period, due to impaired insulin clearance and increased secretion, even and in the absence of insulin resistance [56-58]. Therefore, it is not surprising that exercise can reduce PPG and PPI in people with overweight and obesity who have normal glucose levels, which may be due to improved insulin secretion and action. Subgroup analyses confirmed that exercise is effective for reducing PPG and PPI, regardless of overweight or obese status. However, it should be noted that all included studies had participants with mean BMIs ≥ 27 kg.m^2 and in both subgroups, and therefore these results cannot be generalized to adults with obesity who have lower BMIs. In addition, aging is a major risk factor for elevated fasting and PPG, in part this increased risk is associated with deficits in insulin action, secretion, and clearance [59]. However, the current results suggested that exercise training is effective for reducing PPG and PPI in ages <50yrs as well as ages ≥50yrs. Reduction in insulin action with aging are related to overall body fat and visceral fat, β-cell function, hepatic insulin action, and total body insulin clearance [59], and exercise training may mitigate these age-related deficits. Furthermore, we performed subgroup analyses based on exercise type, and results suggested that aerobic-based training is an effective training mode for reducing both PPG and PPI, where resistance training is effective for reducing PPG, and combined training was not effective for reducing either PPG or PPI. It should be noted, however, that the number of studies in these subgroups varied, and there were limited studies in the resistance and combination training subgroups. Aerobic training involves continuous or interval-based activity using multiple muscles and is an effective training method for reducing fat mass and visceral fat, and for improving insulin resistance and liver metabolism [11,60,61]. Therefore, it is not surprising that aerobic training improves PPG and PPI. The effects of resistance training on PPG may be mediated by increases in lean body mass, which lead to subsequent increases in tissue volume for glucose disposal, as well as decreases in visceral fat [20,62,63]. Surprisingly, however, combined training did not lead to significant changes in PPG and PPI, although the small number of studies did not allow further investigation. In addition, in the meta-regression, no significant moderating effects were observed for exercise characteristics. Due to the lack of significant heterogeneity, the meta-regression results could be considered as expected; exercise training is effective for improving glycaemia regardless of specific exercise characteristics.

To the best of our knowledge, this is the largest meta-analysis to date to have assessed the impact of exercise training modalities on postprandial glycemia markers i.e., PPG and PPI in adults with overweight or obesity, and with cardiometabolic disorders. Although the effect sizes in the current meta-analysis were small, our results confirm the importance of exercise training for improving PPG and PPI. These findings may be clinically important, and from a practical point of view highlight the role of exercise training for people with metabolic disorders, especially since PPG and PPI are important independent risk factors for cardiovascular diseases. The impact of weight loss and nutritional modifications, along with exercise training, either alone or in combination should be investigated in future studies, since combined interventions may be more effective. However, our study had several limitations that should be considered when interpreting the results. There were limited studies in some subgroups that may affect the results, and although we performed subgroup analysis based on exercise type, it was not designed to study the effect of exercise intensity, duration and volume. These exercise training variables require further investigation.

