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REVIEW

Optimal Dose and Modality of Exercise for Reducing Visceral Adipose Tissue in Individuals with Overweight or Obesity: A Systematic Review and Dose–Response Network Meta-Analysis

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Received: 6 March 2026 | **Revised:** 18 September 2026 | **Accepted:** 13 October 2026

Keywords: dose–response | exercise | model-based network meta-analysis | obesity | visceral adipose tissue

ABSTRACT

Background: The aim of this study is to evaluate the comparative efficacy of exercise modalities and doses for reducing visceral adipose tissue (VAT) in individuals with overweight or obesity. Six databases (PubMed, Embase, Web of Science, Scopus, Cochrane Library, and EBSCO) were searched through October 2025, with supplementary searching on August 18, 2026. Two reviewers independently assessed study quality. Randomized controlled trials were synthesized using random-effects pairwise, network, and dose–response meta-analyses.

Results: We included 78 reports involving 4930 participants. Mean age and body mass index (BMI) were 43.1 ± 19.2 years and 30.7 ± 4.8 kg/m^2, respectively; 58.4% were female. Network meta-analysis showed the largest estimated effects versus controls for vigorous aerobic exercise (AE [V]; SMD −0.73, 95% CI −0.96 to −0.51) and high-intensity interval training (HIIT; SMD −0.73, 95% CI −0.94 to −0.53). These were followed by combined exercise (CBE; SMD −0.61, 95% CI −0.81 to −0.41), moderate to vigorous intensity aerobic exercise (AE [MV]; SMD −0.53, 95% CI −0.67 to −0.39), and resistance training (RT; SMD −0.50, 95% CI −0.72 to −0.28). Total exercise dose showed a U-shaped relationship with VAT improvement; the fitted 95% credible interval was below zero at approximately 99–1225 MET-min/week. The model-estimated dose threshold for achieving the Substantial Clinical Benefit Threshold (SCBT; −0.89) was approximately 440 MET-min/week.

Conclusion: Exercise reduces VAT, with AE (V) and HIIT yielding the largest estimated effects versus controls. Approximately 750 MET-min/week appears to yield the greatest model-predicted reduction. Research should refine dose-specific effects on VAT and body composition to support personalized prescriptions.

1 | Introduction

Obesity and its complications have become a severe challenge to global public health, and its prevalence has shown a continuous upward trend over the past few decades [1, 2]. Although body mass index (BMI) is often used as a surrogate marker for metabolic health, increasing pathophysiological evidence suggests that the anatomical distribution of adipose tissue has a far greater impact on health risks than total body fat [3, 4]. As the primary form of ectopic fat deposition, visceral adipose tissue (VAT) possesses high metabolic activity and serves as a central pathological engine driving insulin resistance, cardiovascular disease, and metabolic syndrome by secreting proinflammatory cytokines and interfering with free fatty

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acid metabolism [5, 6]. In contrast, subcutaneous fat is relatively metabolically inert, which explains why some individuals with obesity can maintain metabolic health whereas some normal-weight individuals face extremely high cardiovascular risks [7]. Therefore, precise intervention targeting VAT has become a clinical priority independent of weight loss.

As the cornerstone of lifestyle intervention, regular physical activity has been proven to effectively mobilize visceral fat reserves [8, 9], and the World Health Organization (WHO) as well as various national guidelines accordingly recommend that adults perform 150–300 min of moderate-intensity exercise per week to maintain health [10, 11]. Exercise for weight management also features prominently in the 2026 American College of Sports Medicine (ACSM) fitness trends report [12]. Yet the therapeutic value of exercise extends beyond weight control to metabolically important fat depots, as illustrated by reductions in intrahepatic lipid content following exercise in adults with or without nonalcoholic fatty liver disease (NAFLD) [13]. Translating these benefits into a VAT-specific prescription requires clarification of both exercise modality and dose. Although aerobic exercise (AE) serves as the established benchmark for VAT mobilization [14], the independent efficacy of resistance training (RT) through lean mass accretion remains a subject of intense debate [15, 16]. Nevertheless, in adults with overweight or obesity and Type 2 diabetes mellitus (T2DM), RT improves selected glycemic and lipid indices relative to nonexercising standard care, despite a nonsignificant pooled effect on body weight [17]. Similarly, improvements in insulin resistance and cardiorespiratory fitness with AE-RT protocols in adults with overweight or obesity without diagnosed comorbidities support the cardiometabolic value of combined exercise (CBE) [18], although the theoretical synergy between AE-induced lipolysis and RT-mediated metabolic preservation has not yet been consistently validated for VAT reduction. Furthermore, discordant evidence persists regarding whether high-intensity interval training (HIIT) offers a superior benefit-to-time ratio compared to continuous aerobic exercise [19–21]. Beyond modality selection, the quantitative relationship between exercise dose and VAT reduction for individual exercise modalities remains incompletely characterized. Comparative evidence of VAT reductions with AE, HIIT, and combined AE-RT in adults with prediabetes or T2DM informs modality selection, but the corresponding network meta-analysis did not examine how these effects varied with dose [22]. Previous studies have mostly treated exercise dose as a simple categorical variable or assumed it follows a linear relationship [23], which may obscure the nonlinear characteristics common in biological adaptation, such as the minimum effective threshold or plateau effects at high doses [24]. Due to limited evidence comparing multiple exercise modes and their dose–response relationships within a unified framework, it is currently difficult to find the optimal balance between efficacy and adherence.

