Optimising exercise prescription for cancer-related fatigue, lean mass, physical function and muscle strength in women with breast cancer: a systematic review with dose–response network meta-analysis

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ABSTRACT

Objective To systematically review and investigate the dose–response (DR) relationship of aerobic exercise (AE), resistance exercise (RE) and combined (COMB) exercise programmes on cancer-related fatigue, lean mass, physical function and muscle strength in women with breast cancer during and following primary treatment.
Design Systematic review with DR network meta-analysis (DR-NMA; CRD42023491118).
Data sources CINAHL, Embase, LILACS, PubMed, SciELO, SPORTDiscus and Web of Science were searched from inception to 10 January 2024, with an updated search in April 2025.
Eligibility criteria for selecting studies Randomised controlled trials evaluating COMB, RE or AE in women with breast cancer on the four specified outcomes.
Results 64 trials (n=5156) were included. COMB was the most effective modality for improving fatigue and lean mass, and RE was the most effective for improving physical function and muscle strength. The smallest effective doses of COMB associated with significant changes in fatigue (standardised mean differences (SMD) 0.23 (95% CI 0.18 to 0.28)) and lean mass (SMD 0.30 (95% CI 0.22 to 0.38)) were 18 and 11 metabolic equivalents per week (METs.min/week), respectively. For RE, about 7–8 METs.min/week produced significant improvements in physical function (SMD 0.23 (95% CI 0.15 to 0.30)) and strength (SMD 0.24 (95% CI 0.20 to 0.29)). Fatigue benefits (SMD 0.58 (95% CI 0.46 to 0.69)) plateaued at ~1428 METs.min/week for COMB, whereas no plateau was observed for lean mass or strength across the evaluated dose range, with effects increasing throughout the available dose range.
Conclusions COMB and RE were the most effective interventions across outcomes in women with breast cancer. Benefits for the outcomes assessed in this systematic review with DR-NMA were achieved with exercise doses as low as 10–40 min/week.
PROSPERO registration number CRD42023491118.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Exercise as a benefit for people living with and beyond cancer has gained substantial recognition from multiple international organisations in recent years, with evidence demonstrating that structured exercise programmes can improve survival and reduce cancer recurrence.
⇒ Current guidelines recommend that patients with cancer engage in regular exercise, ideally incorporating two to three times a week of moderate-intensity aerobic exercise and two weekly sessions of resistance exercise, alone or in combination.

WHAT THIS STUDY ADDS

⇒ We identified a non-linear dose–response relationship between exercise and outcomes in women with breast cancer, showing that improvements in fatigue, lean mass, physical function and muscle strength can occur with relatively low weekly exercise doses (~10–40 min/week).
⇒ A plateau effect was observed for fatigue, whereas improvements in lean mass and muscle strength continued to increase across the evaluated dose range, with no clear plateau identified.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ We provide evidence-based dose thresholds for exercise prescription in women with breast cancer, showing that clinically meaningful benefits can occur at very low weekly exercise doses.
⇒ While fatigue improvements appeared to plateau at higher exercise volumes, no plateau was observed for lean mass or muscle strength within the evaluated dose range.
⇒ Our findings support more efficient, personalised exercise programming in clinical practice for women with breast cancer; future research is needed to determine dose optimisation.

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INTRODUCTION

Breast cancer is the most common and deadliest cancer among women worldwide.[1] Despite 5 year survival rates exceeding 90% for patients with localised and regional disease, women living with and beyond breast cancer often experience persistent

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and burdensome treatment-related side effects, including fatigue,[2–4] reductions in lean or muscle mass[5] and impairments in physical function and muscle strength.[6 7] These contribute to a greater symptom burden, diminished quality of life and an increased risk of mortality and cancer recurrence.[2–7]