6. Conclusion

The current results provide evidence that exercise training is an effective strategy for improving PPG and PPI in adults with overweight or obesity, and with cardiometabolic disorders. The beneficial effects of exercise occurred in adults with both normal and impaired glucose levels, aged over and under 50 years, and with both overweight and obesity. Aerobic training seems to be an effective training mode for improving both PPG and PPI, and resistance training appears to be effective for improving PPG. Exercise training characteristics including frequency of exercise, intervention durations, and duration of exercise time, did not moderate the overall effects of exercise training on PPG and PPI.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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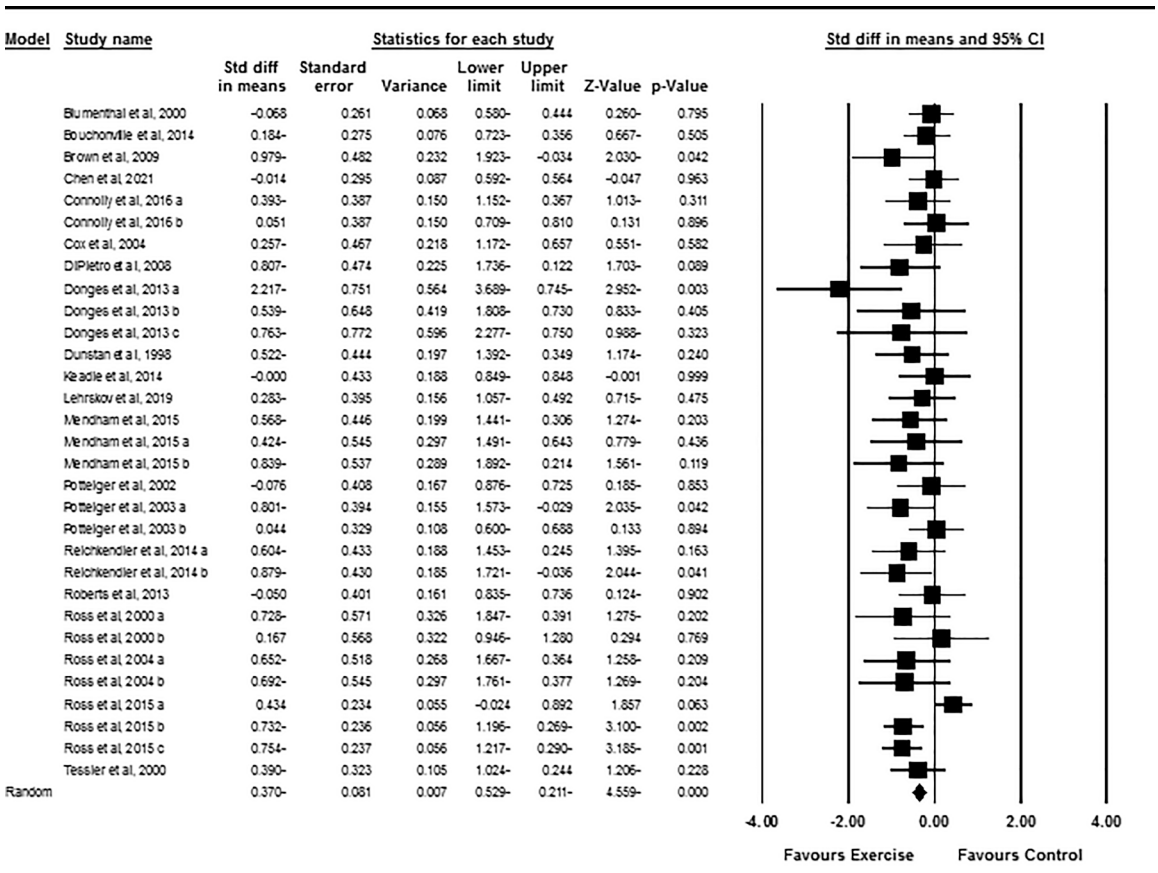

Fig. 3. Forest plot of the effects of exercise training compared with control on PPI. Data are reported as SMD (95 % confidence limits). SMD: standardized mean difference, PPI: postprandial insulinemia.

Data availability

All data generated or analysed during this study are included in this published article and supplementary table and other data can be made available on reasonable request to the corresponding author.

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Author contribution

M Kh conceived and designed the study; analyzed the data and completed the initial draft of the results; drafted the initial manuscript; and approved the final version of the manuscript. M E S conceived and designed the study; revised the manuscript and approved the final version of the manuscript. F G conceived and designed the study; analyzed the data and completed the initial draft of the results; and approved the final version of the manuscript. S K R conceived and designed the study; revised the manuscript; and approved the final version of the manuscript. H R conceived and designed the study; analyzed the data and completed the initial draft of the results; and approved the final version of the manuscript. M H S conceived and designed the study; analyzed the data and completed the initial draft of the results; and approved the final version of the manuscript.

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Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.diabres.2023.110741.

