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ORIGINAL ARTICLE

Effect of Physical Activity on Cardiorespiratory Fitness and Markers of Cardiovascular Disease Risk During Menopause: A Systematic Review and Meta-Analysis of Randomised-Controlled Trials

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Abstract

Purpose The menopause transition is associated with increased cardiovascular disease (CVD) risk. Cardiorespiratory fitness (CRF) is inversely associated with CVD risk in healthy populations. CRF thus represents a responsive target for physical activity (PA) interventions in menopausal populations. The aims were: (1) to investigate the impact of PA interventions on CRF and CVD risk factors, respectively, in perimenopausal and menopausal women, and (2) to examine the association between changes in CRF and CVD risk factors following PA interventions.

Methods Five databases (PubMed, EMBASE, Web of Science, CENTRAL, SPORTDiscus) were searched from inception to December 2023 for randomised controlled trials of PA interventions in menopausal females with non-active controls. The primary outcome was CRF, presented as VO_{2max}. The Cochrane Risk of Bias Tool was used to assess bias. Heterogeneity was observed using I^2. Effect measures were presented as Mean Difference (MD) with 95% Confidence Interval (CI). Meta-regression was conducted to examine the relationship between changes in VO_{2max} and reduction in CVD risk.

Results Seventy-eight studies with 5332 participants were included in meta-analysis. For VO_{2max}, there was a favourable effect of exercise versus control (3.51 mL/kg/min, 95% CI 2.75 to 4.27, 1968 participants, 30 trials). Considerable heterogeneity was observed. Meta-regression indicated a small, significant inverse association between changes in VO_{2max} and changes in systolic blood pressure in sensitivity analysis.

Conclusions All types of PA improved CRF. Moreover, improvements in CRF through PA intervention may be associated with concomitant reductions in systolic blood pressure, which is a major risk for CVD. Many outcomes had unexplained heterogeneity and unclear risk of bias due to lack of transparent reporting. Future research should investigate age and PA intensity as moderator variables. Future RCTs should focus on transparent reporting.

Keywords Menopause · Physical activity · Cardiovascular disease · Meta-analysis

Introduction

Menopause is the cessation of menses for a period of at least 12 months in otherwise healthy women, aged over 45 years [99]. It is associated with an increased risk of cardiovascular disease (CVD) due to endocrinological changes [30, 120]. These changes lead to adverse effects on body composition, lipids, and vascular health that contribute to menopausal associated CVD risk [16, 72]. Such changes begin during perimenopause, the transitional time before menopause where oestrogen starts to decrease. Annually, CVD is responsible for one third of female deaths globally [10], yet women are underrepresented in cardiovascular research [10]. For example, in cardiovascular trials

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registered between 2010 and 2017, only 38.2% of participants were female [64]. This decreases with participant age, as participation was lowest in women aged 61–65 years [64]. These figures represent a gap between disease prevalence and enrolment rates. In fact, the menopause transition is a crucial time to implement early intervention strategies to reduce CVD risk in this population [72]. It is therefore prudent to investigate methods for identifying, stratifying, and mitigating CVD risk in perimenopausal and menopausal women. This is in line with the UK government's 'Women's Health Strategy for England', which sets outs ambitions to prevent 150,000 heart attacks, strokes, and cases of dementia in women by 2032 [34]. The British Menopause Society have subsequently provided recommendations to develop the strategy and highlighted a 'pressing need' for research into non-hormonal therapeutic regimens which maximise benefits and minimise risks to menopausal women [51].

There is a wealth of evidence to suggest that habitual physical activity (PA) during perimenopause and menopause mitigates CVD risk factors [16, 30, 48, 67, 120, 140] including metabolic conditions [30, 67], body composition and blood pressure [48]. In fact, several studies have suggested that higher cardiorespiratory fitness (CRF) may be associated with a more favourable CVD risk profile during the menopause transition [1, 2, 47, 83, 125]. CRF, which can be improved through exercise training, is an indicator of habitual PA that may circumvent the limitations associated with self-report of PA (such as accuracy or recall) [78]. This makes it an important variable for assessing the impact of PA interventions. CRF is inversely associated with cardiovascular events independent of other risk factors in healthy men and women and is a strong predictor of all-cause mortality [76, 78]. Indeed, the American Heart Association has previously called for CRF to be monitored and assessed as a clinical vital sign [117]. Moreover, a recent systematic review found that CRF, expressed as maximal oxygen uptake (VO_{2max}), was a linear predictor of flow-mediated dilation (FMD) in postmenopausal women [17]. This study demonstrates a positive association between CRF and endothelial function in post menopause [17]. Such studies suggest that CRF could be used as a therapeutic target in this population [2], especially since CRF is capable of being modified through inexpensive, accessible intervention [117]. To support this notion, further research on effective PA interventions to improve CRF and the subsequent impact on CVD risk in peri- and post menopause is necessary.

One prior meta-analysis has found that PA interventions of all types (aerobic, resistance, or mixed) were effective in improving CRF in postmenopausal women [70]. However, this study excluded perimenopausal cohorts, a crucial transitional stage for intervention [72], and did not exclude participants already diagnosed with CVD. This limits the application of the results regarding the cardiorespiratory training response in peri-menopausal women, and in women without current CVD.

Furthermore, in line with goals for early intervention strategies to prevent CVD, it is also important to investigate whether improvements in CRF after PA interventions are associated with reduced CVD risk factors in peri-menopausal and menopausal women. Research examining this relationship is lacking. Therefore, the aims of this review are: (1) to investigate the impact of PA interventions on CRF and CVD risk factors, respectively, in perimenopausal and menopausal women, and (2) to examine the association between changes in CRF and CVD risk factors following PA interventions.

Methods

This protocol was prospectively registered on the Prospero International Prospective Register of Systematic reviews (CRD42023478916). This report is written in accordance with the guidelines of the Preferred Reporting Items for Systematic Reviews and Meta Analyses (PRISMA) 2020 statement [106]. Included studies all sought informed consent from participants and adhered to the Declaration of Helsinki.

Eligibility Criteria

Studies had to be published, peer-reviewed, and in the English language, with no date restriction. Studies were included based on predetermined Participant, Intervention, Comparison, Outcome and Study Design (PICOS) criteria. Table 1 indicates the inclusion and exclusion criteria for studies in the review. In this report, we understand 'physical activity (PA)' to refer to any bodily movement that increases energy expenditure, and 'exercise' as a subset of PA that is structured, planned and repetitive [22].

Search Methods

Searches were performed on the following five databases from inception to December 2023: PubMed (NLM), EMBASE (Elsevier), Web of Science (Clarivate Core Collection), Cochrane Central Register of Controlled Trials (CENTRAL), SPORTDiscus (EBSCO). A search strategy was developed for PubMed (Online Resource 1) and modified for each database by AW and a specialist subject librarian. The Cochrane highly sensitive search strategy for identifying randomised trials in PubMed (sensitivity and precision maximising version) was added to the search strategy [27].

Table 1 Eligibility criteria for included studies

Inclusion Criteria	
Participants	Studies of perimenopausal and postmenopausal women of at least 45 years of age according to National Institute for Health and Care Excellence criteria or STRAW + 10[52] [97]
Intervention	Physical activity interventions involving any or combinations of aerobic, low intensity, resistance, mobility, mixed, supervised, unsupervised, group, or individual exercise training. Interventions had to be structured and at least two weeks in duration. Studies including intervention arms that involve other components, such as dietary, were included if the PA intervention data and comparison could be extracted in isolation or the additional component was a consistent co-intervention (such as PA and diet versus diet). Studies including physical activity and hormone replacement therapy (HRT) were included.
Comparator	Non-active (no PA) control/comparison groups of perimenopausal and postmenopausal women (can include HRT).
Outcomes	Cardiorespiratory fitness, blood pressure, body composition, blood lipids, measures of vascular function (such as FMD, carotid artery intima-media thickness [cIMT], carotid stiffness and strain). Outcomes must have been measured at pre and post intervention (or presented as change score).
Study Design	Parallel randomised controlled trials, cluster randomised controlled trials
Exclusion Criteria	
Participants	Studies of women not experiencing perimenopause or menopause. Studies of women experiencing surgical or premature menopause. Studies of women with known cardiovascular disease, cancer, or other significant, chronic, medical conditions.
Intervention	Studies that combined physical activity with pharmacological intervention.
Comparator	Physically active control/comparisons, any comparator that was not relevant to the review (such as behavioural therapy) unless this was a consistent co-intervention in the PA arm.
Outcomes	Outcomes that were not presented at pre and post intervention (or as a change score). Non-parametric data [58].
Study Design	Commentaries, opinion articles, systematic (or other) reviews, case studies, cohort studies, observational studies, non-randomised controlled studies, pre and post studies.

All searches were conducted by the first author (AW) with support from a specialist subject librarian and uploaded into Covidence [144]. Duplicates and non-randomised control trials were removed by Covidence automation tools. A random 30% selection of these removals was checked for accuracy. All titles and abstracts were screened (AW) and a 30% sample were independently screened by three reviewers (AMJ, MF, VJ) [86, 110]. Full-text screening was completed by AW with 20% independently screened by two reviewers (MF, VJ) [86, 110]. All disagreements were resolved through discussion. Where multiple publications of the same trial were retrieved, these were classed as one study. The earliest paper was used as the primary reference, but data were extracted for outcomes across each paper to ensure completeness of data.

Data Extraction

Data extraction was conducted through Covidence using a standardised data extraction form that was created a priori and piloted to assess suitability. Data were extracted by one reviewer (AW) with all forms verified by a second, experienced reviewer (AB) [86]. All disagreements were resolved through discussion. Data were extracted relating to study characteristics (publication year, authors, country, study design), participant characteristics and context (including age, ethnicity, menopause stage), intervention-related information (description, mode of delivery, type of PA, duration, intensity), and measurements of the outcomes (e.g., VO_{2max}, blood pressure, lipids, body composition).