This study is the first to employ Bayesian Model-Based Dose–Response Network Meta-Analysis to systematically evaluate the comparative efficacy of different exercise modalities in reducing VAT and other anthropometric indices in individuals with overweight or obesity and to plot dose–response curves [25, 26]. By identifying the minimum effective dose that triggers clinically significant benefits and the optimal dose, and combining this with estimates of the Substantial Clinical Benefit Threshold (SCBT), this study attempts to provide evidence-based, quantifiable, and precise exercise prescriptions for metabolic health management, thereby optimizing the control of VAT and other metabolic risks in the populations with obesity [27].

2 | Methods

2.1 | Protocol and Registration

The design and implementation of this study followed a pre-established protocol, which has been registered on the PROSPERO (Registration Number CRD420251139974). To ensure methodological rigor and reporting transparency, this meta-analysis was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension statement for Network Meta-Analyses (PRISMA-NMA) [28] and the Cochrane Handbook for the Systematic Review of Interventions [29]. Furthermore, because all data used in this study were based on previously published studies, ethical approval and patient informed consent were not required.

2.2 | Search Strategy and Study Selection

A systematic search was performed in PubMed, Web of Science, the Cochrane Library, EBSCO, Embase, and Scopus databases from their inception to October 2025, with an updated search in August 2026, to identify studies published globally in any language assessing the effects of different exercise modalities on VAT in individuals with overweight or obesity. Additionally, published reviews and meta-analyses in relevant fields were manually searched to identify potentially missing studies. The included-study lists of these reviews were cross-checked against our original list of included studies, and potentially eligible additional studies underwent full-text assessment. Population eligibility was determined according to our inclusion criteria using information reported in the full text and baseline characteristics. The search strategy was constructed around the PICOS framework using controlled vocabulary and free-text terms: (P) participants: individuals with overweight and/or obesity; (I) interventions: exercise and physical activity; (C) comparisons: other exercise modalities or intensities, no exercise, or nonexercise interventions; (O) outcomes: primary outcome: VAT; secondary outcomes: total body fat (TBF), weight, and body mass index (BMI); and (S) study design: randomized controlled trials. The detailed search strategy is presented in Appendix S2. Two investigators (Y.D. and D.L.) independently screened the titles and abstracts of the literature and subsequently assessed the full texts of potentially eligible studies. Any discrepancies were resolved through discussion with a third investigator (XW). EndNote X9 software was used for the screening and management of the retrieved literature.

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2.3 | Eligibility Criteria

Included studies were required to meet all the following criteria:

1. Study participants met the diagnostic criteria for overweight or obesity without age restrictions. For adults, the BMI thresholds used for inclusion in this review were $\geq 25\,kg/m^2$ (overweight) or $\geq 30\,kg/m^2$ (obesity) for European and Black populations, and $\geq 24\,kg/m^2$ (overweight) or $\geq 28\,kg/m^2$ (obesity) for Asian populations. For studies where BMI was not reported, body fat percentage ($\geq 30\%$ for females and $\geq 25\%$ for males) was accepted as the basis for inclusion. Trials focusing on comorbidities, such as type 2 diabetes, prediabetes, nonalcoholic fatty liver disease/nonalcoholic steatohepatitis (NAFLD/NASH), or metabolic syndrome, were also eligible provided that participants met the review's overweight or obesity criteria and all other inclusion criteria were satisfied.
2. The study design compared exercise interventions of different types or intensities or compared exercise interventions with a control group. Exercise was defined as planned, structured, and repetitive physical activity [30]. Exercise interventions were categorized into five distinct types based on existing research (Table S1). Considering that physiological adaptation and stable therapeutic effects typically require a minimum of 4 weeks of intervention [34], the intervention duration of included studies was required to be at least 4 weeks.
3. The control group received any form of nonexercise intervention, such as usual care or a waitlist.
4. Study outcomes included changes in VAT or related visceral adiposity indicators. Eligible measures included VAT assessed using magnetic resonance imaging (MRI), computed tomography (CT), or dual-energy X-ray absorptiometry (DXA), as well as ultrasonographic intra-abdominal fat thickness, bioelectrical impedance analysis (BIA)–derived visceral fat estimates, and formula-derived visceral adiposity indices.
5. The study type was a randomized controlled trial (RCT).

Studies meeting any of the following criteria were excluded:

1. Acute studies with an intervention duration of less than 4 weeks.
2. Exercise interventions combined with other intervention measures. However, in the present review, studies were eligible for inclusion if the intervention and control groups received identical cointerventions, thereby isolating the independent effects of exercise. Studies were excluded if exercise was combined with dietary interventions but the control group did not receive equivalent dietary interventions.
3. Studies utilizing a crossover design.
4. Duplicate publications, conference abstracts, study protocols, or review articles.
5. Studies from which valid data could not be obtained or extracted.

2.4 | Data Extraction

Two researchers (YD and DL) independently extracted data from the included studies using a predesigned form. Any disagreements were resolved through consultation with a third researcher (X.W.). The extracted data included first author information, publication year, participant demographic characteristics (such as age, gender, total number of participants, BMI, and body fat percentage), detailed information on interventions (type, duration, frequency, and period), and the primary and secondary outcome indicators of interest. The measurement methods and units of VAT-related outcomes were also recorded. If study data were incomplete, we attempted to contact the corresponding author via email to request the necessary information.

2.5 | Data Coding and Management

To standardize exercise dose across different studies, we adopted "MET-min/week" as the standardized dose unit. The dose calculation method was defined as follows: exercise dose (MET-min/week) = metabolic equivalents of tasks (MET) × duration of a single exercise session × frequency per week, where MET values for exercise types were assigned based on the 2011 Compendium of Physical Activities [35]. Specific rules were followed for the coding of primary exercise intervention duration: Only core exercise time was extracted, whereas time for warm-up and cool-down was excluded. Intervention durations reported ambiguously or using progressive protocols were averaged to ensure data consistency. Finally, to facilitate the construction of the dose–response model, all calculated continuous dose values were categorized into six preset levels: 0, 300, 600, 900, 1200, and 1500 MET-min/week, thereby ensuring network connectivity and adequate sample size for each dose level [36]. Specific dose categorizations and corresponding raw information for each study are listed in Appendix S20.