References

[1] Blüher M. Obesity: global epidemiology and pathogenesis. Nat Rev Endocrinol 2019;15(5):288–98.
[2] Chobot A, et al. Obesity and diabetes-Not only a simple link between two epidemics. Diabetes Metab Res Rev 2018;34(7):e3042.
[3] Van Gaal LF, Mertens IL, De Block CE. Mechanisms linking obesity with cardiovascular disease. Nature 2006;444(7121):875–80.
[4] Cavalot F, et al. Postprandial blood glucose is a stronger predictor of cardiovascular events than fasting blood glucose in type 2 diabetes mellitus, particularly in women: lessons from the San Luigi Gonzaga Diabetes Study. J Clin Endocrinol Metab 2006;91(3):813–9.
[5] Blaak EE, et al. Impact of postprandial glycaemia on health and prevention of disease. Obes Rev 2012;13(10):923–84.
[6] Lin HJ, et al. Postprandial glucose improves the risk prediction of cardiovascular death beyond the metabolic syndrome in the nondiabetic population. Diabetes Care 2009;32(9):1721–6.

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[7] Lautt WW. Postprandial insulin resistance as an early predictor of cardiovascular risk. Ther Clin Risk Manag 2007;3(5):761–70.
[8] Cavalot F, et al. Postprandial blood glucose predicts cardiovascular events and all-cause mortality in type 2 diabetes in a 14-year follow-up: lessons from the San Luigi Gonzaga Diabetes Study. Diabetes Care 2011;34(10):2237–43.
[9] Johnson NA, et al. Aerobic exercise training reduces hepatic and visceral lipids in obese individuals without weight loss. Hepatology 2009;50(4):1105–12.
[10] Verheggen R, et al. A systematic review and meta-analysis on the effects of exercise training versus hypocaloric diet: distinct effects on body weight and visceral adipose tissue. Obes Rev 2016;17(8):664–90.
[11] Ismail I, et al. A systematic review and meta-analysis of the effect of aerobic vs. resistance exercise training on visceral fat. Obes Rev 2012;13(1):68–91.
[12] Ditschuneit HH, et al. Metabolic and weight-loss effects of a long-term dietary intervention in obese patients. Am J Clin Nutr 1999;69(2):198–204.
[13] Sackner-Bernstein J, Kanter D, Kaul S. Dietary intervention for overweight and obese adults: comparison of low-carbohydrate and low-fat diets. A meta-analysis. PLoS ONE 2015;10(10):e0139817.
[14] Astrup A, et al. The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies. Int J Obes (Lond) 2000;24(12):1545–52.
[15] Ross R, et al. Exercise-induced reduction in obesity and insulin resistance in women: a randomized controlled trial. Obes Res 2004;12(5):789–98.
[16] Lee S, et al. Exercise without weight loss is an effective strategy for obesity reduction in obese individuals with and without Type 2 diabetes. J Appl Physiol 2005;99(3):1220–5.
[17] Khalafi M, Symonds ME. The impact of high-intensity interval training on inflammatory markers in metabolic disorders: a meta-analysis. Scand J Med Sci Sports 2020;30(11):2020–36.
[18] Shabkhiz F, et al. Resistance training attenuates circulating FGF-21 and myostatin and improves insulin resistance in elderly men with and without type 2 diabetes mellitus: a randomised controlled clinical trial. Eur J Sport Sci 2021;21(4):636–45.
[19] Khalafi M, Symonds ME, Akbari A. The impact of exercise training versus caloric restriction on inflammation markers: a systemic review and meta-analysis. Crit Rev Food Sci Nutr 2022;62(15):4226–41.
[20] Khalafi M, et al. Effect of resistance training with and without caloric restriction on visceral fat: a systemic review and meta-analysis. Obes Rev 2021;22(9):e13275.
[21] Khalafi M, Symonds ME. The impact of high intensity interval training on liver fat content in overweight or obese adults: a meta-analysis. Physiol Behav 2021;236:113416.
[22] Khalafi M, et al. A comparison of the impact of exercise training with dietary intervention versus dietary intervention alone on insulin resistance and glucose regulation in individual with overweight or obesity: a systemic review and meta-analysis. Crit Rev Food Sci Nutr 2022:1–15.
[23] Smart NA, et al. Effect of exercise training on liver function in adults who are overweight or exhibit fatty liver disease: a systematic review and meta-analysis. Br J Sports Med 2018;52(13):834–43.