Continuous data where outcomes were reported post intervention, or as a change score, were sought from text and tables. Authors were contacted for missing data. If no response was received, data were sought from figures if available or not included in the meta-analysis. Data from intervention arms that combined additional components (such as dietary components or supplements) were extracted if the additional component was a consistent co-intervention (i.e. exercise and diet versus diet alone) [57]. Data were extracted as mean and SD, 95% confidence interval (CI), or standard error (SE).

Risk of Bias

The Cochrane Collaboration's tool for assessing risk of bias was used [56]; six specific domains (sequence generation, allocation concealment, blinding, incomplete outcome data, selective outcome reporting, and any other sources of bias) were assessed. One reviewer (AW) assessed the risk of bias with a second reviewer (AB) independently verifying results. Each domain was graded as either high, low, or unclear risk. In PA trials, it is not possible to blind participants (and often not researchers) to the intervention. Therefore, all studies were assigned a high risk of performance bias. It is important to note that this should not indicate that the trial methodology is poor, but that the limitations of lack of blinding have been taking into consideration by reviewers.

We judged studies with >20% of data missing due to attrition as high risk of attrition bias. We considered studies with baseline differences likely to affect the outcome of the analysis, less than 65% adherence to the exercise intervention, and contamination in the control group (that is, the control group engaged in, or significantly increased, their baseline PA) as high risk of 'other sources of bias' based on similar reviews of PA interventions [44, 73].

Strategy for Data Synthesis

Studies were synthesised into separate meta-analyses according to the relevant comparison (exercise versus control, exercise and diet versus diet, and exercise and supplement/placebo versus supplement/placebo). This ensures the effects of PA can be isolated because co-intervention is consistent across both the PA group and the control group. Differences in effect size can thus be assumed to be due to PA. For each comparison, where outcome data from ≥two trials were available, pooled intervention effect estimates expressed as mean difference (difference between means) and their 95% CIs were presented. RevMan was used to conduct the meta-analyses and generate forest plots [26]. Based on the Cochrane Handbook's recommendations, the inverse-variance (DerSimonion and Laird) random-effects method for meta-analysis was utilised to combine data [33]. Mean and SD for either change from baseline or post-intervention values were combined in the meta-analysis [33]. RevMan was used to convert SE or CI into SD. Where units of measurements varied, these were converted to the most common measure (e.g., high-density lipoprotein [HDL] converted from mg/dL to mmol/L). Multiple eligible intervention arms in comparison to a single control group (such as multiple exercise interventions) were combined as recommended by Higgins et al. [57].

Heterogeneity

Statistical and clinical/methodological heterogeneity were assessed. The magnitude of effects and the direction were considered in assessment of heterogeneity. The I^2 statistic, representing the impact of heterogeneity, was interpreted as 0–40%: might not be important, 30%–60%: may represent moderate heterogeneity, 50%–90%: may represent substantial heterogeneity, and 75%–100%: considerable heterogeneity [33]. Heterogeneity was investigated by removing the largest outlier from the analysis (non-overlap of CIs with other studies) to assess the impact on the I^2 statistic. Heterogeneity was also investigated using subgroup analysis for exercise type and intervention length.

Subgroup Analysis

Subgroup analysis was performed to (1) investigate heterogeneity of PA intervention on CRF, and (2) determine the effect of characteristics on the magnitude of effect [115]. A priori, subgroup analysis was to be performed on menopause status (perimenopause or menopause), HRT status (yes or no), exercise type (aerobic, resistance or mixed), and intervention length (three months or fewer, > three months to six months, > six months). However, insufficient studies were available to assess menopause status and HRT status. Subgroup analyses were performed for intervention length and exercise type to determine whether these moderate effect estimate where statistically favourable effects were observed. To reduce the risk of false positive comparisons, no other subgroup analyses were performed [115]. Subgroup analyses are presented for transparency and to informally compare the magnitude of effect estimates. Subgroup analyses were only conducted for outcomes where there were at least ten studies [115]. A formal test for statistical differences between subgroups is presented.

Moderator analysis was conducted using method of moments meta-regression to address study aim two: to investigate the association between changes in CRF (expressed as VO_{2max}) and CVD risk markers following PA interventions. Meta-regression was performed using Comprehensive Meta-Analysis v4 [15]. Change in VO_{2max} was entered into the model as the explanatory (independent) variable. This was calculated using the difference between mean pre and post VO_{2max} scores of participants in the exercise/PA groups only to reflect the effects of the intervention. Each CVD risk factor with outcome data available from at least 10 studies were entered into the model as an outcome (dependent) variable.

Sensitivity Analysis

Studies with a high overall risk of bias were removed from the results to assess the impact on the effect estimate. Because all studies were judged to have high risk of performance bias, we considered studies judged to have a high risk of bias in at least three other domains as high risk. As above, sensitivity analysis was also performed to assess the impact of outliers.

In the regression analysis, outlying cases were identified via multiple construct techniques (i.e. visual inspection of scatterplots for data points lying far away from the centroid of the data) [3]. Sensitivity analyses were performed by removing the single largest outlier for each variable to assess their influence. If influential outliers were present, the results of the model were reported with and without the outlier.

Assessment of Reporting Bias

To investigate publication bias in the primary outcome, a funnel plot was generated to explore the possibility of small study effects (where studies with a small sample size report larger effects). This was followed up with Egger's test of the intercept [37] to aid in interpretation. For all statistical analyses, significance was accepted at $P<0.05$.

Results

Selection of Studies

In total, 8743 records were returned from the database searches. After removing duplicates, 3449 studies were screened via title and abstract. A total of 332 full-text studies were screened for inclusion and 254 were excluded with reasons detailed in Fig. 1 with reasons for each study in Online Resource 2. A total of 78 studies were included, with 88 associated publications, since five studies had multiple publications.

Study Design and Details

Table 2 summarises characteristics for each included study. Thirteen studies were conducted in both Brazil [7, 14, 21, 31, 32, 85, 103, 104, 107, 114, 118, 119, 122] and the United States [25, 39, 41–43, 54, 63, 66, 92, 95, 100, 136, 146], nine were conducted in South Korea [65, 79, 80, 108, 123, 129–132], seven were conducted in Iran [11, 68, 71, 116, 126, 133, 138], four each were conducted in Canada [18, 59, 74, 113], Finland [8, 9, 82, 96], Portugal [6, 102, 109, 121] and Spain [28, 29, 46, 124], three were conducted in both China [53, 60, 149] and Turkey [45, 50, 142], two were conducted in Sweden [13, 101], and one each conducted in Australia [150], Denmark [137], Egypt [139], Pakistan [12],

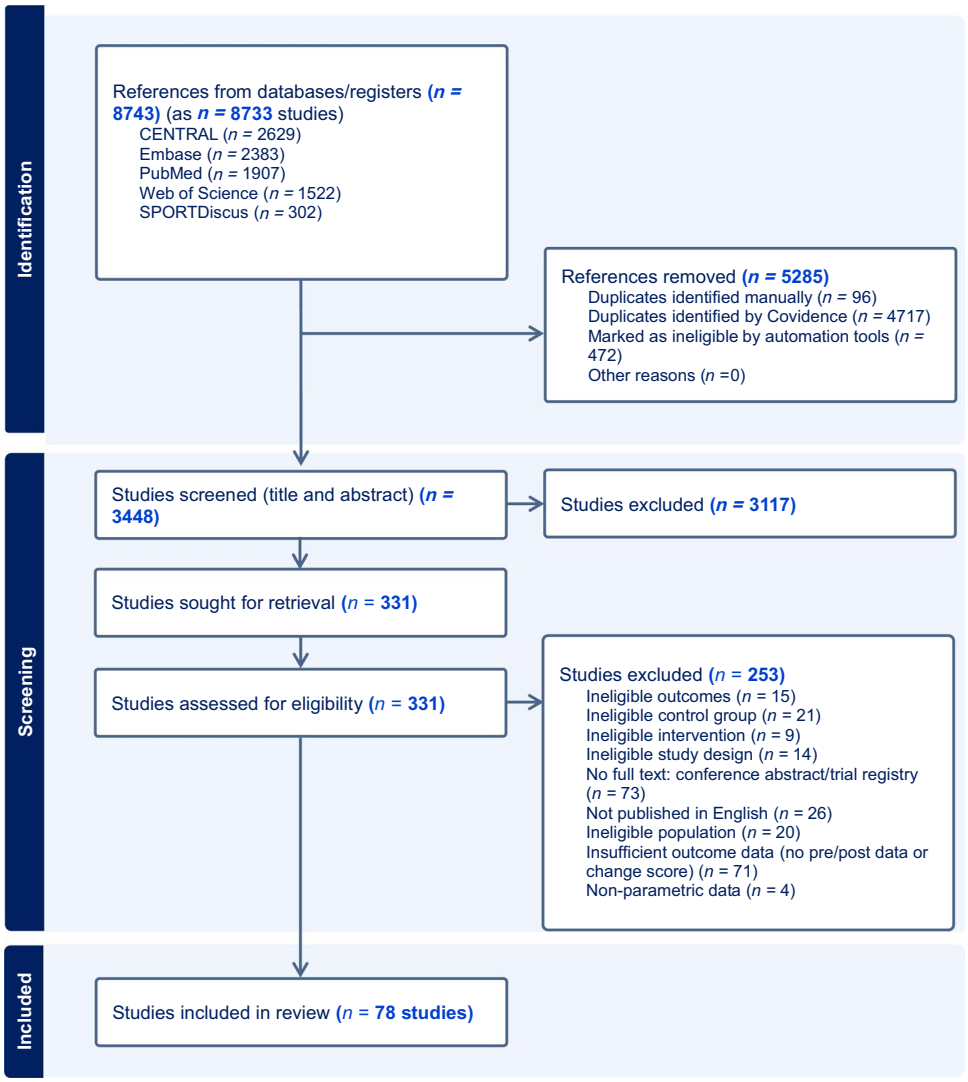

Fig. 1 PRISMA Flow Diagram. Source: Page MJ, et al. [106]