2.6 | Risk of Bias and Certainty of Evidence

Two researchers (Y.D. and D.L.) assessed the risk of bias (ROB) of included studies using the Cochrane Risk of Bias tool [37]. This tool comprises seven distinct domains: (a) random sequence generation, (b) allocation concealment, (c) blinding of participants and personnel, (d) blinding of outcome assessment, (e) incomplete outcome data, (f) selective reporting, and (g) other bias. During the assessment, we specifically considered the inherent difficulty of blinding participants in exercise intervention studies. Therefore, we excluded this domain from the overall risk of bias rating and considered "blinding of outcome assessment" as a critical quality criterion. The overall risk of bias for each study was determined according to the following criteria: low ROB if no more than three domains were rated as unclear risk and all remaining domains were rated as low risk; moderate ROB if exactly one high-risk domain existed or if no high-risk domains existed but four or more domains were rated

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as unclear risk; and high ROB if two or more high-risk domains existed [38]. We employed the Grading of Recommendations, Assessment, Development and Evaluation (GRADE) framework to evaluate the certainty of evidence for the effect estimates of exercise interventions derived from pairwise and network meta-analyses [39].

2.7 | Measures of Treatment Effect

The primary outcome of this study was the change in VAT, and secondary outcomes included changes in TBF, weight, and BMI. We prioritized the mean difference of the "change from baseline" as the effect size unless the original text only reported endpoint differences. For outcomes with consistent measurement units (weight and BMI), we used the weighted mean difference (MD); for outcomes with different measurement units or methods (VAT and TBF), the standardized mean difference (SMD) was adopted to enhance comparability. When the standard deviation of the change was not reported, we calculated the change standard deviation using the formula recommended by the Cochrane Handbook by assuming a correlation coefficient of $r = 0.5$ between baseline and follow-up measurements to reflect moderate measurement repeatability and obtain a relatively conservative estimate [29]. All effect sizes were reported with 95% confidence intervals (CI); for the MBNMA model, due to the Bayesian framework, 95% credible intervals (CrI) were reported.

2.8 | Statistical Analysis

2.8.1 | Pairwise Meta-Analyses

We used random-effects models to conduct pairwise meta-analyses, pooling effects on VAT, TBF, weight, and BMI for comparisons of each exercise modality with a control or another exercise modality. For comparisons supported by a single study, the study-specific effect estimate and 95% confidence interval were presented without pooling. Heterogeneity was assessed using Cochran's Q test and the I^2 statistic, where an I^2 value greater than 50% was generally considered to indicate substantial statistical heterogeneity [40]. Potential small-study effects or publication bias were evaluated by visually inspecting funnel plot symmetry (Appendix S7). Comparison-adjusted funnel plots and Egger's regression test were used for the network meta-analyses (Appendix S13) [41].

2.8.2 | Network Meta-Analyses

Before conducting the network meta-analysis, we first compared the distribution of potential effect modifiers across studies, such as baseline age, sex ratio, BMI, VAT levels, and intervention duration, to verify the fundamental assumptions of transitivity and result generalizability [42]. Subsequently, network plots were generated to visually summarize the direct comparisons between different exercise types and the control group. Within the frequentist framework, we employed a random-effects network meta-analysis using no exercise or lifestyle maintenance as the common comparator [43]. Changes in outcome indicators were estimated as SMD or MD, both reported with 95% confidence intervals, and the effects of each exercise type relative to the control group were displayed via forest plots. To compare the overall efficacy of various exercise modalities, we calculated P scores based on the network model to rank the relative therapeutic effects of different exercise types in improving VAT and other outcomes [44]. P scores range from 0 to 1, where a higher value indicates a superior ranking of that exercise for the corresponding outcome [42]. Network heterogeneity was assessed using τ^2 and I^2 statistics, and predictive intervals were added to forest plots to visualize the potential range of true effects. Network inconsistency was tested using the node-splitting method and the "design-by-treatment interaction" model [36, 45]. If significant inconsistency was found, it was addressed in the sensitivity analysis and discussion of results. Network meta-analyses were performed using the "netmeta" package in R software.

2.8.3 | Dose–Response Network Meta-Analyses

A Model-Based Network Meta-Analysis (MBNMA) based on a random-effects Bayesian model was employed to evaluate the dose–response relationship between different exercise doses and improvements in VAT, TBF, Weight, and BMI [25] Prior to analysis, we checked network transitivity [36] connectivity [46] and data consistency [47] to ensure that the fundamental assumptions of MBNMA were met (Appendix S15). Regarding the selection of dose–response functions, we fitted multiple models including Emax, restricted cubic spline, exponential, and quadratic functions [26]. Model goodness-of-fit was comprehensively evaluated using indicators such as the deviance information criterion (DIC), between-study standard deviation (SD), residual deviance, and the number of parameters (pD). For VAT and other outcomes, random-effects quadratic models were selected for primary reporting based on model fit, convergence, and clinical plausibility (Appendix S16). In the MBNMA, doses were modeled using precategorized MET-min/week levels. We estimated the effect values and their 95% CrI for different dose levels relative to the control group and further calculated the potential minimum effective dose (defined as the dose point where the upper limit of the CrI first falls below 0) and the "plateau" of benefit at high doses [48]. MBNMA analysis was performed using the "MBNMA dose" package in R, and dose–response curves were generated using the "ggplot2" package.