[24] Fedewa MV, Hathaway ED, Ward-Ritacco CL. Effect of exercise training on C reactive protein: a systematic review and meta-analysis of randomised and non-randomised controlled trials. Br J Sports Med 2017;51(8):670–6.
[25] Battista F, et al. Effect of exercise on cardiometabolic health of adults with overweight or obesity: Focus on blood pressure, insulin resistance, and intrahepatic fat—a systematic review and meta-analysis. Obes Rev 2021;22(S4):e13269.
[26] Sargeant JA, et al. The effect of exercise training on intrahepatic triglyceride and hepatic insulin sensitivity: a systematic review and meta-analysis. Obes Rev 2018;19(10):1446–59.
[27] Snowling NJ, Hopkins WG. Effects of different modes of exercise training on glucose control and risk factors for complications in type 2 diabetic patients: a meta-analysis. Diabetes Care 2006;29(11):2518–27.
[28] Jelleyman C, et al. The effects of high-intensity interval training on glucose regulation and insulin resistance: a meta-analysis. Obes Rev 2015;16(11):942–61.
[29] Loh R, et al. Effects of interrupting prolonged sitting with physical activity breaks on blood glucose, insulin and triacylglycerol measures: a systematic review and meta-analysis. Sports Med 2020;50(2):295–330.
[30] Quan M, et al. Effects of interrupting prolonged sitting on postprandial glycemia and insulin responses: a network meta-analysis. J Sport Health Sci 2021;10(4):419–29.
[31] Khalafi M, et al. High-intensity interval exercise versus moderate-intensity continuous exercise on postprandial glucose and insulin responses: a systematic review and meta-analysis. Obes Rev 2022:e13459.
[32] Khalafi M, et al. The impact of high-intensity interval training on postprandial glucose and insulin: a systematic review and meta-analysis. Diabetes Res Clin Pract 2022:109815.
[33] Smart NA, et al. Validation of a new tool for the assessment of study quality and reporting in exercise training studies: TESTEX. JBI Evid Implement 2015;13(1):9–18.
[34] Tufanaru C, et al. Fixed or random effects meta-analysis? Common methodological issues in systematic reviews of effectiveness. Int J Evid Based Healthc 2015;13(3):196–207.
[35] Connolly LJ, et al. Low-volume high-intensity swim training is superior to high-volume low-intensity training in relation to insulin sensitivity and glucose control in inactive middle-aged women. Eur J Appl Physiol 2016;116(10):1889–97.
[36] Donges CE, et al. Comparative effects of single-mode vs. duration-matched concurrent exercise training on body composition, low-grade inflammation, and glucose regulation in sedentary, overweight, middle-aged men. Appl Physiol Nutr Metab 2013;38(7):779–88.
[37] Karstoft K, et al. The effects of free-living interval-walking training on glycemic control, body composition, and physical fitness in type 2 diabetic patients: a randomized, controlled trial. Diabetes Care 2013;36(2):228–36.
[38] Mendham AE, et al. Rugby-specific small-sided games training is an effective alternative to stationary cycling at reducing clinical risk factors associated with the development of type 2 diabetes: a randomized, controlled trial. PLoS ONE 2015;10(6).
[39] Reichkendler MH, et al. Only minor additional metabolic health benefits of high as opposed to moderate dose physical exercise in young, moderately overweight men. Obesity 2014;22(5):1220–32.
[40] Ross R, et al. Reduction in obesity and related comorbid conditions after diet-induced weight loss or exercise-induced weight loss in men. A randomized, controlled trial. Ann Intern Med 2000;133(2):92–103.
[41] Ross R, et al. Effects of exercise amount and intensity on abdominal obesity and glucose tolerance in obese adults: a randomized trial. Ann Intern Med 2015;162(5):325–34.
[42] Winding KM, et al. The effect on glycaemic control of low-volume high-intensity interval training versus endurance training in individuals with type 2 diabetes. Diabetes Obes Metab 2018;20(5):1131–9.
[43] Potteiger JA, et al. Glucose and insulin responses following 16 months of exercise training in overweight adults: the Midwest Exercise Trial. Metabolism 2003;52(9):1175–81.