Exercise for people living with and beyond cancer has gained substantial recognition from multiple international organisations in recent years[8–13] and has been further supported by evidence demonstrating that structured exercise programmes can improve survival and reduce cancer recurrence.[14–16] Current guidelines recommend that patients with cancer engage in regular exercise, ideally incorporating two to three times a week of moderate-intensity aerobic exercise (AE) and two weekly sessions of resistance exercise (RE), alone or combined (COMB) within a periodised exercise programme.[8–13] We[17–20] and others[21–23] previously investigated the effects of different exercise programmes in women with breast cancer, demonstrating beneficial effects of RE and COMB on outcomes such as cancer-related fatigue, lean mass, muscle strength and physical function. However, these analyses did not account for exercise dose, and whether a potential therapeutic range of exercise exists, encompassing minimum and optimal doses as well as a plateau effect, remains unclear. This gap is particularly evident in breast cancer.[8–13]

The frequentist dose–response network meta-analysis (DR-NMA), recently developed by Petropoulou *et al*,[24] represents a novel extension of standard NMA that models the functional relationship between doses and responses.[24 25] This approach allows the estimation of dose-dependent effects and the prediction of treatment responses across a continuous range of doses.[26] However, to our knowledge, no previous study has applied DR-NMA to examine the DR relationships between different exercise modalities, prescribed exercise doses and outcomes in women with breast cancer. Therefore, this systematic review with DR-NMA aims to investigate the DR relationship of AE, RE and COMB exercise programmes on cancer-related fatigue, lean mass, physical function and muscle strength in women with breast cancer. Specifically, we aim to identify the minimum effective dose, potential plateau points and the most effective exercise modalities for these outcomes, providing evidence to inform more precise and targeted exercise prescriptions for women living with and beyond breast cancer.

METHODS

Search strategy and study selection procedure

All procedures were reported in line with the Cochrane Back Review Group,[27] which provides rigorous methodological standards for systematic reviews of non-pharmacological interventions such as exercise, as well as the Implementing PRISMA in Exercise, Rehabilitation, Sport medicine and SporTs science (PERSiST)[28] and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses for Network Meta-Analyses (PRISMA-NMA)[29] statement.

The present study is a follow-up to our previous systematic review and NMA (PROSPERO identifier: CRD42023491118) investigating the potential interference effect of prescribing COMB in women diagnosed with breast cancer.[20] For the present analysis, we have included randomised trials evaluating the effects of COMB, RE or AE on breast cancer-related fatigue, lean mass, physical function and muscle strength. These outcomes are commonly assessed in *exercise oncology* trials and are strongly associated with clinical endpoints, including symptom burden and mortality.[2–7] Additionally, studies had to provide information on exercise prescription characteristics (modality, duration, frequency, volume (number of exercises and number of sets for RE; duration for AE) and intensity (percentage of one repetition maximum (1-RM) and maximum repetitions (RMs)) for RE; percentage of maximal heart rate and percentage of heart rate reserve for AE) in women diagnosed with breast cancer at any stage. The exclusion criteria were (1) women with breast cancer randomised to exercise interventions lasting <4 weeks; (2) women with breast cancer randomised to exercise interventions combined with any nutritional approach (eg, healthy diet, caloric restriction and protein supplementation) and (3) studies written in a language other than English, Portuguese or Spanish.

A systematic search was conducted by a researcher (PL) using *CINAHL*, *Embase*, *LILACS*, *PubMed*, *SciELO*, *SPORTDiscus* and *Web of Science* databases from inception to 10 January 2024 (CRD42023491118). A manual search and an updated search in PubMed (5 April 2025) were also undertaken. Eligibility was assessed independently in triplicate (PL, CBS and TM). The search strategy was structured according to the *Population, Intervention, Comparison, Outcome* (PICO) framework and combined terms related to breast cancer, exercise interventions (AE, RE and COMB) and randomised controlled trials. Information on the outcomes and exercise prescription parameters (eg, frequency, intensity, duration and volume) was considered during the screening and data extraction stages. The search strategy undertaken in our previous study is presented in online supplemental appendix S1.

Data extraction

Data were extracted using a standardised form. Relevant information included demographic and clinical characteristics (age, body mass index (BMI), treatment status, advanced disease, metastasis, breast surgery, radiotherapy, chemotherapy and hormone therapy). Exercise prescription characteristics, such as modality (AE, RE and/or COMB), delivery method (supervised, non-supervised and hybrid), duration, frequency, dose (or volume) and intensity were also extracted.