2.8.4 | Additional Analyses

We conducted univariable random-effects meta-regression to explore associations between potential effect modifiers and the overall intervention effect on VAT. Baseline body fat percentage, intervention duration, intervention frequency, and percentage of females were entered separately as continuous covariates. Between-study heterogeneity was estimated using restricted maximum likelihood, and regression coefficients were reported with 95% confidence intervals and p values. We additionally conducted network meta-regression to examine associations between these covariates and the relative effects of different exercise modalities.

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To enhance the clinical applicability of the findings, we estimated the exercise dose required to achieve the SCBT for VAT improvement in individuals with overweight or obesity [49]. The specific procedure was as follows:

1. For studies providing the required standard deviation data, baseline and change-score standard deviations were pooled separately across available trial arms within each study. Corresponding data within each study were expressed in consistent measurement units. The pooled standard deviations were calculated using the sample-size-weighted variance formula:

$$s_{i,t} = \sqrt{\frac{\sum_j (n_{ij,t} - 1) s_{ij,t}^2}{\sum_j (n_{ij,t} - 1)}}$$

Here, i denotes the study, j denotes an arm with available data, and t denotes baseline or change scores. The terms $n_{ij,t}$ and $s_{ij,t}$ represent the corresponding sample size and standard deviation, respectively.

2. For each study, the distribution-based threshold was defined as 0.5 times its pooled baseline standard deviation. This value was divided by the pooled change-score standard deviation to express the threshold on a standardized scale. Because negative values indicate VAT reductions, study-specific thresholds and the overall SCBT were calculated as follows:

$$\mathrm{SCBT}_i = -\frac{0.5 s_{i,\mathrm{baseline}}}{s_{i,\mathrm{change}}}$$

$$\mathrm{SCBT} = \frac{1}{K} \sum_{i=1}^{K} \mathrm{SCBT}_i$$

Here, K denotes the number of studies providing the required data, and the overall SCBT is the arithmetic mean of the study-specific thresholds. Given the absence of a universally accepted absolute clinical threshold for exercise-induced VAT reductions, we adopted this distribution-based approach as a reference for dose interpretation.

3. The SCBT was applied to the dose–response curves predicted by the network model. Intersections between the posterior median curve and the SCBT identified model-estimated doses at which the threshold was reached. These were considered alongside intervals with an upper 95% CrI below zero to identify dose ranges satisfying both criteria. Crossing doses were obtained by interpolation between adjacent prediction points.

We further assessed robustness by excluding studies at high risk of bias and examining whether effect estimates changed substantially. To assess the influence of dietary restrictions, we excluded studies in which both intervention and control groups received energy restriction and examined the stability of exercise rankings. We additionally repeated the VAT network meta-analysis after excluding studies using nonreference or indirect VAT measurement methods. Effect estimates and exercise modality rankings were compared with those of the main analysis to assess the robustness of the results to measurement methods (Appendix S12).

3 | Results

3.1 | Study Selection and Characteristics

The study selection process is illustrated in Figure S1. A total of 2884 records were retrieved from electronic databases. After duplicate removal and title/abstract screening, 130 reports were assessed for eligibility, of which 58 were excluded. Review cross-checking and backward citation tracking yielded six additional eligible reports. Ultimately, 78 reports of randomized controlled trials were included. These reports involved 4930 participants, with 2879 (58.4%) recorded as female. The pooled mean age was 43.1 ± 19.2 years. The pooled mean BMI was $30.7 \pm 4.8\,\mathrm{kg/m^2}$. Exercise frequency ranged from two to seven sessions per week, and intervention periods included in the analyses ranged from 8 to 64 weeks. A summary of the characteristics of all included studies is provided in Appendix S3.

3.2 | Risk of Bias

Among the 78 study reports included in this meta-analysis, 44 were judged to have a low ROB, 31 had a moderate ROB, and 3 were classified as high ROB. Detailed information regarding the ROB assessment is presented in Appendix S4.

3.3 | Certainty of Evidence

The certainty of evidence for the majority of comparisons of VAT in this study ranged from very low to moderate levels. Similarly, the certainty of evidence for most comparisons regarding TBF, weight, and BMI ranged from very low to moderate levels, with a small proportion of comparisons supported by high-quality evidence. Appendix S14 summarizes the specific results of the evidence quality assessment.

3.4 | Pairwise Meta-Analyses

First, pairwise meta-analyses were conducted, and forest plots for VAT based on exercise modality were provided, containing detailed trial-level information (Figure S2). Compared with the control group, AE (MV) (SMD = −0.55, 95% CI: −0.69 to −0.42, $I^2 = 38\%$), AE (V) (SMD = −0.72, 95% CI: −0.98 to −0.45, $I^2 = 60\%$), CBE (SMD = −0.61, 95% CI: −0.92 to −0.30, $I^2 = 74\%$), HIIT (SMD = −0.70, 95% CI: −1.03 to −0.37, $I^2 = 69\%$), and RT (SMD = −0.60, 95% CI: −0.93 to −0.26, $I^2 = 71\%$) all significantly reduced VAT, although heterogeneity varied across comparisons. Funnel plots are presented in Appendices S7 and S13, and Egger's test showed no statistical evidence of small-study effects ($p = 0.460$).

Forest plots for secondary outcomes (TBF, weight, and BMI) are shown in Appendix S6. The results indicated that, except for RT, which showed no significant effect on BMI compared

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with the control group, all other exercise categories significantly reduced TBF, weight, and BMI in individuals with overweight or obesity. Egger's test showed no statistical evidence of small-study effects for TBF or Weight but suggested possible small-study effects for BMI ($p = 0.042$). The corresponding funnel plots and test results are presented in Appendices S7 and S13.