[44] Ashton RE, et al. Effects of short-term, medium-term and long-term resistance exercise training on cardiometabolic health outcomes in adults: systematic review with meta-analysis. Br J Sports Med 2020;54(6):341–8.
[45] Lin X, et al. Effects of exercise training on cardiorespiratory fitness and biomarkers of cardiometabolic health: a systematic review and meta-analysis of randomized controlled trials. J Am Heart Assoc 2015;4(7):e002014.
[46] Battista F, et al. Effect of exercise on cardiometabolic health of adults with overweight or obesity: focus on blood pressure, insulin resistance, and intrahepatic fat—a systematic review and meta-analysis. Obes Rev 2021;22:e13269.
[47] Umpierre D, et al. Physical activity advice only or structured exercise training and association with HbA1c levels in type 2 diabetes: a systematic review and meta-analysis. JAMA 2011;305(17):1790–9.
[48] Frampton J, et al. The effect of a single bout of continuous aerobic exercise on glucose, insulin and glucagon concentrations compared to resting conditions in healthy adults: a systematic review, meta-analysis and meta-regression. Sports Medicine 2021;51(9):1949–66.
[49] Hrubeniuk TJ, et al. The ability of exercise to meaningfully improve glucose tolerance in people living with prediabetes: a meta-analysis. Scand J Med Sci Sports 2020;30(2):209–16.
[50] Group, D.S., E.D.E. Group. Glucose tolerance and cardiovascular mortality: comparison of fasting and 2-hour diagnostic criteria. Arch Int Med 2001;161(3):397–405.
[51] Gastaldelli A. Abdominal fat: does it predict the development of type 2 diabetes? Oxford University Press; 2008. p. 1118–9.
[52] Merz KE, Thurmond DC. Role of skeletal muscle in insulin resistance and glucose uptake. Compr Physiol 2011;10(3):785–809.
[53] Meyer C, et al. Role of human liver, kidney, and skeletal muscle in postprandial glucose homeostasis. Am J Physiol-Endocrinol Metab 2002;282(2):E419–27.
[54] Ivy JL. Role of exercise training in the prevention and treatment of insulin resistance and non-insulin-dependent diabetes mellitus. Sports Med 1997;24:321–36.
[55] Marinho R, et al. Regulation of hepatic TRB3/Akt interaction induced by physical exercise and its effect on the hepatic glucose production in an insulin resistance state. Diabetol Metab Syndr 2015;7:1–7.
[56] van Vliet S, et al. Obesity is associated with increased basal and postprandial β-cell insulin secretion even in the absence of insulin resistance. Diabetes 2020;69(10):2112–9.
[57] Kahn SE, et al. Quantification of the relationship between insulin sensitivity and β-cell function in human subjects: evidence for a hyperbolic function. Diabetes 1993;42(11):1663–72.
[58] Bergman RN, et al. Accurate assessment of β-cell function: the hyperbolic correction. Diabetes 2002;51(suppl_1):S212–20.
[59] Basu R, et al. Mechanisms of the age-associated deterioration in glucose tolerance: contribution of alterations in insulin secretion, action, and clearance. Diabetes 2003;52(7):1738–48.
[60] Yaribeygi H, et al. Molecular mechanisms by which aerobic exercise induces insulin sensitivity. J Cell Physiol 2019;234(8):12385–92.
[61] Gregory JM, et al. Aerobic exercise training improves hepatic and muscle insulin sensitivity, but reduces splanchnic glucose uptake in obese humans with type 2 diabetes. Nutr Diabetes 2019;9(1):25.
[62] Dunstan DW, et al. High-intensity resistance training improves glycemic control in older patients with type 2 diabetes. Diabetes Care 2002;25(10):1729–36.
[63] Frank P, et al. Strength training improves muscle aerobic capacity and glucose tolerance in elderly. Scand J Med Sci Sports 2016;26(7):764–73.
[64] Blumenthal JA, et al. Exercise and weight loss reduce blood pressure in men and women with mild hypertension - Effects on cardiovascular, metabolic, and hemodynamic functioning. Arch Intern Med 2000;160(13):1947–58.
[65] Bouchonville M, et al. Weight loss, exercise or both and cardiometabolic risk factors in obese older adults: results of a randomized controlled trial. Int J Obes (Lond) 2014;38(3):423–31.
[66] Brown AJ, et al. Effects of Exercise on Lipoprotein Particles in Women with Polycystic Ovary Syndrome. Med Sci Sports Exerc 2009;41(3):497–504.