The prescribed exercise dose was expressed in metabolic equivalents per week (METs.min/week), calculated as the product of session duration (min), frequency (sessions per week) and the assigned intensity in METs for each exercise modality. The assigned MET values were assigned according to the *Compendium of Physical Activities*[30 31] and were conservative, given the deconditioning and toxicities experienced by women with breast cancer during or following primary treatment.[32] For RE, the session duration was calculated as the time required to complete the prescribed number of exercises, sets per exercise and rest intervals between sets, with assigned values of 3 METs for moderate-intensity (50%–69% of 1-RM) and 6 METs for high-intensity RE (≥70% of 1-RM).[30 31] For AE, session duration was defined as the prescribed time of continuous AE or high-intensity interval training per session, with assigned values of 4.5 METs for moderate-intensity and 6 METs for high-intensity AE.[30 31] The total METs for COMB was defined as the sum of AE and RE components.

The outcomes were cancer-related fatigue or vitality, lean mass or muscle mass, physical function and lower-limb muscle strength. We extracted mean, SD, SE and/or 95% CI from the outcomes at baseline and post-assessment time points as well as within-group and between-group differences. When the SD of the change was not reported, it was calculated assuming a correlation of r=0.5 between the baseline and post-intervention assessment measures.[33]

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Risk of bias assessment and certainty of evidence (Grading of Recommendations Assessment, Development and Evaluation)

The risk of bias at the outcome level was evaluated according to the Cochrane risk-of-bias tool 2.0,[34] performed independently by two researchers (CTM and PC), with disagreements resolved by a third researcher (PL). Risk-of-bias assessments were conducted for each primary outcome within each study. When multiple post-intervention time points were reported, the assessment was based on the time point closest to the end of the intervention. The Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach for NMA[35] was undertaken to assess the certainty of evidence.

Data synthesis and analysis

All analyses were conducted using frequentist approaches in R (R Development Core Team, Vienna, Austria). Continuous outcome data in analyses were summarised as standardised mean differences (SMD) with 95% CI due to heterogeneity in outcome measures across studies and substantial variability in absolute values driven by demographic and clinical differences. SMD values were estimated using Hedges' g,[36] which applies a correction factor to Cohen's d[37] to reduce bias in studies with small sample sizes. Following Hedge's g, SMD values of 0 to ≤0.5 were interpreted as small, 0.51 to 0.79 as medium or moderate and ≥0.8 as large effects; however, these thresholds were not applied rigidly, and interpretation considered the clinical context and the precision of the estimates (95% CI). Where available, intention-to-treat (ITT) data were preferentially extracted. When ITT data were not reported, per-protocol or complete-case data were used.

Several frequentist models were applied, including the standard NMA[38] as well as a series of DR-NMA models to account for different functional forms of the DR relationship. Given the anticipated clinical and methodological between-study variability (ie, heterogeneity), we applied a random-effects model to estimate the comparative effects of AE, RE, COMB and control interventions. The heterogeneity was estimated using the generalised DerSimonian and Laird estimator and the Cochran's heterogeneity statistic (Q)-profile approach and assessed using inconsistency statistic (I^2) and the magnitude of the between-study variance (τ^2) within each intervention comparison.

The frequentist NMA model was performed following the current PRISMA guideline for NMA,[38 39] using the graph-theoretical approach implemented in the R package netmeta.[39] Global inconsistency within each network was evaluated using the random-effects design-by-treatment interaction model and the loop-specific approach.[40] For each NMA, we assessed a priori the transitivity assumption, which implies that the distribution of the potential treatment effect modifiers is balanced across the available direct comparisons.[41] We used treatment status, age, BMI, stage III–IV and metastasis as potential intervention effect modifiers (online supplemental tables S1–S4). In the presence of inconsistency, direct and indirect evidence were separated to assess local inconsistency.[42] Extreme-study effects (ie, outliers) were explored using the forward search (FS) algorithm[43] implemented in the R package 'NMAoutlier'.[44] Obvious outliers were excluded in sensitivity analyses to assess the robustness of results. The publication bias effect was also examined using comparison-adjusted funnel plots.[45] Intervention effects within the NMA were ranked according to P-scores,[46] which represent the frequentist analogue of the Bayesian surface under the cumulative ranking curve (SUCRA)[47] and reflect the relative ranking of interventions based on effect estimates and their uncertainty.