3.5 | Network Meta-Analysis

Regarding changes in VAT, the network meta-analysis synthesized data from 78 study reports, with an analytical sample of 4930 participants. Compared with the control group, all five exercise modalities elicited significant reductions in VAT (Table S2). Specifically, AE (V) (SMD = −0.73, 95% CI: −0.96 to −0.51) and HIIT (SMD = −0.73, 95% CI: −0.94 to −0.53) yielded the largest estimated reductions in VAT, followed by CBE (SMD = −0.61, 95% CI: −0.81 to −0.41), AE (MV) (SMD = −0.53, 95% CI: −0.67 to −0.39), and RT (SMD = −0.50, 95% CI: −0.72 to −0.28). Cumulative ranking curves for the exercise modalities are presented in Figure S3; *P*-score rankings placed HIIT (*P* score = 0.845) and AE (V) (*P* score = 0.842) highest (Appendix S10). Moderate-to-high between-study heterogeneity was observed ($\tau^2 = 0.1240$, $I^2 = 55.6\%$). The global inconsistency test using the random-effects design-by-treatment interaction model was not statistically significant ($Q = 22.14$, $p = 0.775$), and local node-splitting analyses revealed no significant discrepancies between direct and indirect evidence (Appendix S9).

We also conducted NMA for secondary outcomes to explore whether the effects of different exercise modalities on these outcomes were consistent with their effects on VAT. A total of 62 ($N = 3218$), 65 ($N = 3302$), and 54 ($N = 2404$) study reports evaluated TBF, weight, and BMI, respectively. Compared with the control group, AE (MV), AE (V), CBE, and HIIT all significantly reduced TBF, weight, and BMI. RT significantly reduced TBF and BMI in individuals with overweight or obesity but had no statistically significant effect on body weight. *P*-score rankings indicated that AE (MV) ranked highest for TBF reduction, AE (V) ranked highest for weight reduction, and HIIT ranked highest for BMI reduction (Table S2 and Appendix S10).

3.6 | Dose–Response NMAs

We observed a nonlinear U-shaped dose–response relationship between total exercise volume and VAT (Figure S4). The fitted curve's 95% credible interval was below zero across approximately 99–1225 MET-min/week, with the maximum predicted effect occurring at 750 MET-min/week (SMD = −1.10, 95% CrI: −1.46 to −0.71). Using a distribution-based calculation, the SCBT for VAT reduction in individuals with overweight or obesity was SMD = −0.89, and the minimum predicted exercise dose required to reach this threshold was approximately 440 MET-min/week. With 600 MET-min/week used as the reference dose for the lower limit of WHO-recommended physical activity [10], the predicted mean effect was SMD = −1.05 (95% CrI: −1.43 to −0.66). This provides a comparative framework for analyzing the effectiveness of different exercise doses.

Figure S5 presents the dose–response relationships for the five exercise modalities. AE (MV), AE (V), CBE, and HIIT all showed U-shaped dose–response relationships with VAT, whereas RT showed a nonlinear inverse relationship. All five exercise modalities had dose ranges within which the 95% credible intervals were below zero. The estimated minimum effective dose was lowest for HIIT (130 MET-min/week, SMD = −0.26), followed by AE (V) (269 MET-min/week, SMD = −0.70), RT (471 MET-min/week, SMD = −0.68), AE (MV) (472 MET-min/week, SMD = −0.73), and CBE (624 MET-min/week, SMD = −1.08). Ranking analysis showed that AE (V) at 750 MET-min/week ranked highest among the exercise modality–dose combinations compared (Appendix S18). Appendix S19 presents the predicted effects of reaching the lower limit of WHO-recommended physical activity [10]. In addition, Figure S6 provides exercise recommendation schemes based on the predicted dose–response results for VAT improvement with different exercise modalities.

Additionally, we analyzed the dose–response relationships of total exercise volume and different exercise modalities with secondary outcomes. The overall dose–response curves for TBF and BMI generally followed the pattern observed for VAT, with effects emerging at lower doses, strengthening as dose increased, and attenuating at higher doses; body weight continued to decline across the evaluated dose range. Dose–response patterns varied across exercise modalities. HIIT and CBE both showed nonlinear dose–response relationships with TBF and body weight, with effects strengthening as dose increased within certain dose ranges; the predicted body weight benefit of HIIT tended to plateau at higher doses. AE (V) showed U-shaped curves across all secondary outcomes, and AE (MV) and RT showed similar trends for some outcomes, but RT showed no statistically significant effective dose range for either body weight or BMI (Appendix S17).

3.7 | Network Meta-Regression and Sensitivity Analyses

Overall meta-regression revealed that baseline body fat percentage significantly moderated the effect of exercise interventions on VAT (Figure S7). Specifically, study populations with lower baseline body fat percentages showed greater VAT reductions following exercise interventions, whereas higher baseline body fat percentages were associated with attenuated VAT reductions ($\beta = 0.031$, 95% CI: 0.008–0.054). Intervention duration, exercise frequency, and the proportion of female participants did not show significant moderating effects. Furthermore, we analyzed the associations between study characteristics and the efficacy of different exercise modalities. Regarding VAT, a higher proportion of female participants was associated with reduced VAT-lowering efficacy of AE (MV) ($\beta = 0.21$, 95% CI: 0.01–0.42) and RT ($\beta = 0.29$, 95% CI: 0.04–0.53, $p = 0.023$). For TBF, higher baseline body fat percentages were associated with greater fat loss effects of AE (V) ($\beta = -0.33$, 95% CI: −0.62 to −0.04), whereas higher exercise frequency was associated with greater fat loss effects of RT ($\beta = -0.34$, 95% CI: −0.66 to −0.03). Regarding body weight, higher baseline body fat percentages were associated with attenuated weight loss effects of AE (MV) ($\beta = 0.93$, 95% CI: 0.19–1.66) and enhanced weight loss effects of AE (V)

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($\beta = -0.79$, 95% CI: −1.17 to −0.40); higher exercise frequency was associated with attenuated weight loss effects of AE (MV) ($\beta = 0.54$, 95% CI: 0.01 to 1.07). For BMI, a higher proportion of female participants was associated with reduced efficacy of AE (V) ($\beta = 1.17$, 95% CI: 0.58–1.76) (Appendix S11).