Table 2 Characteristics of included studies

Study ID	Country	Comparisons	Arms Included in Analysis	Type	Mode	Adherence	Length	Frequency
Akazawa 2012 [5]	Japan	Exercise versus control	2	Aerobic	Cycling/Walking	>80%	8 weeks	2–3/week minimum
Akazawa 2013 [4]	Japan	Exercise and supplement/placebo versus supplement/placebo	4	Aerobic	Cycling/Walking	>80%	8 weeks	3/week minimum
Aragão 2014[6]	Portugal	Exercise versus control	2	Mixed	Step training and weights training	>80%	12 months	3/week
Araujo 2023 [7]	Brazil	Exercise versus control	2	Aerobic	Rowing	Not reported	10 weeks	3/week
Asikainen 2002 [8]	Finland	Exercise versus control	5	Aerobic	Walking	>80%	24 weeks	5/week
Asikainen 2002a [9]	Finland	Exercise versus control	3	Aerobic	Walking	>80%	15 weeks	5/week
Azadpour 2017 [11]	Iran	Exercise versus control	2	Aerobic	Walking or jogging	Not reported	10 weeks	3/week
Basharat 2019 [12]	Pakistan	Exercise and supplement/placebo versus supplement/placebo	4	Aerobic	Not reported	Not reported	16 weeks	5/week
Bergstrom 2009 [13]	Sweden	Exercise versus control	2	Aerobic	Walking	>80%	12 months	4/week
Bocalini 2010 [14]	Brazil	Exercise versus control	2	Resistance	Weight training	>80%	24 weeks	3/week
Brochu 2009 [18]	Canada	Exercise and diet versus diet	2	Resistance	Weight training	Not reported	6 months	3/week
Cardoso 2016 [21]	Brazil	Exercise versus control	2	Resistance	Weight training	<80%	12 weeks	3–5/week progressive
Church 2007 [25]	USA	Exercise versus control	4	Aerobic	Cycle ergometer and treadmill	>80%	6 months	3–4/week
Colado 2009 [28]	Spain	Exercise versus control	3	Resistance	Aquatic and Thera band resistance	>80%	24 weeks	2–3/week progressive
Coll-Risco 2019 [29]	Spain	Exercise and other, non-active intervention versus other, non-active intervention	2	Mixed	Circuit training and aerobic exercise	<80%	16 weeks	3/week
ConceicÃo 2013 [31]	Brazil	Exercise versus control	2	Resistance	Weight training	Not reported	16 weeks	3/week
Correa 2014 [32]	Brazil	Exercise versus control	3	Resistance	Weight training	Not reported	12 weeks	Not reported
Elliott 2002 [38]	United Kingdom	Exercise versus control	2	Resistance	Weight training	>80%	8 weeks	3/week
Evans 2021[39]	USA	Exercise and diet versus diet	3	Mixed	Aerobic and weight training	<80%	6 months	3/week
Figueroa 2003 [41]	USA	Exercise versus control, exercise and other, non-active intervention versus other, non-active intervention	4	Resistance	Weight training and weight bearing exercises	<80%	12 months	3/week
Flores 2022 [42]	USA	Exercise versus control	2	Mixed	Weight bearing impact with weighted vest	Not reported	12 months	3/week
Fox 1996 [43]	USA	Exercise and diet versus diet	3	Mixed	Walking and weight training	>80%	24 weeks	5/week
Gomez-Tomas 2018 [46]	Spain	Exercise versus control	2	Resistance	Thera band	Not reported	12 months	3/week
Gelecek 2012 [45]	Turkey	Exercise versus control	2	Resistance	Weight training	>80%	12 weeks	3/week

Table 2 (continued)

Study ID	Country	Comparisons	Arms Included in Analysis	Type	Mode	Adherence	Length	Frequency
Guzel 2022 [50]	Turkey	Exercise versus control	2	Aerobic	Walking or jogging	>80%	10 weeks	3/week
He 2022 [53]	China	Exercise versus control	4	Aerobic	Walking or jogging	Not reported	12 weeks	5/week
Henagan 2011 [54]	USA	Exercise versus control	2	Resistance	Weight training	>80%	12 weeks	3/week
Hintze 2018 [59]	Canada	Exercise and diet versus diet	2	Resistance	Weight training	<80%	12 months	3/week first 6 months, 2/week thereafter
Hu 2017 [60]	China	Exercise versus control	2	Aerobic	Walking	Not reported	16 weeks	3/week
Jaime 2019 [63]	USA	Exercise versus control	2	Resistance	Weight training	>80%	12 weeks	Unclear
Jo 2020 [65]	South Korea	Exercise versus control	3	Aerobic	Exergame and Treadmill	>80%	12 weeks	unclear
Joseph 2001 [66]	USA	Exercise and diet versus diet	2	Resistance	Weight training	>80%	7 weeks	4/week
Kazemi 2023 [68]	Iran	Exercise versus control	3	Mixed	Weight training and running/walking	Not reported	8 weeks	3/week
Khosravi 2018 [71]	Iran	Exercise versus control	2	Aerobic	Weight training	Not reported	6 Months	3/week
Klentrou 2007 [74]	Canada	Exercise versus control	2	Mixed	Weighted vest	>80%	12 weeks	3/week
Kobayashi 2022 [75]	Japan	Exercise versus control	3	Aerobic	Jogging or running	Not reported	8 weeks	4/week
Lee 2012 [79]	South Korea	Exercise versus control	2	Aerobic	Yoga	Not reported	16 weeks	3/week
Lee 2021 [80]	South Korea	Exercise versus control	2	Aerobic	Taekwondo	Not reported	16 weeks	5/week
Luoto 2012 [82]	Finland	Exercise versus control	2	Aerobic	Nordic walking and other aerobic activities	Not reported	6 months	4/week
Maesta 2007 [85]	Brazil	Exercise and supplement/placebo versus supplement/placebo	4	Resistance	Weight training	>80%	16 weeks	3/week
Miyaki 2012 [89]	Japan	Exercise versus control	2	Aerobic	Weight training	>80%	8 weeks	3–5/week
Moreau 2001 [92]	USA	Exercise versus control	2	Aerobic	Walking or cycling	>80%	24 weeks	Self-selected
Morrison 1986 [95]	USA	Exercise versus control	2	Aerobic	Walking and jogging	Not reported	8 months	3/week
Munukka 2016 [96]	Finland	Exercise versus control	2	Resistance	Aquatic resistance training	>80%	16 weeks	3/week
Nicklas 2009 [100]	USA	Exercise and diet versus diet	3	Aerobic	Walking	>80%	20 weeks	3/week
Nilsson 2023 [101]	Sweden	Exercise versus control	2	Resistance	Weight training	<80%	15 weeks	3/week
Novaes 2014 [102]	Portugal	Exercise versus control	3	Mixed	Weight training and water aerobics	Not reported	24 weeks	3/week
Orsatti 2008 [103]	Brazil	Exercise versus control	2	Resistance	Weight training	Not reported	16 weeks	3/week
Orsatti 2010 [104]	Brazil	Exercise and supplement/placebo versus supplement/placebo	4	Resistance	Weight training	>80%	9 months	2/week
Paolillo 2017 [107]	Brazil	Exercise versus control	2	Aerobic	Treadmill	Not reported	6 months	2/week

Table 2 (continued)

Study ID	Country	Comparisons	Arms Included in Analysis	Type	Mode	Adherence	Length	Frequency
Park 2015 [108]	South Korea	Exercise versus control	2	Mixed	Running and weight training	Not reported	12 weeks	3/week
Pereira 2023 [109]	Portugal	Exercise versus control	2	Aerobic	Handball	<80%	36 weeks	2–3/week
Ready 1996 [113]	Canada	Exercise versus control	3	Aerobic	Walking	>80%	24 weeks	3/week
Rezende Barbosa 2019 [114]	Brazil	Exercise versus control	2	Mixed	Running and weight training	>80%	18 weeks	3/week
Roghani 2013 [116]	Iran	Exercise versus control	3	Aerobic	Treadmill	Not reported	6 weeks	3/week
Rossi 2016 [118]	Brazil	Exercise versus control	3	Mixed	Aerobic and weight training	Not reported	16 weeks	Not reported
Rossi 2018 [119]	Brazil	Exercise versus control	3	Mixed	Aerobic and weight training	Not reported	16 weeks	Not reported
Santa-Clara 2003 [121]	Portugal	Exercise versus control	4	Aerobic	Walking and jogging, cycling, rowing	Not reported	6 months	3–4/week
Sardeli 2022 [122]	Brazil	Exercise versus control	2	Mixed	Weight training and treadmill	>80%	16 weeks	3/week
Seo 2012 [123]	Korea	Exercise and supplement/placebo versus supplement/placebo	4	Mixed	Resistance training and aerobic	Not reported	12 weeks	3/week
Serrano-GuzmÃn 2016 [124]	Spain	Exercise versus control	2	Aerobic	Dancing	>80%	8 weeks	3/week
Shabani 2018 [126]	Iran	Exercise versus control	2	Mixed	Weight training, treadmill, cycling	Not reported	8 weeks	3/week
Shaw 2016 [127]	South Africa	Exercise versus control	2	Resistance	Weight training	Not reported	6 weeks	2/week
Shen 2013 [128]	Taiwan	Exercise versus control	2	Aerobic	Step aerobics	>80%	10 weeks	3/week
Shin 2014 [129]	South Korea	Exercise versus control	2	Aerobic	Tennis	Not reported	12 weeks	3/week
Son 2016 [130]	South Korea	Exercise versus control	2	Mixed	Resistance bands and walking	Not reported	12 weeks	3/week
Son 2020 [131]	South Korea	Exercise versus control	2	Resistance	Resistance bands	Not reported	12 weeks	3/week
Son 2021 [132]	Korea	Exercise versus control	2	Resistance	Resistance bands	>80%	12 weeks	3/week
Soori 2017 [133]	Iran	Exercise versus control	4	Mixed	Weight training and swimming/water walking	Not reported	10 weeks	3/week
Stefanick 1998 [136]	USA	Exercise versus control and exercise and diet versus diet alone	4	Aerobic	Brisk walking or jogging	Not reported	12 months	3/week minimum
Svendsen 1993 [137]	Denmark	Exercise versus control	2	Mixed	Cycling, stair walking, treadmill running	>80%	12 weeks	3/week
Taghian 2022 [138]	Iran	Exercise versus control	2	Mixed	Weight training and aerobic activity	Not reported	12 weeks	Not reported
Taha 2016 [139]	Egypt	Exercise versus control	2	Aerobic	Treadmill intervals	Not reported	10 weeks	3/week
Teoman 2004 [142]	Turkey	Exercise versus control	2	Mixed	Resistance bands and cycling/step aerobics	Not reported	6 weeks	3/week
Wong 2019 [146]	USA	Exercise versus control	2	Aerobic	Swimming	>80%	20 weeks	3–4 days/week

Table 2 (continued)

Study ID	Country	Comparisons	Arms Included in Analysis	Type	Mode	Adherence	Length	Frequency
Yoshizawa 2010 [147]	Japan	Exercise and supplement/placebo versus supplement/placebo	4	Aerobic	Walking or cycling	>80%	8 weeks	3–5/week progressive
Zhang 2014 [149]	China	Exercise versus control	2	Aerobic	Walking	Not reported	12 weeks	3/week
Zhang 2019 [150]	Australia	Exercise versus control	2	Aerobic	Bike sprints	>80%	8 weeks	3/week

South Africa [127], Taiwan [128], and the United Kingdom [38].