The frequentist DR-NMA model[24] was conducted using the R package netdose[25 48] to evaluate the DR effects on the outcomes of interest, separately for each exercise modality. To characterise the exercise DR relationship, we evaluated several functional forms, including linear, exponential, quadratic polynomial and first-order fractional polynomial (FP1; logarithmic transformation) transformations with power p from the set (−2, −1, −0.5, 0, 0.5, 1, 2, 3).[49 50] FP1 models were considered to balance flexibility and parsimony, following methodological recommendations,[24] while avoiding overfitting associated with higher-order polynomials, particularly in settings with a limited number of dose levels and studies. Restricted cubic splines[51] with three knots were used, following the same methodological framework.[24] Knots were placed at the 10%, 50% and 90% percentiles of the dose distribution, with an alternative specification at the 25%, 50% and 75% percentiles of the dose distribution.

Model adequacy relative to model complexity was evaluated for all models using the Q/degrees of freedom (df) ratio, which was used as an indicator of model adequacy relative to model complexity, thereby reflecting the balance between model fit and parsimony. Model selection followed a structured approach and was based on a combination of model adequacy (Q/df) and heterogeneity (τ^2), complemented by graphical inspection of the fitted DR plots, with preference given to models that substantially reduced heterogeneity while maintaining adequate model fit. When multiple models showed similar adequacy, preference was given to simpler and more interpretable models to ensure robust estimation of the minimum effective dose and plateau. Forest plots and DR plots were generated for each outcome, using threshold settings to define the benchmark response and the plateau point. The Benchmark Dose Lower Confidence Limit (BMDL) was calculated as a conservative approximation of the minimum effective dose, based on a benchmark response of 0.1 (corresponding to a 10% improvement). Conservative default thresholds were used for the BMDL (0.1) and for the plateau (0.001). To assess robustness, sensitivity analyses were performed with alternative thresholds (benchmark 0.05 and 0.2; plateau 0.001–0.003). Exploratory subgroup DR-NMA were conducted to examine whether treatment status (current treatment vs previous treatment), age (middle-aged vs older), BMI (overweight vs obese), stage III–IV (below or equal 50% vs above 50%) and metastasis (below or equal 50% vs above 50%) modified the median dose observed across outcomes.

Equity, diversity and inclusion statement

Our research team comprises researchers from multiple countries, institutions and disciplinary backgrounds, reflecting substantial geographic and academic diversity. The team includes women and men at different career stages, from early-career to senior academics, contributing to a collaborative and inclusive scientific environment.

RESULTS

A total of 3402 records were identified, with 2146 screened after duplicate removal. Ultimately, 126 articles[52–177] describing 112 studies were included in the primary analysis, of which 70 articles[52 56–59 61 64–66 68–71 73–76 78 82 84 87 89 90 97–99 103–105 107 109–115 117 118 124 126 128–133 139 140 142 146 149 151–154 156 158 166–169 171–173 176] describing 61 randomised trials (plus three from an updated search)[178–180] were included in the DR analysis (online supplemental figure S1).

Study, participant and intervention characteristics

A total of 5156 women with breast cancer (median age 52.7 (IQR 49.5 to 55.1) years; median BMI 27.8 (IQR 25.9 to

29.4) kg/m^2) were included. Most studies involved middle-aged women (90.6%), the majority of whom were overweight (57.8%) and had early-stage disease (71.9%) at the study level. In studies conducted during primary treatment (39.1%), most patients underwent breast surgery (median 100% (IQR 63.3% to 100%)), radiotherapy (median 73.2% (IQR 48.3% to 100%)) and chemotherapy (median 100% (IQR 87.3% to 100%)). For studies conducted following primary treatment (59.4%), most patients had received breast surgery (median 100% (IQR 87.2% to 100%)), radiotherapy (median 71.0% (IQR 55.0% to 84.0%)) and chemotherapy (median 73.3% (IQR 44.9% to 90.7%)) and were receiving hormonal therapy (median 68.3% (53.3% to 83.4%)).