Sensitivity analyses showed that the ranking of exercise modalities for VAT remained unchanged after excluding studies with a high risk of bias, supporting the robustness of the results. After excluding studies involving concurrent caloric restriction, VAT effect estimates and exercise modality rankings also remained broadly consistent with the primary analysis. After excluding studies using nonreference or indirect VAT measurement methods, all five exercise modalities retained statistically significant VAT reductions compared with control, and AE (V) and HIIT remained the top two modalities; only the relative positions of AE (MV) and RT were reversed (Appendix S12).

4 | Discussion

4.1 | Principal Findings

This meta-analysis of randomized controlled trials compared the effects of different exercise interventions on VAT and related indicators in individuals with overweight or obesity. It is the first to employ network dose–response meta-analysis to evaluate the effects of diverse exercise modalities and estimate optimal exercise dose standards. First, both pairwise and network comparisons consistently confirmed that all evaluated exercise modalities were significantly superior to nonexercise control groups in reducing VAT among individuals with overweight or obesity, with AE (V) and HIIT demonstrating the most pronounced effects. Second, the network dose–response meta-analysis revealed a nonlinear U-shaped dose–response relationship between total exercise volume and VAT reduction. Health benefits of exercise began to emerge at lower doses (approximately 99 MET-min/week), increasing with dose magnitude and peaking at approximately 750 MET-min/week. Beyond this dose, the marginal benefit of VAT reduction from additional exercise volume tended to diminish. Third, different exercise modalities exhibited distinct dose–response characteristics. HIIT demonstrated superior efficiency, capable of triggering significant VAT reduction with a lower cumulative dose (130 MET-min/week). Fourth, the study identified the dose required to reach the SCBT for meaningful VAT improvement as approximately 440 MET-min/week. Finally, AE (MV) and HIIT showed unique advantages in improving secondary outcomes such as TBF and BMI, respectively, highlighting the necessity of formulating targeted exercise prescriptions based on specific metabolic goals. These findings provide critical quantitative evidence for developing future exercise guidelines to improve VAT and related indices in individuals with overweight or obesity.

4.2 | Comparison With Other Studies

Our findings align with previous meta-analytic evidence regarding exercise management of VAT and significantly expand upon it. Compared with previous pairwise meta-analyses that confirmed the high efficiency of aerobic exercise [9, 14], the innovation of this study lies in the application of a Bayesian network dose–response model. Building on earlier categorical dose comparisons [23], this approach further describes dose–response trajectories across exercise modalities. Our results not only corroborate the similar estimated efficacy of AE (V) and HIIT in improving VAT as reported by Chen et al. [50] but also suggest the potential time-efficiency advantages of these two modes from a dose–response perspective: At higher exercise intensities, the same total MET-min/week dose can be accumulated within a shorter exercise duration. The clinical relevance of this time efficiency should also be considered in relation to outcome-specific responses. Direct comparisons in patients with T2DM and overweight or obesity suggested greater improvements in cardiorespiratory fitness with HIIT than with moderate-intensity continuous training (MICT) but no clear between-group differences in body composition or HbA1c [51]. For VAT itself, evidence in patients with metabolic dysfunction-associated steatotic liver disease (MASLD) supports reductions with both HIIT and MICT relative to nonexercise controls, whereas limited head-to-head comparisons did not establish a difference in VAT reduction between them [52]. The favorable AE (V) and HIIT estimates in our analysis are also compatible with the "intensity-dependent" hypothesis of visceral fat oxidation. Mechanistically, compared to low-to-moderate intensity exercise, high-intensity exercise induces more intense secretion of catecholamines, growth hormone, and atrial natriuretic peptide, thereby driving more significant lipolysis in visceral adipocytes that possess a high density of β-adrenergic receptors [53]. Beyond these classical neuroendocrine pathways, emerging evidence highlights the role of skeletal muscle as an endocrine organ during high-intensity exertion [54]. Vigorous muscle contractions stimulate the secretion of specific myokines (e.g., irisin and IL-6) that mediate muscle-adipose tissue crosstalk. These signaling molecules have been shown to promote the "browning" of white adipocytes and upregulate uncoupling protein 1 (UCP1) expression in experimental studies [55, 56], thereby amplifying thermogenesis and lipid oxidation in a way that low-intensity activity may not sufficiently trigger [57]. Furthermore, the excess postexercise oxygen consumption (EPOC) effect triggered by high-intensity exercise may explain why shorter duration HIIT protocols can still maintain fat-loss efficacy [21].