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[67] Cassidy S, et al. High intensity intermittent exercise improves cardiac structure and function and reduces liver fat in patients with type 2 diabetes: a randomised controlled trial. Diabetologia 2016;59(1):56–66.
[68] Chen CN, et al. Effects of combined high-protein diet and exercise intervention on cardiometabolic health in middle-aged obese adults: a randomized controlled trial. Front Cardiovasc Med 2021;8.
[69] Cox KL, et al. Independent and additive effects of energy restriction and exercise on glucose and insulin concentrations in sedentary overweight men. Am J Clin Nutr 2004;80(2):308–16.
[70] DiPietro L, Yeckel CW, Dziura J. Progressive improvement in glucose tolerance following lower-intensity resistance versus moderate-intensity aerobic training in older women. J Phys Act Health 2008;5(6):854–69.
[71] Dunstan DW, et al. Effects of a short-term circuit weight training program on glycaemic control in NIDDM. Diabetes Res Clin Pract 1998;40(1):53–61.
[72] Hallsworth K, et al. Resistance exercise reduces liver fat and its mediators in non-alcoholic fatty liver disease independent of weight loss. Gut 2011;60(9):1278–83.
[73] Hallsworth K, et al. Modified high-intensity interval training reduces liver fat and improves cardiac function in non-alcoholic fatty liver disease: a randomized controlled trial. Clin Sci 2015;129(12):1097–105.
[74] Houghton D, et al. Exercise reduces liver lipids and visceral adiposity in patients with nonalcoholic steatohepatitis in a randomized controlled trial. Clin Gastroenterol Hepatol 2017;15(1):p. 96-+.
[75] Keadle SK, et al. The independent and combined effects of exercise training and reducing sedentary behavior on cardiometabolic risk factors. Appl Physiol Nutr Metab 2014;39(7):770–80.
[76] Lehrskov LL, et al. Effects of exercise training and IL-6 receptor blockade on gastric emptying and GLP-1 secretion in obese humans: secondary analyses from a double blind randomized clinical trial. Front Physiol 2019;10.
[77] Mendham AE, et al. A 12-week sports-based exercise programme for inactive Indigenous Australian men improved clinical risk factors associated with type 2 diabetes mellitus. J Sci Med Sport 2015;18(4):438–43.
[78] Nygaard H, et al. Long-term effects of daily postprandial physical activity on blood glucose: a randomized controlled trial. Appl Physiol Nutr Metab 2017;42(4):430–7.
[79] Potteiger JA, Jacobsen DJ, Donnelly JE. A comparison of methods for analyzing glucose and insulin areas under the curve following nine months of exercise in overweight adults. Int J Obes (Lond) 2002;26(1):87–9.
[80] Roberts CK, et al. Resistance training increases SHBG in overweight/obese, young men. Metab-Clin Exp 2013;62(5):725–33.
[81] Suntisawee S, et al. The effects of functional exercise training on obesity with impaired glucose tolerance. Sport Mont 2021;19(3):3–10.
[82] Tessier D, et al. Effects of aerobic physical exercise in the elderly with type 2 diabetes mellitus. Arch Gerontol Geriatr 2000;31(2):121–32.

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