78 exercise programmes were analysed: AE (46.2%), RE (28.2%) and COMB (25.6%). Exercise programmes were mostly supervised (75.6%) and had a median duration of 12 weeks (IQR 10 to 16). The estimated prescribed exercise doses ranged from 108 to 945 METs.min/week for AE, 48 to 693 METs.min/week for RE and 243 to 1785 METs.min/week for COMB. Most control groups were usual care, non-intervention or waitlist/delayed control groups (80.6%), while the remaining had some type of minimal exposure, such as stretching, relaxation, education/brochure, physiotherapy and physical activity counselling. The characteristics of the studies are presented in online supplemental table S5. The risk of bias assessment for each outcome is presented in online supplemental tables S6–S9.

Analysis of the outcomes

For each outcome, the NMA and DR-NMA models were evaluated using model adequacy (Q/df) and heterogeneity measures. Among the DR-NMA models, FP1 (p=0) provided the best balance between model adequacy (Q/df) and heterogeneity for lean mass, physical function and muscle strength. For cancer-related fatigue, the best balance between model adequacy (Q/df) and heterogeneity was achieved with FP1 (p=0.5), followed by FP1 (p=0). To ensure comparability across models and maintain consistency, FP1 (p=0) was selected for all final analyses (online supplemental tables S10–S13).

Standard NMA model

For each NMA, the design-by-treatment interaction model showed no evidence of inconsistency for cancer-related fatigue (p=0.795), lean mass (p=0.960), physical function (p=0.287) and muscle strength (p=0.470; online supplemental tables S1–S4).

Cancer-related fatigue

COMB was ranked as the most effective intervention to improve cancer-related fatigue compared with controls, with a moderate effect (SMD 0.75 (95% CI 0.49 to 1.00)) and a P-score of 97.2% (figure 1 and online supplemental table S14). Also, AE (SMD 0.44 (95% CI 0.19 to 0.69)) and RE (SMD 0.41 (95% CI 0.11 to 0.71)) led to improvements in cancer-related fatigue compared with controls.

The heterogeneity τ^2 was 0.198 (I^2 83.6%). Visual assessment of comparison-adjusted funnel plots suggested evidence of publication bias (p=0.041; online supplemental figure S2). The FS algorithm identified one potential outlier.[57] After excluding this outlier (online supplemental table S15, figure S3), the sensitivity analysis produced results consistent with the primary analyses.