All studies were parallel randomised controlled trials (RCT). Sixty-five studies reported an exercise versus control comparison (83%). Seven studies reported an exercise and diet versus diet alone comparison (9%) [18, 39, 43, 59, 66, 100, 136]. Six studies reported on a comparison of exercise and supplement versus supplement alone (lactotripeptides [147], soy protein [85], soy isoflavone [104], garlic [12, 123], ginger [12], curcumin [4]) or exercise and placebo versus placebo alone (8%). Finally, two studies reported on a comparison of exercise and other, non-active intervention (such as counselling) versus other, non-active intervention alone (3%) [29, 41].

Participant and Intervention Characteristics

Participant Characteristics

Two studies included only women who were perimenopausal [29, 149]. Definitions of perimenopause for the purpose of the study criteria were not provided. All other studies recruited only women who were post menopause according to the definition of no menstrual bleeding in the last 12 months. Forty-one studies specifically excluded women taking HRT (Table 2), while only one study specifically included taking HRT for at least one year as inclusion criteria [142]. Eight studies reported that HRT use across groups was mixed across groups [6, 8, 9, 25, 41, 43, 66, 92]. Twenty-eight studies did not report on the use of HRT in participants (Table 2). The mean and median ages were 58.6 ± 5.5 and 57.4 years, respectively, with an age range from 47.7 years [139] to 76 years [130].

The total number of participants included in all trial arms relevant to the analysis was 5332. Of these, 2328 were in control comparison groups (control, diet, supplement, or placebo alone). A further 3004 were participants in intervention (exercise) groups.

Study Interventions

Thirty-six studies investigated aerobic exercise only, 22 studies investigated resistance exercise only, and 20 studies investigated mixed aerobic and resistance exercises. Intervention length was three months or fewer in 37 studies, > 3–6 months in 31 studies.

The adherence to exercise interventions was reported as > 80% in 34 studies, < 80% in seven studies, and not reported in 37 studies. Mean attrition was 13%, with a range of 52% to 2%. Eighteen trials reported an attrition of over 20%.

Risk of Bias in Included Studies

Studies were assessed for bias using the Cochrane Risk of Bias tool. Figure 2 shows the summary of authors' judgements across eight domains: selection bias, performance bias, detection bias, attrition bias, reporting bias, and three other sources of bias (baseline differences, adherence, and contamination). The risk of bias summary graph by study is available in Online Resource 3.

Three studies were judged to have a high risk of selection bias due to inappropriate randomisation sequence generation or lack of allocation concealment from researchers [7, 139]) or uneven groups with no indicated allocation ratio [92]. Twenty-three studies were judged to have a low risk of selection bias due to appropriate sequence generation and allocation concealment procedures [6, 13, 21, 25, 29, 39, 45, 46, 82, 96, 100, 101, 104, 107, 109, 114, 124, 127, 130, 131, 136, 142, 146]. The remaining studies did not report either sequence generation or allocation concealment methods. Due to the nature of the intervention(s), all trials were judged to be at a high risk of performance bias. Eighteen studies were judged to be at a low risk of detection bias because they reported that outcome assessors were blinded to allocation [6, 7, 25, 29, 45, 63, 80, 96, 100, 101, 107, 124, 127, 130–132, 137, 146]. Two studies had an unclear risk of detection bias due to inconsistent reporting or blinding of outcome assessors for some outcomes but not all [74, 114]. The remaining studies were judged to have a high risk of

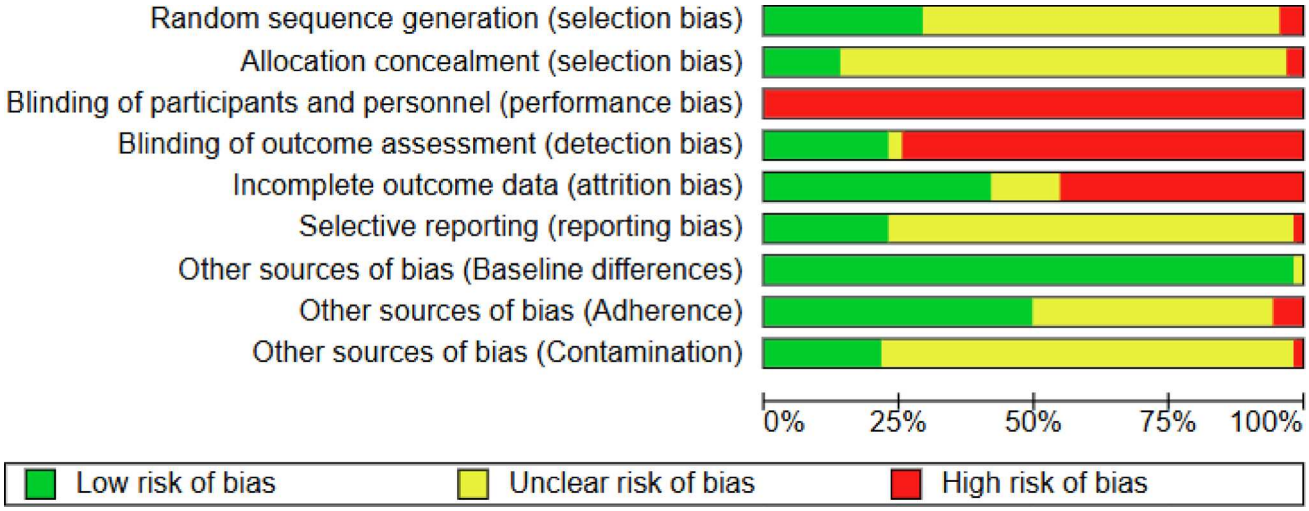

Fig. 2 Summary of authors judgements across eight domains for all 78 studies

detection bias for failing to report whether assessors were blinded to allocation.

Thirty-three studies were judged to be at low risk of attrition bias due to having more than 95% of outcome data available, appropriate handling of missing data (up to 20%) by reporting complete baseline statistics and using baseline statistics as covariates, or intention-to-treat analysis [5, 8, 9, 11, 14, 29, 32, 50, 53, 54, 66, 68, 75, 79, 80, 92, 96, 102, 103, 122–124, 126, 127, 130–132, 136, 137, 139, 146, 147]. Ten studies were judged to be at unclear risk of attrition bias due to missing CONSORT diagrams or missing or inconsistent *N* in data Tables [4, 12, 25, 31, 38, 42, 89, 129, 133, 142]. The remaining studies were judged to be at high risk of attrition bias due to > 20% missing data and inappropriate handling (such as incomplete baseline statistics). One study was judged to be at high risk of reporting bias for selective reporting of outcome [150]. Eighteen studies were judged to have a low risk of reporting bias due to the presence of pre-registered or published protocols with consistent outcomes [6, 25, 29, 39, 42, 59, 65, 82, 96, 100, 101, 107, 114, 122, 126, 131, 137, 146]. The remaining studies had unclear risk of reporting bias as the authors were unable to locate pre-registered protocols.

One study was judged to have an unclear risk of bias for baseline differences due to lack of reporting [31]. The remaining studies reported no differences between baseline statistics. Four studies were judged to have a high risk of bias due to adherence to exercise interventions of < 80% [18, 42, 59, 71]. One study was judged to have a high risk of bias for contamination because the control group received counselling on behaviour change during the intervention which could have influenced their PA habits [13].

There were sufficient studies for three comparisons to be made: (1) exercise versus control, (2) exercise and diet versus diet alone, and (3) exercise and supplement/placebo versus supplement/placebo alone.

Exercise Versus Control

VO_{2max}

Thirty-one studies reported relative VO_{2max} or VO_{2peak} as an outcome. Table 3 indicates which studies reported peak and which studies reported max. Seventeen studies used direct methods to measure VO_{2max} or VO_{2peak} through breath-by-breath gas analysis. Of these, 14 used a treadmill and three used a cycle ergometer. Four studies reported indirect, estimated methods to report VO_{2max} or VO_{2peak}. Each used a walking test to estimate VO_{2max}. Ten studies did not report how VO_{2max} or VO_{2peak} was measured.

Overall, there was a statistically significant favourable effect of exercise on relative VO_{2max}/VO_{2peak} in comparison to the control group (MD 3.51 mL/kg/min, 95% CI 2.75 to 4.27, 1968 participants, 30 trials, $I^2 = 92\%$, $P \leq 0.001$). Considerable heterogeneity was observed. Online resource 6 presents forest plots for all analyses in exercise versus control.