The role of RT in reducing VAT has long been controversial [15]. Our overall network ranking indicated that the estimated VAT-reducing effect of RT was less favorable than that of AE (V), which may be attributed to its relatively lower energy expenditure per session. Nevertheless, simple ranking metrics may underestimate the unique clinical value of RT in preserving lean body mass and maintaining resting metabolic rate, which are critical for long-term metabolic health. This perspective is also relevant to CBE: Although it did not yield the largest estimated VAT reduction in our analysis, ranking on a single outcome cannot fully represent its value in comprehensive treatment. Evidence specifically for AE-RT combinations in patients with T2DM and overweight or obesity extends from improvements in HbA1c and quality of life [58] to better walking and sit-to-stand performance [59] relative to nonexercising standard care. Thus, when metabolic control and physical function are parallel goals, the choice of a combined approach may be informed

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by considerations beyond VAT ranking. Furthermore, through network meta-regression analysis, we identified the proportion of female participants as a potential study-level effect modifier, with smaller RT-associated VAT reductions in studies with a higher proportion of females. This finding provides a new perspective for interpreting previous literature on visceral adiposity and muscle-related outcomes [60, 61]. This sex-specific difference may be closely related to the hormonal environment. Males tend to have central fat accumulation, and their VAT possesses a higher density of androgen receptors. The acute elevation of basal testosterone levels induced by RT may interact specifically with these receptors, promoting muscle hypertrophy while more effectively mobilizing visceral fat reserves, thereby facilitating visceral fat oxidation more efficiently [62]. The estrogenic environment in females tends to maintain subcutaneous fat reserves, which may to some extent attenuate the direct mobilization effect of RT on VAT. Therefore, a simple ranking of "superior versus inferior" may obscure the potential value of RT for specific populations, suggesting that clinical prescriptions need to consider sex differences. Beyond sex-related differences, our overall meta-regression also revealed a counterintuitive finding: study populations with higher baseline body fat percentages experienced attenuated VAT reductions. This may indicate "metabolic inflexibility" in severe obesity, where blunted catecholamine sensitivity and impaired adipose tissue blood flow hinder lipid mobilization despite exercise stimuli [1, 63]. Consequently, patients with severe obesity might require a longer induction period or multimodal interventions to overcome this initial metabolic resistance.

The nonlinear dose–response curve observed in this study is another core contribution. The pattern of onset at low doses, peak effect at moderate doses (approximately 750 MET-min/week for VAT), and diminishing returns at higher doses was observed for VAT, with broadly similar trajectories for TBF and BMI, whereas the fitted body-weight curve continued to decline across the evaluated dose range. The nonlinear pattern also broadly aligns with recent findings in various independent health fields, such as cognitive function in the elderly [27], mental and physical health [64], and glycemic control in type 2 diabetes [65]. The convergence of evidence across these fields supports a more universal physiological perspective: for the prevention and management of multiple chronic diseases, there may be outcome-specific "optimal dose windows" that balance physiological adaptation with stress load, thereby maximizing health benefits. This phenomenon likely reflects a multifaceted interplay between physiological adaptations and real-world implementation challenges. Physiologically, mechanisms such as increased postexercise energy intake or reduced nonexercise activity thermogenesis may partially blunt the net caloric deficit at extreme exercise volumes. More notably, on a pragmatic level, this trajectory may reflect the tension between exercise prescription and patient adherence. Although physiological benefits might theoretically continue to accrue, the observed diminishing returns may reflect the challenge of sustaining such high exercise loads, where participant drop-outs or reduced compliance could attenuate the aggregated group-level benefit. Therefore, our model may reflect effectiveness constrained by biology and behavior rather than merely idealized physiological potential. Consequently, the model-estimated optimal total physical activity volume for VAT (approximately 750 MET-min/week) and the effective dose ranges for each exercise modality collectively provide a potential pragmatic dosing benchmark, aimed at balancing therapeutic benefits with feasibility. Furthermore, previous evidence indicates that both exercise and dietary restriction can reduce VAT [66]. Exercise therefore remains an important nonpharmacological component of metabolic health management [67]. A moderate exercise volume (e.g., approximately 750 MET-min/week) was associated with a model-predicted mean VAT reduction that met the distribution-based SCBT, further reinforcing the potential of exercise in managing visceral obesity and its associated metabolic risks. From a pathophysiological perspective, our findings align with evidence that improvements in body composition are not fully captured by changes in body weight [68]. The RT findings in our network—favorable estimates for VAT, TBF, and BMI without a statistically significant body-weight effect—illustrate this distinction. Pooled evidence in patients with T2DM and overweight or obesity shows a related divergence: Glycemic improvements with HIIT were not accompanied by clear pooled changes in body fat or fat-free mass [69], whereas AE improved glycemic control and body fat percentage despite a nonsignificant pooled effect on body weight [70]. This suggests that in clinical practice, improvements in VAT, waist circumference, BMI, and other body composition metrics should be regarded as complementary indicators of cardiometabolic risk alongside changes in body weight [71].

4.3 | Clinical Implications

This study provides several key findings with significant clinical implications (Figure S8). First, the information we provide can be used to guide recommendations on exercise dose and modality to reduce the VAT burden in individuals with overweight or obesity. By identifying the model-estimated dose corresponding to the distribution-based SCBT for VAT reduction (approximately 440 MET-min/week) and the window for optimal benefit, we provide specific quantitative references for clinical decision-making. Considering the physical limitations of individuals with overweight and obesity, this "optimal dose" should be viewed as a long-term reference target rather than an immediate starting point. A strategy of progressive increase, starting from a tolerable lower dose and gradually advancing toward the target level, is recommended to minimize the risk of injury and enhance adherence. Second, accumulating 150–250 min of AE (MV) per week may serve as a reference for exercise scheduling; for patients seeking time efficiency and able to tolerate higher intensities, approximately 75–120 min of HIIT or AE (V) per week may be considered. Actual durations should be adjusted according to exercise intensity to align the weekly dose with the beneficial dose range for the corresponding modality. This stratified strategy aims to balance efficacy with adherence. Third, regression analysis results suggest that sex and baseline body fat status should be considered when developing individualized exercise prescriptions. For patients with insufficient VAT improvement who also need to maintain muscle function, combining RT with aerobic exercise may be considered, with adjustments based on individual responses. Fourth, we also translated the dose–response results into practical, specific exercise plan examples (Figure S6), which clinicians can use to formulate personalized exercise prescriptions

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that align with patient preferences and capabilities. Finally, given the association between VAT and cardiovascular disease, clinical management should recognize VAT improvement as an important therapeutic target alongside weight loss, promoting a shift in obesity treatment toward a precision medicine model oriented by cardiometabolic risk.