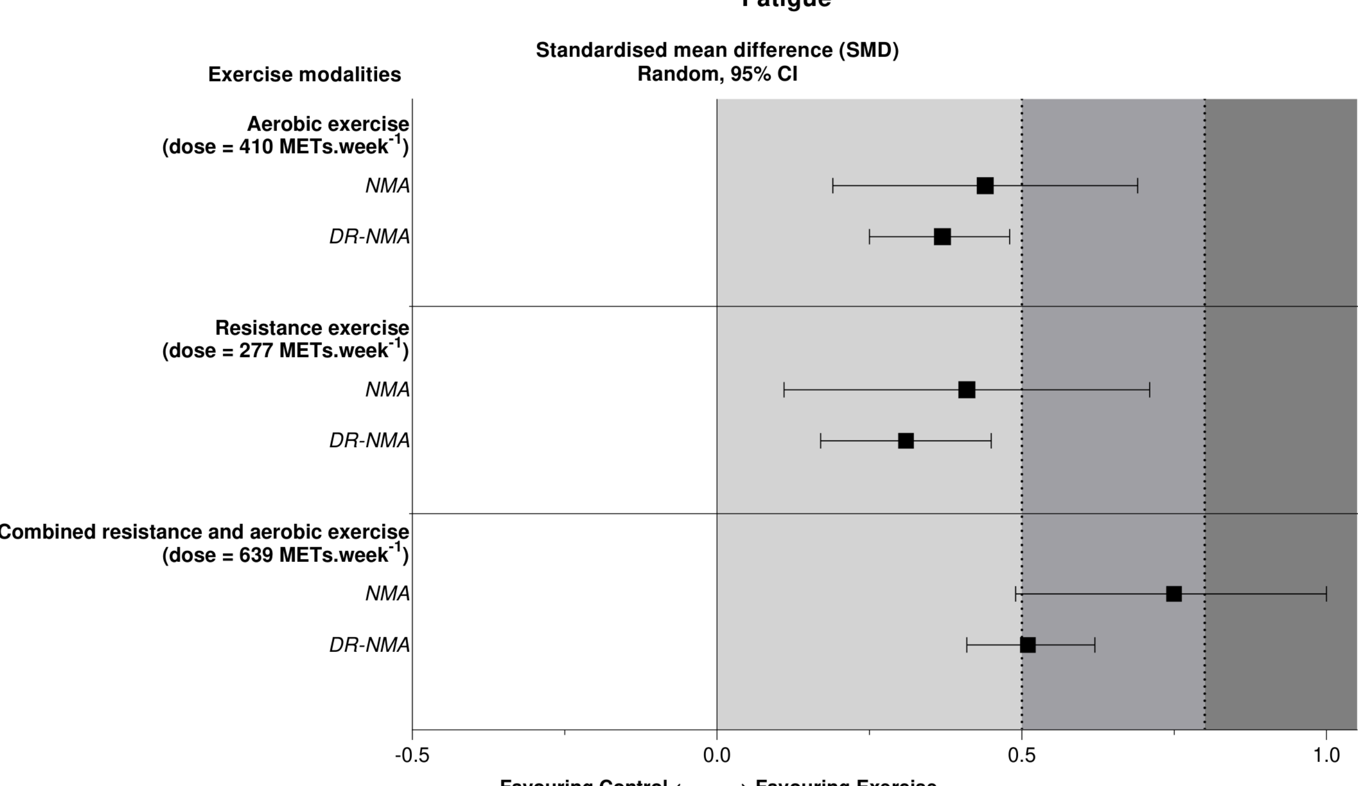

Figure 1 Forest plot demonstrating the NMA and DR-NMA estimates of standardised mean difference and 95% CI for cancer-related fatigue. Predictions were based on the common dose of each exercise modality, defined as the median dose observed across the included studies. DR-NMA, dose–response network meta-analysis; METs.min/week, metabolic equivalents per week; NMA, network meta-analysis.

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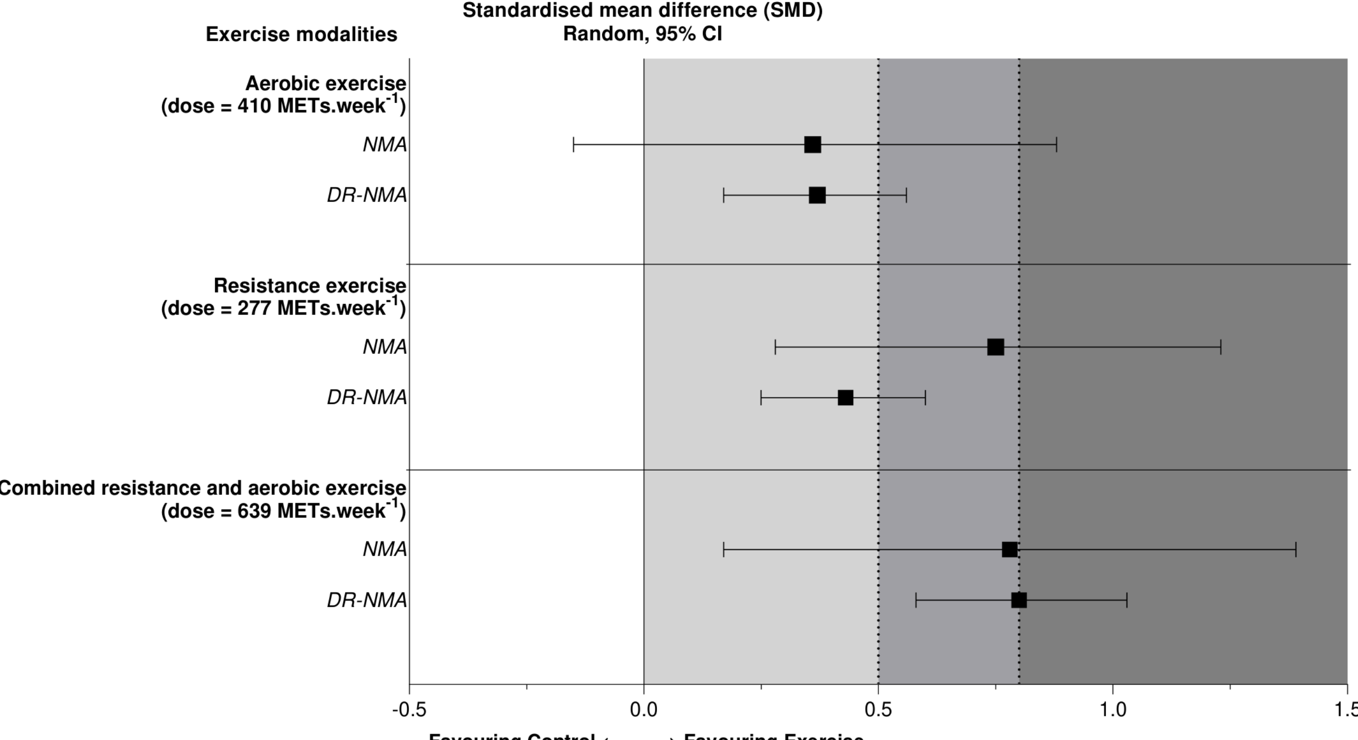