In sensitivity analysis, the effect remained statistically significant when removing studies with a high risk of bias in four or more domains [7, 71, 150](MD 3.42 mL/kg/min, 95% CI 2.65 to 4.19, 1918 participants, 28 trials, $I^2 = 92\%$, $P \leq 0.001$), and when removing the largest outlier [65] (MD 3.18 mL/kg/min, 95% CI 2.49 to 3.87, 1914 participants, 29 trials, $I^2 = 90\%$, $P \leq 0.001$). Heterogeneity remained considerable.

Table 4 indicates the results of the subgroup analysis. The statistical test for subgroup differences indicated a statistically significant subgroup effect for intervention length

Table 3 Summary of studies that reported VO_{2max}/VO_{2peak} as an outcome and the corresponding method used

Study ID	Peak (VO_{2peak}) or Max (VO_{2max})	Direct or Indirect	Method
Akazawa 2013 [4]	Peak	Direct	Cycle ergometer
Araujo 2023 [7]	Peak	Direct	Cycle ergometer
Asikainen 2002 [8]	Max	Direct	Treadmill
Asikainen 2002a [9]	Max	Direct	Treadmill
Azadpour 2017 [11]	Max	Direct	Treadmill
Bocalini 2010 [14]	Max	Direct	Bruce Treadmill protocol
Brochu 2009 [18]	Max	Not reported	Not reported
Church 2007 [25]	Max	Not reported	Not reported
Jo 2020 [65]	Peak	Direct	Treadmill
Kazemi 2023 [68]	Peak	Indirect	6-minute walk test
Khosravi 2018 [71]	Max	Not reported	Not reported
Luoto 2012 [82]	Max	Indirect	Walk test
Miyaki 2012 [89]	Peak	Direct	Cycle ergometer
Morrison 1986 [95]	Max	Direct	Treadmill
Munukka 2016 [96]	Peak	Indirect	UKK 2 km walking test
Nicklas 2009 [100]	Max	Direct	Treadmill
Novaes 2014 [102]	Peak	Direct	Bruce Treadmill protocol
Park 2015 [108]	Max	Direct	Balke treadmill protocol
Pereira 2023 [109]	Peak	Direct	Treadmill
Ready 1996 [113]	Peak	Direct	Balke treadmill protocol
Sardeli 2022 [122]	Peak	Direct	Treadmill
Shabani 2018 [126]	Max	Not reported	Not reported
Shaw 2016 [127]	Max	Not reported	Not reported
Shen 2013 [128]	Max	Not reported	Not reported
Shin 2014 [129]	Max	Direct	Bruce Treadmill protocol
Stefanick 1998 [136]	Max	Not reported	Not reported
Svendsen 1993 [137]	Max	Not reported	Not reported
Teoman 2004 [142]	Max	Indirect	6-minute walk test
Wong 2019 [146]	Max	Direct	Bruce Treadmill protocol
Yoshizawa 2010 [147]	Max	Not reported	Not reported
Zhang 2019 [150]	Max	Not reported	Not reported

UKK = Urho Kaleva Kekkonen Institute

($P \leq 0.001$). The magnitude of effect was highest for interventions of 3 months or fewer. No significant subgroup effect was observed for exercise type. It should be noted that subgroups had uneven covariate distribution (only three studies in >6 months) which may affect confidence in the results. In addition, although heterogeneity was reduced within subgroups, substantial heterogeneity remained.

Results are presented in Table 5 for the subgroup analysis for exercise type and intervention length. The overall meta-analysis showed high heterogeneity that was not meaningfully reduced by predefined subgroup analyses (Exercise Type and Intervention Length). Where at least ten studies were available, a post hoc subgroup analysis combining both Exercise Type and Intervention Length was undertaken to explore residual heterogeneity.

Sensitivity analyses were conducted by removing a single outlier in each subgroup. This substantially reduced heterogeneity without affecting the direction or magnitude of the pooled effect. For Aerobic/3 months, removal of the largest outlier [65] reduced I^2 to 0% (MD 4.69 mL/kg/min, 95% CI 3.95 to 5.43, 253 participants, 8 trials, $P \leq 0.01$). For Aerobic/>3–6 months, removal of the largest outlier [25] reduced I^2 to 49% (MD 3.06 mL/kg/min, 95% CI 2.05 to 4.06, 640 participants, 7 trials, $P \leq 0.01$). For Aerobic >6 months, removal of the largest outlier [95] reduced I^2 to 0% (MD 3.12 mL/kg/min, 95% CI 1.98 to 4.25, 133 participants, 2 trials, $P \leq 0.01$).

Publication Bias for Primary Outcome

The funnel plot (Fig. 3) indicates possible missing studies toward the bottom left of the funnel. Using Egger's test of the intercept, the intercept (B0) is 3.35299, 95% confidence interval (2.28467, 4.42131), with $t = 6.42904$, df = 28. The 1-tailed P-value is <0.0001. This suggested the presence of small-study effects and significant publication bias.

We used Duval and Tweedie's trim-and-fill method with a random-effects model to evaluate publication bias. However, the analysis did not impute any missing studies, and the adjusted pooled effect size remained unchanged. This result, in conjunction with the Egger's test and visual inspection of the funnel plot suggest that while small-study effects were present, publication bias may not be the sole factor responsible for the asymmetry, especially where heterogeneity is present [62].

CVD Risk Factors

Table 6 provides summary statistics for each CVD risk factor for the comparison of exercise versus control. There was a statistically significant favourable effect of exercise on FMD, systolic blood pressure (SBP), Body fat %, body-mass index (BMI), body mass, fat mass, fat free mass (FFM), HDL-C, low-density lipoprotein cholesterol (LDL-C), total cholesterol to HDL ratio (TC: HDL), triglycerides (TG), waist circumference (WC), and waist-to-hip ratio (WHR). No effect was observed for diastolic blood pressure (DBP), total cholesterol, or hip circumference (HC). Online Resource 4 provides details of subgroup analysis (further information on sensitivity analysis can be found in Online

Table 4 Subgroup differences for VO_{2max}/VO_{2peak} across covariates: exercise type and intervention length

Sub analysis	Subgroup	No. of studies	*N*	MD	Lower 95% CI	Upper 95% CI	I^2 (%)	*P* (overall effect)	Test for subgroup differences
Exercise Type	Aerobic	20	1562	4.06	2.86	5.25	91	$P \leq 0.001$	
	Resistance	4	189	1.83	0.19	3.47	93	$P = 0.03$	
	Mixed	6	217	3.3	1.72	4.88	60	$P \leq 0.001$	
	Subgroup Differences								$P = 0.10$
Intervention Length	3 months or fewer	15	548	4.34	3.06	5.61	84	$P \leq 0.001$	
	>3–6 months	12	1262	2.28	1.48	3.08	89	$P \leq 0.001$	
	>6 months	3	158	3.67	1.79	5.54	53	$P \leq 0.001$	
	Subgroup Differences								$P \leq 0.001$*

MD mean difference, *CI* confidence interval. Heterogeneity reported using I^2 statistic. * = significant subgroup difference.

Table 5 Subgroup differences for VO_{2max}/VO_{2peak} across covariates: exercise type and intervention length

Sub analysis	Subgroup	No. of studies	*N*	MD	Lower 95% CI	Upper 95% CI	I^2 (%)	*P*
Type/Length	Aerobic/3 months or fewer	9	300	5.28	3.58	6.99	77	$P \leq 0.01$
	Aerobic/>3–6 months	8	1104	2.75	1.5	4.01	84	$P \leq 0.01$
	Aerobic/>6 months	3	158	3.67	1.79	5.54	53	$P \leq 0.01$

MD mean difference, *CI* confidence interval. Heterogeneity reported using I^2 statistic

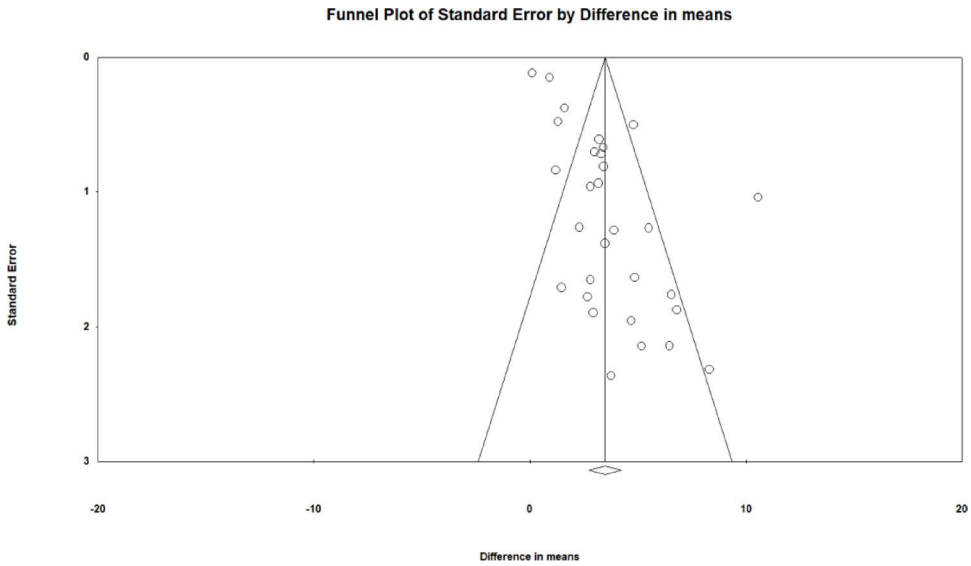

Fig. 3 Funnel plot analysis of 30 studies reporting VO_{2max}/VO_{2peak}

Resource 5. Forest plots for meta-analysis are presented in Online Resource 6).

Change in VO_{2max} as a Moderator Variable

In meta-regression analyses, change in VO_{2max} in the exercise group was not a significant moderator variable in any other CVD risk factors. Sensitivity analyses were performed by removing the single largest outlier for each variable. This resulted in a significant moderator effect in SBP effect estimate. For transparency, we report both regression results and provide scatterplots for the analysis with and without the outlying study.