4.4 | Potential Limitations

This study has several limitations. First, although we confirmed the robustness of the findings by excluding trials involving concurrent energy restriction, subtle differences in background lifestyle recommendations across the remaining studies may still introduce a small degree of residual confounding. Second, as most trials ranged from 8 to 24 weeks in duration, the present study primarily reflects medium- to short-term metabolic adaptations to exercise, with long-term maintenance effects awaiting clarification from longitudinal studies of longer duration. Third, exercise doses calculated using standardized MET values may not fully capture interindividual heterogeneity in energy expenditure efficiency, and potential fluctuations in some "maintain lifestyle" control groups could interfere with the isolation of pure exercise effects. VAT assessment methods also varied, and indirect estimates are not interchangeable with CT or MRI measurements, although excluding studies using nonreference or indirect methods did not change the direction of VAT effects versus control. In addition, meta-regression findings represent study-level associations and require individual-level validation. Finally, the SCBT identified in this study was estimated based on statistical distribution characteristics and lacks direct anchoring to hard clinical outcomes. Consequently, this threshold should be interpreted as a distribution-based reference for benefit, with its relevance to long-term clinical prognosis requiring further validation.

5 | Conclusion

This systematic review and dose–response network meta-analysis provides comprehensive quantitative evidence for the efficacy of exercise in reducing VAT in individuals with overweight or obesity. The results demonstrate that the therapeutic effects are modality- and dose-dependent. The model-estimated overall exercise benefit peaked at a dosage of approximately 750 MET-min/week. AE (V) and HIIT showed the largest estimated VAT reductions compared with the control group. These findings provide a scientific basis for formulating personalized, precise exercise prescriptions in clinical practice, facilitating the more effective application of exercise into the management of obesity and related metabolic diseases. Future research should focus on the evaluation of long-term intervention effects and further explore the differential responses of different population subgroups to specific exercise protocols.

Author Contributions

X.W. and Y.D. had full access to all the data in this systematic review and take responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: X.W., Y.D., and D.L. Acquisition, analysis, or interpretation of data: all authors. Drafting of the manuscript: X.W. and Y.D. Critical revision of the manuscript for important intellectual content: all authors. Statistical analysis: Y.D., D.L., and Z.Z. Administrative, technical, or material support: X.W. and Y.D. Study supervision: X.W. All authors read and approved the final manuscript.

Acknowledgments

The work was supported by the National Key Research and Development Program of China (Grant Number 2023YFC3606104).

Funding

This work was supported by the National Key Research and Development Program of China (2023YFC3606104).

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Table S1:** The classifications of exercise training. **Table S2:** Network meta-analysis matrix of VAT and secondary outcomes. **Figure S1:** Literature review flowchart. RCT, randomized controlled trial. **Figure S2:** Pairwise comparison of all exercise interventions on VAT effects. I^2: quantitative indicators of heterogeneity; *p* value: statistically significant *p* values for pooled results; power: statistical power for pooled effect size. 95% CI = 95% confidence interval. AE (MV), moderate to vigorous intensity aerobic exercise; AE (V), vigorous intensity aerobic exercise; CBE, combined exercise; CON, control; HIIT, high-intensity interval training; RT, resistance exercise. **Figure S3:** Network plot and cumulative ranking probability profiles. The network plot illustrates direct comparisons among exercise interventions for visceral adipose tissue reduction. Each node represents an intervention type; node size reflects the number of participants. Lines represent direct comparisons between interventions; line thickness indicates the number of studies. Cumulative ranking probability curves show the probability of each intervention being among the top *r* interventions for VAT reduction, where *r* denotes the rank on the horizontal axis. For a given *r*, higher curves indicate a greater cumulative probability of being among the top *r* interventions. AE (MV), moderate to vigorous intensity aerobic exercise; AE (V), vigorous intensity aerobic exercise; CBE, combined exercise; CON, control; HIIT, high-intensity interval training; RT, resistance exercise. **Figure S4:** Dose–response relationship between total exercise (weekly) and VAT level in individuals with overweight or obesity. Shaded area represents 95% credible intervals. Vertical lines indicate exercise dose categories; horizontal line represents the substantial clinical benefit threshold. **Figure S5:** Dose–response relationship between different exercises (weekly) and VAT levels in individuals with overweight or obesity. Dose–response curves showing the relationship between weekly exercise volume and VAT reduction (Hedges' *g*) for five exercise types. Solid curves represent fitted relationships; shaded areas indicate 95% credible intervals. Vertical dashed lines show recommended exercise volumes. Negative values indicate greater VAT reduction. AE (MV), moderate to vigorous intensity aerobic exercise; AE (V), vigorous intensity aerobic exercise; CBE, combined exercise; CON, control; HIIT: high-intensity interval training; RT, resistance exercise. **Figure S6:** Clinical cheat sheet. The recommended duration (min/week) was calculated by dividing the specific MET-min/week thresholds by the average MET value assigned to each exercise modality. "Sessions × minute" provides practical examples to achieve these weekly targets. Opt, optimal. **Figure S7:** Meta-regression of moderators of exercise intervention effects on VAT. Bubble plots showing the associations between four potential moderators and standardized mean differences (Hedges' *g*) in visceral adipose tissue. (A) Intervention period (weeks); (B) exercise frequency (sessions/week); (C) baseline body fat percentage; and (D) percent female. Bubble size reflects study weight. Solid lines indicate meta-regression fits with 95% confidence intervals (dotted lines). **Figure S8:** Practical implications. **Appendix S1:** Supporting Information.

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