Figure 2 Forest plot demonstrating the NMA and DR-NMA estimates of standardised mean difference and 95% CI for lean mass. Predictions were based on the common dose of each exercise modality, defined as the median dose observed across the included studies. DR-NMA, dose–response network meta-analysis; METs.min/week, metabolic equivalents per week; NMA, network meta-analysis.

Lean mass

Both COMB (SMD 0.78 (95% CI 0.17 to 1.39); moderate effect; P-score 79.7%) and RE (SMD 0.75 (95% CI 0.28 to 1.23); moderate effect; P-score 78.5%) were ranked as the most effective interventions to increase lean mass compared with controls (figure 2 and online supplemental table S16).

The heterogeneity τ^2 was 0.470 (I^2 87.8%), with no evidence of publication bias (p=0.433; online supplemental figure S4). Sensitivity analysis omitting the outlier[153] provided RE (SMD 0.73 (95% CI 0.29 to 1.18)) as the most effective intervention (P-score 85.7%; online supplemental table S17, figure S5).

Physical function

RE was ranked as the most effective intervention to improve physical function compared with controls, with a large SMD of 0.89 (95% CI 0.38 to 1.41) and a P-score of 84.2%. Both COMB (SMD 0.69 (95% CI 0.14 to 1.24)) and AE (SMD 0.59 (95% CI 0.13 to 1.06)) also led to improvements in physical function compared with controls (figure 3A and online supplemental table S18).

The heterogeneity τ^2 was 0.360 (I^2 86.0%). Visual assessment of comparison-adjusted funnel plots suggested evidence of publication bias (p=0.003; online supplemental figure S6). Sensitivity analysis omitting the outlier[151] provided COMB (SMD 0.64 (95% CI 0.21 to 1.07)) as the most effective intervention (P-score 77.1%; online supplemental table S19, figure S7).

Muscle strength

RE was ranked as the most effective intervention to improve lower-body muscle strength compared with controls (SMD 1.05 (95% CI 0.71 to 1.40); large effect) and AE (SMD 0.60 (95% CI 0.10 to 1.10); moderate effect), with a P-score of 92.0% (figure 3B and online supplemental table S18). COMB also led to improvements in muscle strength compared with controls (SMD 0.84 (95% CI 0.35 to 1.32)) but AE did not (SMD 0.45 (95% CI −0.03 to 0.93)).

The heterogeneity τ^2 was 0.379 (I^2 88.6%). Visual assessment of comparison-adjusted funnel plots suggested evidence of publication bias (p=0.003; online supplemental figure S8). Sensitivity analysis omitting the outlier[129] provided a statistically significant difference between RE and COMB (SMD 0.50 (95% CI 0.00 to 0.99); online supplemental table S20, figure S9).