Figure 4 shows the regression graph for change in VO_{2max} on the size of effect for SBP. The coefficient for change in VO_{2max} was −0.0286 (95% CI −1.3971 to 1.3400, *Z*-value −0.04, $Q = 0.00$, $P = 0.96$).

When the largest outlier was removed [65], the test of the model became significant (coefficient for change in VO_{2max}= −1.8180, 95% CI −3.6105 to −0.0254, *Z*-value −1.99, $Q = 3.95$, df = 1, $P = 0.047$). This indicates that for every one-unit increase in VO_{2max} (mL/kg/min), SBP is reduced by −1.8180 mmHg. Figure 5 shows the regression graph for change in VO_{2max} on the size of effect for SBP without the outlying study.

The variation of true effects about the regression line (T^2) is 11.5030 and the SD (T) is 3.3916. The $I^2 = 73.96\%$, indicating that 73.95% of variation around the line is due to variation in true effects rather than sampling error. In addition, the test for heterogeneity yielded *Q*-value of 49.92,

Table 6 Mean difference of CVD risk factors in RCTs comparing exercise versus control

Outcome	No. of studies	*N*	MD	Lower 95% CI	Upper 95% CI	I^2 (%)	*P* (overall effect)
FMD*	3	91	6.27	5.35	7.2	58	$P \leq 0.001$
SBP (mmHg)*	30	1637	−3.36	−5.64	−1.07	90	$P = 0.004$
DBP (mmHg)	31	1659	−2.23	−4.61	0.14	98	$P = 0.07$
Body Fat (%)*	34	1984	−2.15	−3.01	−1.29	77	$P \leq 0.001$
BMI*	46	2139	−0.59	−0.91	−0.26	54	$P \leq 0.001$
Body Mass (kg)*	47	2587	−1.24	−2.06	−0.42	59	$P = 0.003$
Fat mass (kg)*	18	1086	−2.79	−4.21	−1.36	69	$P \leq 0.001$
FFM (kg)*	8	465	2.15	1.15	3.14	29	$P \leq 0.001$
Total cholesterol (mmol/L)	28	1180	−0.15	−0.32	0.02	67	$P = 0.08$
HDL-C (mmol/L)*	29	1627	0.11	0.05	0.16	72	$P \leq 0.001$
LDL-C (mmol/L)*	27	1581	−0.09	−0.19	0	37	$P = 0.05$
TC: HDL*	6	303	−0.34	−0.59	−0.08	43	$P = 0.009$
Triglycerides (mmol/L)*	24	920	−0.14	−0.25	−0.02	75	$P = 0.02$
WC (cm)*	18	1064	−2.22	−3.46	−0.98	20	$P \leq 0.001$
HC (cm)	4	105	−3.62	−9.20	1.97	43	$P = 0.20$
WHR*	12	418	−0.02	−0.03	−0.02	39	$P \leq 0.001$

Effect estimates are reported as mean differences (MD) and 95% confidence intervals, between exercise and usual care groups. Heterogeneity reported using I^2 statistic. * = statistically favourable result. *FMD* flow mediated dilation, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *BMI* body mass index, *FFM* fat free mass, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol, *TC:HDL* total cholesterol: high-density lipoprotein cholesterol ratio, *WC* waist circumference, *HC* hip circumference, *WHR* waist-to-hip ratio.

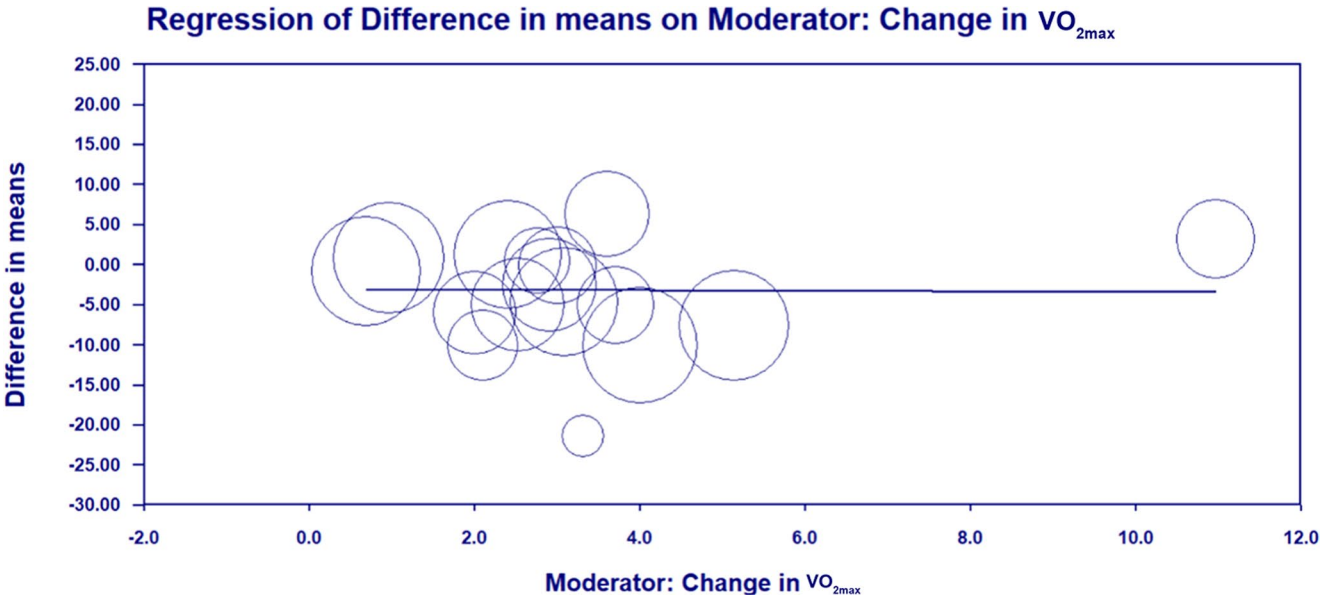

Fig. 4 Change in VO_{2max} as the moderator variable and effect estimate (difference between means) for exercise versus control for systolic blood pressure (16 observations)

df = 13, $P > 0.0001$. This indicates that the unexplained variance is not zero, and the covariate (change in VO_{2max}) cannot fully explain the variation in effects.

The R^2 analogue is 0.66, which indicated that the model could explain 66% of the variation in true effects.

Exercise and Diet Versus Diet

Meta-analyses were performed for all studies comparing exercise and diet combined versus diet alone (Table 7). A statistically significant effect was observed for exercise and diet versus diet alone on both relative and absolute VO_{2max}/VO_{2peak}, body fat %, fat mass (kg), and HC (cm). Only relative and absolute VO_{2max}/VO_{2peak}

Exercise and Supplement/Placebo Versus Supplement/Placebo Alone

Meta-analyses were performed for both exercise and supplement versus supplement, and exercise and placebo

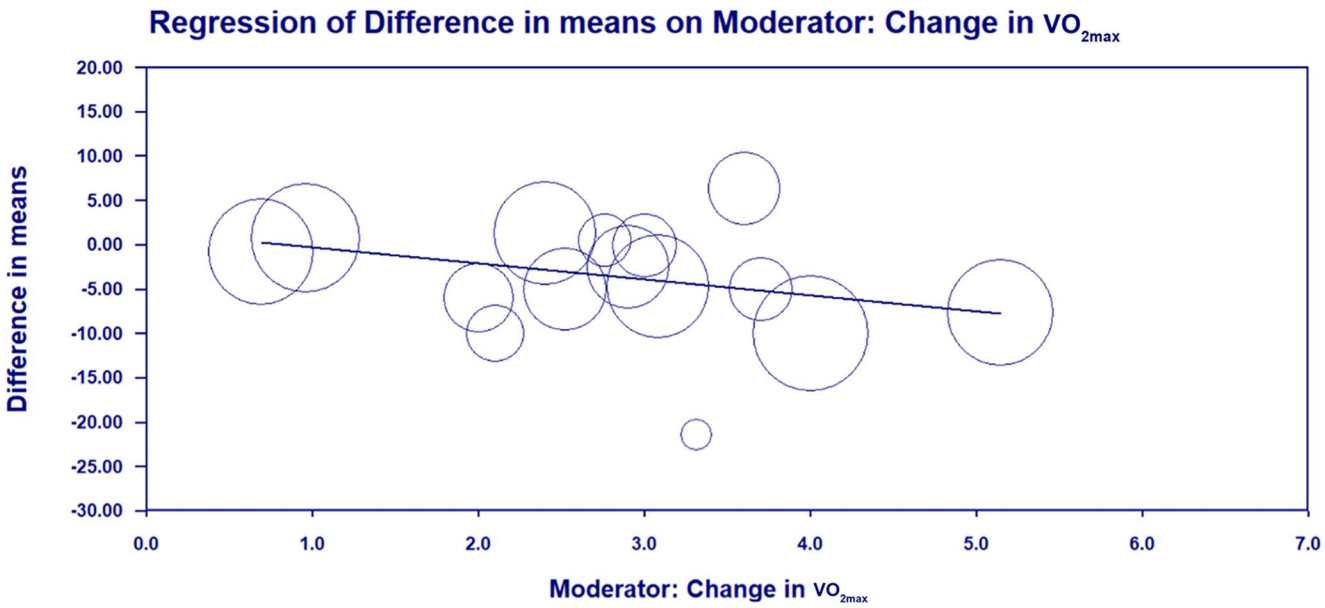

Fig. 5 Change in VO_{2max} as the moderator variable and effect estimate (difference between means) for exercise versus control for systolic blood pressure with outlying study removed (15 observations)

Table 7 Mean difference of CVD risk factors in RCTs comparing exercise and diet versus diet alone

Outcome	No. of studies	*N*	MD	Lower 95% CI	Upper 95% CI	I^2 (%)	*P* (overall effect)
VO_{2max}/VO_{2peak} (mL/kg/min)*	4	389	2.22	0.69	3.75	75	$P=0.004$
VO_{2max}/VO_{2peak} (L/min)*	2	184	0.16	0.04	0.28	60	$P=0.009$
SBP (mmHg)	4	355	0.18	−2.89	2.53	20	$P=0.91$
DBP (mmHg)	4	365	−1.47	−3.11	0.16	23	$P=0.08$
Body Fat (%)*	4	216	−0.9	−1.71	−0.09	0	$P=0.03$
BMI	5	282	−0.66	−1.34	0.03	35	$P=0.06$
Body Mass (kg)	8	564	−0.64	−1.37	0.1	0	$P=0.09$
Fat mass (kg)*	5	327	−1.53	−2.31	−0.75	0	$P\leq0.001$
FFM (kg)	2	73	−0.4	−2.57	1.77	0	$P=0.72$
Total cholesterol (mmol/L)	5	396	0.04	−0.22	0.15	26	$P=0.71$
HDL-C (mmol/L)	5	430	−0.02	−0.07	0.02	12	$P=0.37$
LDL-C (mmol/L)	5	430	0.01	−0.11	0.14	9	$P=0.83$
TC: HDL	2	196	−0.01	−0.2	0.22	0	$P=0.92$
Triglycerides (mmol/L)	5	430	0.06	−0.06	0.18	0	$P=0.35$
WC (cm)	3	221	−1.17	−2.41	0.08	0	$P=0.07$
HC (cm)*	2	202	−1.71	−3.41	−0.01	0	$P=0.05$
WHR	5	342	0	−0.01	0.01	18	$P=0.65$

Effect estimates are reported as mean differences (MD) and 95% confidence intervals, between exercise and diet combined versus diet alone. Heterogeneity reported using I^2 statistic. * = statistically favourable result. *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *BMI* body mass index, *FFM* fat free mass, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol, *TC:HDL* total cholesterol: high-density lipoprotein cholesterol ratio, *WC* waist circumference, *HC* hip circumference, *WHR* waist-to-hip ratio

versus placebo, with supplement/placebo as a consistent co-intervention. Tables 8 and 9 indicate the results of the meta-analysis for each available outcome, respectively. Only relative VO_{2max}/VO_{2peak} (mL/kg/min) displayed a statistically favourable effect of exercise and supplement versus supplement (MD 4.61 mL/kg/min, 95% CI 2.26 to 6.95, 2 trials, 49 participants, $I^2=0\%$) and exercise and placebo versus placebo (MD 3.36 mL/kg/min, 95% CI 0.47 to 6.26, 2 trials, 45 participants, $I^2=0\%$).

Discussion

Summary of Results

This review investigated the effects of PA and exercise interventions, both aerobic and resistance training, on VO_{2max} and cardiovascular risk factors in perimenopausal and postmenopausal women, across various comparisons. This is the first meta-analysis to investigate the impact of changes in

Table 8 Mean difference of CVD risk factors in RCTs comparing exercise and supplement versus supplement alone

Outcome	No. of studies	*N*	MD	Lower 95% CI	Upper 95% CI	I^2 (%)	*P* (overall effect)
VO_{2max}/VO_{2peak} (mL/kg/min)*	2	49	4.61	2.26	6.95	0	$P \leq 0.001$
SBP (mmHg)	3	65	−3.04	−10.79	4.71	0	$P = 0.44$
DBP (mmHg)	3	65	−0.9	−6.24	4.44	0	$P = 0.74$
Body Fat (%)	4	235	−0.39	−2.79	2.02	60	$P = 0.75$
BMI	5	249	0.3	−1.14	1.74	68	$P = 0.68$
Body Mass (kg)	4	225	0.64	−4.71	6	88	$P = 0.81$
Total cholesterol (mmol/L)	3	207	−0.01	−0.65	0.63	77	$P = 0.97$
HDL-C (mmol/L)	5	249	−0.04	−0.19	0.1	46	$P = 0.55$
LDL-C (mmol/L)	3	73	0.12	−0.35	0.6	37	$P = 0.61$
Triglycerides (mmol/L)	5	249	0.09	−0.2	0.37	68	$P = 0.54$

Effect estimates are reported as mean differences (MD) and 95% confidence intervals, between exercise and supplement combined versus supplement alone. Heterogeneity reported using I^2 statistic. * = statistically favourable result. *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *BMI* body mass index, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol

Table 9 Mean difference of CVD risk factors in RCTs comparing exercise and placebo versus placebo alone

Outcome	No. of studies	*N*	MD	Lower 95% CI	Upper 95% CI	I^2 (%)	*P* (overall effect)
VO_{2max}/VO_{2peak} (mL/kg/min)*	2	45	3.36	0.47	6.26	0	$P = 0.02$
SBP (mmHg)	3	59	2.77	−7.96	13.5	58	$P = 0.61$
DBP (mmHg)	3	59	−0.8	−5.42	3.81	0	$P = 0.78$
Body Fat (%)	3	72	−2.25	−5.03	0.53	41	$P = 0.11$
BMI	4	81	0.17	−1.34	1.68	40	$P = 0.82$
Body Mass (kg)	3	59	0.36	−3.16	3.89	0	$P = 0.84$
Total cholesterol (mmol/L)	2	42	0.53	−0.13	1.2	0	$P = 0.12$
HDL-C (mmol/L)	4	81	0.1	−0.06	0.27	0	$P = 0.21$
LDL-C (mmol/L)	3	67	0.23	−0.09	0.55	0	$P = 0.15$
Triglycerides (mmol/L)	4	81	−0.08	−0.58	0.41	79	$P = 0.74$

Effect estimates are reported as mean differences (MD) and 95% confidence intervals, between exercise and supplement combined versus supplement alone. Heterogeneity reported using I^2 statistic. * = statistically favourable result. *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *BMI* body mass index, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol

CRF in association with CVD risk factors in this population. We found statistically favourable improvements in VO_{2max}/VO_{2peak}, FMD, SBP, body fat %, BMI, body mass, fat mass, FFM, HDL-C, LDL-C, TC: HDL, triglycerides, WC, and WHR, in interventions comparing exercise alone versus control. In other comparisons, evidence suggested a statistically favourable increase in VO_{2max}/VO_{2peak}, body fat %, fat mass, and HC in exercise and diet versus diet alone. In comparisons of very limited studies, there was evidence to suggest a statistically favourable change in VO_{2max}/VO_{2peak} in studies comparing exercise and supplement versus supplement alone, and exercise and placebo versus placebo alone.

In meta-regression analyses, change in VO_{2max} in the exercise group was not a significant moderator variable in any other CVD risk factors. However, when sensitivity analyses were performed, the model for SBP became significant, indicating potential moderator effects of change in VO_{2max} on SBP.

Primary Outcome

In our main comparison (exercise versus control), PA interventions of all types (aerobic, resistance, or mixed) increased VO_{2max} in perimenopausal and postmenopausal women with a mean effect estimate of 3.51 mL/kg/min. Previous research has indicated that in the context of survival benefit, each 3.5 mL/kg/min increase in VO_{2max} (corresponding to one metabolic equivalent [MET]), is associated with a 10%–25% improvement in survival benefit in large, longitudinal studies [117]. For example, one study of 5721 asymptomatic women (aged 52.4 ± 10.8 years) indicated that each MET increase in exercise capacity was associated with a 17% reduction in all-cause mortality rate [49]. An additional 20-year follow-up study of 2994 asymptomatic women, aged 20–80 years, indicated a 20% reduction in risk of cardiovascular death for every MET of exercise capacity [91]. Thus, these data are of clinical significance and highlight the importance of PA interventions in this population that aim to increase CRF. Improvement of 1-MET (3.5 mL/kg/min) is achievable by most individuals [117] and is demonstrated in this review.

A previous meta-analysis of 56 studies including aerobic interventions in postmenopausal women found statistically significant favourable results for exercise versus control on VO_{2max} (SMD = 1.43, 95% CI 1.17 to 1.70) [61]. In addition, a previous systematic review investigating

exercise modalities on CRF in this population found that all PA types increase VO_{2max} (SMD: 1.15; 95% CI: 0.87, 1.42; $P=0.001$) [70]. These effect measures are presented as SMD which can be difficult to interpret in terms of the clinical relevance since SMD is expressed in units of SD, rather than an absolute estimate of intervention effect [58]. Our analysis strengthens the results of the latter review by including peri-menopausal women across all PA modalities. Our meta-analysis indicated an effect estimate of 3.51 mL/kg/min (MD) which provides an absolute estimation that can be used to assess clinical relevance. Resistance training is capable of increasing VO_{2max} through increases in muscle mass, myoglobin concentration, capillary density, and stroke volume in older adults [55, 81]. This suggests that incorporating strength or resistance training into interventions can have maximal benefits for health that include improved CRF (and improved survival), as well as hypertrophy and increases in bone density [105]. This lends further support and emphasis to the recommended two days per week of muscle strengthening activities recommended by the UK Chief Medical Officer and the World Health Organization [98, 145].

A statistical subgroup difference was detected for intervention length, with larger effects evidenced through interventions of three months or fewer in length. This does not appear related to greater reductions in body mass, as changes in VO_{2max} were not related to effect estimate for body mass in this study. There were also no subgroup differences for intervention length in relation to body mass. Although physiological adaptations to exercise can indeed occur in as little as 2–6 weeks of training [84], previous contrasting research indicates that greater improvements in CRF can be obtained with durations of more than 12 weeks [148]. However, adherence may be an influencing factor in the degree of improvement [90]. In fact, in this review, studies of >3months were more likely to report<80% adherence to the intervention (6/23 studies, 26%) compared to interventions of three months or fewer (1/18 studies, 6%). Thus, it may be poorer adherence in longer interventions that resulted in lesser improvements in CRF. This finding highlights the necessity of designing PA interventions that incorporate evidence-based behaviour change techniques (BCTs) into intervention design. BCTs influence the desired behaviours (PA behaviour), may improve adherence (and thus improve outcomes) and ultimately provide a foundation for long-term uptake of such behaviours beyond the intervention [87, 88].

In comparisons of exercise and diet versus diet alone, a statistically favourable but smaller effect was observed for relative VO_{2max}/VO_{2peak}. An explanation for the smaller effect measure may be that diet can cause reductions in body mass which can lead to improvements in relative VO_{2max}/VO_{2peak} (mL/kg/min) while absolute VO_{2max}/VO_{2peak} (L/min) remains stable [134]. In comparisons of exercise and supplement/placebo versus supplement/placebo alone, statistically favourable effects were observed with the addition of exercise. However, these were based on few studies with very small sample sizes and these results are likely to be influenced by small study effects.

Secondary Outcomes

In the comparison of exercise versus control, exercise produced a statistically favourable effect on body composition measures (body fat %, BMI, body mass, fat mass, fat free mass, WC, and WHR), vascular measures (FMD and SBP), and blood lipids (HDL-C, LDL-C, TC: HDL, and triglycerides). Although some effect estimates are modest (such as blood lipids), some results may represent a decrease in CVD risk. For example, the effect estimate for WC was −2.22 cm (95% CI −3.46 to −0.98) and WHR was −0.02 (95% CI −0.03 to −0.02) compared to control. Higher WC and WHR is associated with higher trunk fat, which, in turn, is associated with increased CVD risk in postmenopausal women [23]. A previous meta-regression analysis of prospective studies has demonstrated that each 1 cm increase in WC and each 0.01 increase in WHR increased the risk of a CVD event by 2% and 5%, respectively [77]. In addition, the effect estimate for FMD was 6.27% (95% CI 5.35 to 7.2). Previous research suggests that there is a 4% decrease in the risk of future CV events for every 1% increase in FMD in asymptomatic individuals [112]. This suggests that the effect estimates for body composition and FMD measures seen in this meta-analysis may represent clinically important reduction in risk.

For those variables eligible for subgroup analysis, mixed type interventions were statistically favourable for body fat %, BMI, body mass, HDL-C, triglycerides, and WHR compared to control with no statistical subgroup difference between exercise types. These results suggest that interventions combining both aerobic and resistance training modalities could be effective in improving a variety of CVD risk factors while also conferring benefits associated with resistance training, particularly in older adults [105].

A previous systematic review of aerobic exercise interventions in postmenopausal women found significant increase in FMD in controlled intervention studies, with significant association with improvement in blood pressure and VO_{2max} [17]. Another meta-analyses of aerobic exercise interventions found significant improvements in a multitude of body composition measures (BMI, WC, body fat), SBP, and blood lipids (LDL-C, HDL-C) [61]. The present review aligns with previously published results and widens the scope to indicate that mixed-type interventions can provide

similar benefits to aerobic interventions in terms of CVD risk in postmenopausal women.

There was a lack of evidence for a decrease in CVD risk factors in exercise and diet versus diet alone. Diet alone can be an effective mitigator of CVD risk factors in menopause [20] which may provide an explanation. However, these meta-analyses are based on fewer studies with smaller sample sizes, and small study effects may influence results. Regardless, an increase in CRF makes the addition of exercise to diet worthwhile since CRF is a predictor of long-term mortality independent of other risk factors [117].

Meta-regression

In meta-regression, VO_{2max} was found to moderate the effect estimate of SBP after removal of a single outlier (with fifteen studies remaining). This is a potentially clinically significant effect, with each 1 mL/kg/min increase in VO_{2max} resulting in a −1.82 mmHg decrease in SBP. Previous research indicates that the lowest risk for CVD is in the range of 120–124 mmHg. After this, risk increases with each 5-mmHg increment [19]. For example, in a network meta-analysis including 144,220 individuals, CVD risk was reduced by 29% for those with SBP 120–124 mmHg compared to 130–134 mmHg, and 42% compared to those with SBP of 140–144 mmHg [19]. Assuming a 1-MET increase in VO_{2max} (3.5 mL/kg/min), a reduction in SBP of −6.37 mmHg may be achievable, which could result in substantially reduced CVD risk.

Increases in VO_{2max} after long-term exercise training are largely due to an increase in stroke volume, which in turn is determined by cardiac and vascular structure and function [93]. These adaptations are also associated with improved endothelial function, including expression of endothelial nitric oxide synthase [141], which is critical for regulation of systemic blood pressure [143]. Previous research has also found an inverse association between CRF and blood pressure [24]. Indeed, this would indicate improvements in endothelial function (expressed as FMD) as found in this review and others [17], although there were too few studies to conduct meta-regression on the impact of changes in VO_{2max} on the effect estimate of FMD. This provides a plausible explanation for the findings in this review, but there remains a high degree of heterogeneity. In addition, the lack of association between changes in VO_{2max} and body mass indicates that improvements in VO_{2max} go beyond solely weight loss (when VO_{2max} is expressed as mL/kg/min).

VO_{2max} was not associated with changes in any other CVD risk factor analysed in this review, and sensitivity analysis did not affect the result. Previously, higher CRF has been shown to have a protective effect on overall mortality as well as inverse associations with traditional risk factors such as dyslipidaemia and diabetes mellitus [24]. There are several potential reasons for the lack of relationships in this study. First, the high heterogeneity present among the studies may have influenced the results and resulted in a poor model fit. Another reason may be mechanistic. The physiological mechanisms that influence CRF after exercise training (such as improved autonomic function, enhanced NO production, capillarisation [94]), overlap with those that influence SBP [35, 40], making an inverse linear relationship possible. Moreover, SBP is responsive to exercise interventions as little as two weeks in duration [36]. In contrast, lipoproteins and lipids, for example, may have fewer overlapping mechanisms with changes in CRF and may require additional components, such as dietary, or specific exercise intensities, to yield improvements [69]. This is reflected in our review where effect sizes for blood lipids were modest, with wide CIs. Intervention length may also be a factor; in this review, the longest interventions did not exceed 12 months in duration. Conversely, longitudinal studies that quantify risk of cardiovascular events and all-cause mortality in association with CRF have follow-up durations of up to 26 years [76, 111]. Regardless, statistically significant improvements in CVD risk factors were noted, as well as improvements to VO_{2max}, in the present review. The lack of specific association could suggest that the mechanisms that regulate some CVD risk factors are downstream to those that result in direct exercise-induced improvements in VO_{2max} (such as stroke volume, endothelial function, and autonomic function) rather than mechanistically related. The implication is that changes in CRF may not necessarily be a useful proxy for reductions in other CVD risk factors over short-term intervention durations. While meta-regression is a useful first step, further research is needed to explore this relationship.

Strengths and Limitations

This review examines a large body of evidence (78 trials and 5332 participants) to analyse the effects of PA interventions on CRF and CVD risk in perimenopausal and postmenopausal women. The key strengths of this review are the investigation of the association between changes in CRF and CVD risk factors, and the estimation of changes in CRF as MD which builds upon previous reviews [70] to provide clinical context. In addition, it is the first review to include comparisons of exercise and diet versus diet alone, and exercise versus supplement/placebo versus supplement/placebo alone, making it a comprehensive review of PA trials in menopausal cohorts. There are various limitations of this study. Limitations of included studies include high risk of detection and attrition bias, and a lack of reporting leading to unclear bias across domains, which limits the overall

quality of the body of evidence. Limitations of the review process include the use of English-only reports, potentially missing data which we could not obtain, and publication bias for the primary outcome, which could suggest that missing, unpublished data could affect the results. Additionally, comparisons of exercise and diet versus diet alone, and exercise versus supplement/placebo versus supplement/placebo alone, had fewer studies with smaller sample sizes, which may have resulted in small-study effects. Also, some a priori subgroup analyses, such as HRT and menopause status, could not be completed due to insufficient data. In fact, only two studies out of 78 included perimenopausal women, and thus the results have limited applicability to this sub-population. Finally, subgroup analysis (including meta-regression) is observational in nature and causal relationships cannot be elucidated. The results of this review thus provide a foundational basis for future research.

There was high and unexplained heterogeneity for the primary outcome and for many secondary outcomes. Post hoc analysis reduced this for some variables, and sensitivity analyses for the primary outcome indicated that heterogeneity may be explained by a small number of influential studies. However, the post hoc nature of the analysis limits interpretability [135]. It is possible that heterogeneity is a result of the diverse modalities of PA (Table 2) within the subcategories of 'aerobic', 'resistance' and 'mixed', including sports, exercise modes, and varied prescriptions of intensity, which made it challenging to combine and categorise by exercise intensity. In addition, the studies included a wide age range (47 to 76 years) which could lead to differing physiological profiles. These results should therefore be interpreted with caution.

Future reviews and meta-analyses could investigate the impact of different PA intensities on CRF and CVD and consider the use of age as a moderator variable in meta-regression in this population. Future RCTs should aim to recruit beyond the categories of 'premenopausal' and 'postmenopausal'; there is a paucity of literature involving those going through the perimenopausal transition despite this time-period being crucial for the development of CVD risk. In addition, future RCTs in this area should be robustly designed to reduce bias and reported according to international standards to allow for higher confidence in findings.

Conclusions

Our results indicate that PA interventions of all types (aerobic, resistance, and mixed) elicit meaningful improvements in CRF compared to non-active controls. CRF is inversely related to CVD risk and thus these results suggest that PA, either alone or as a co-intervention, could reduce CVD risk in the presence of improved VO_{2max}. This provides additional support for mixed-type interventions that confer benefits of both aerobic and resistance exercise. In addition, improvements in VO_{2max} could potentially lead to reductions in SBP. For $VO_{2max,}$ larger effect estimates were seen in shorter interventions of fewer than three months, but this may indicate better adherence to interventions of shorter length. In other comparisons, exercise in combination with a co-intervention (diet, supplement, placebo) was more effective at increasing VO_{2max} than the co-intervention alone. High unexplained heterogeneity was present. Some potential sources of heterogeneity that were not investigated include PA intensity and age. Future interventions should incorporate evidence-based BCTs to optimise adherence and subsequent long-term uptake of PA behaviour. Future RCTs should focus on robust design and reporting.

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Data Availability Data is available from the York St John University's open data repository, RaYDaR: https://figshare.com/s/778ddf79fb9cbfedbc00.

Declaration

Conflict of interest The authors have no relevant financial or non-financial interests to disclose.

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