DR-NMA model

Cancer-related fatigue

A forest plot with the DR-NMA results is presented in figure 1. All effect estimates were based on predictions at the common dose of each intervention, defined as the median dose observed across studies. The results indicated that COMB at its common dose of 639 METs.min/week (SMD 0.51 (95% CI 0.41 to 0.62)) was the most effective, followed by AE at 410 METs.min/week (SMD 0.37 (95% CI 0.25 to 0.48)) and RE at 274 METs.min/week (SMD 0.31 (95% CI 0.17 to 0.45)). Subgroup DR-NMA did not indicate differences based on treatment status (p=0.305), age (p=0.291), BMI (p=0.212) and stage III–IV (p=0.277) and metastasis (p=0.149; online supplemental table S21).

The DR curves indicated that the smallest effective dose associated with significant changes in cancer-related fatigue was estimated at ~18 METs.min/week for COMB (SMD 0.23 (95% CI 0.18 to 0.28)), ~15 METs.min/week for AE (SMD 0.17 (95% CI 0.11 to 0.22)) and ~27 METs.min/week for RE (SMD 0.18 (95% CI 0.10 to 0.26)).

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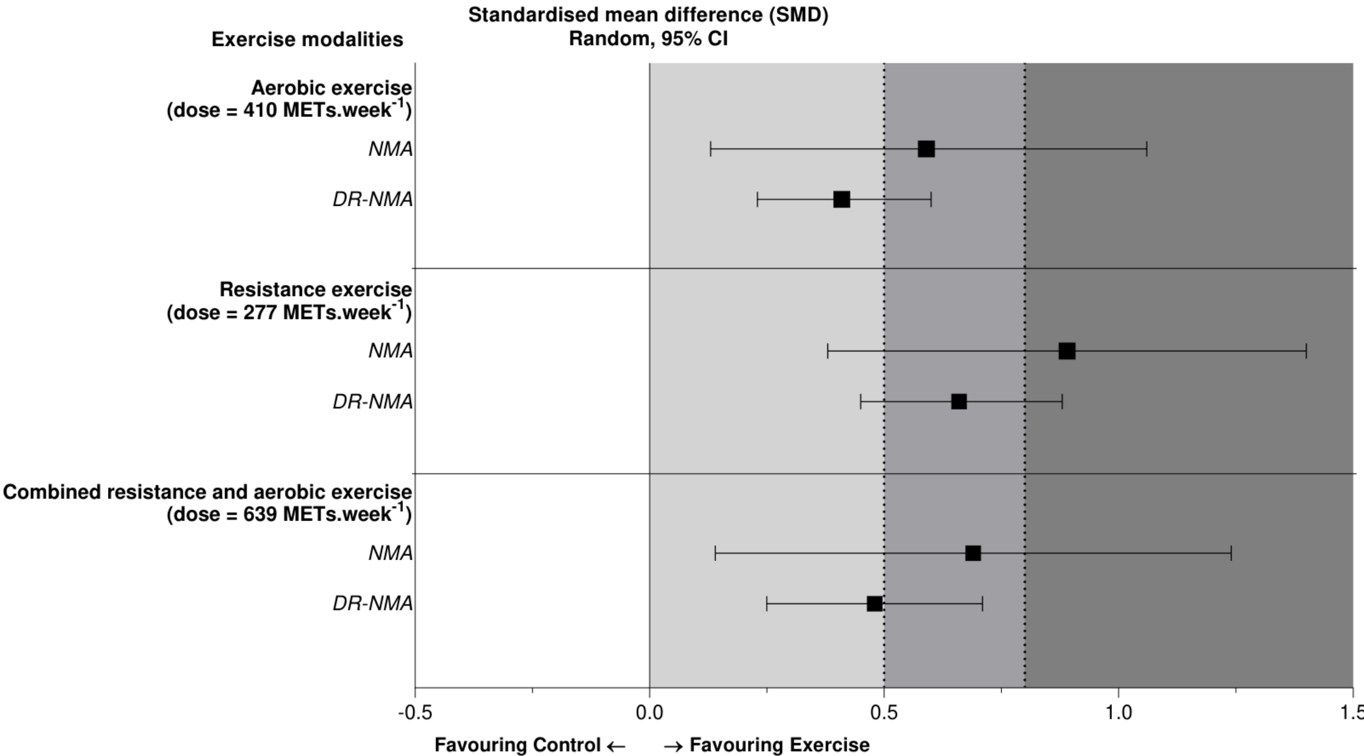

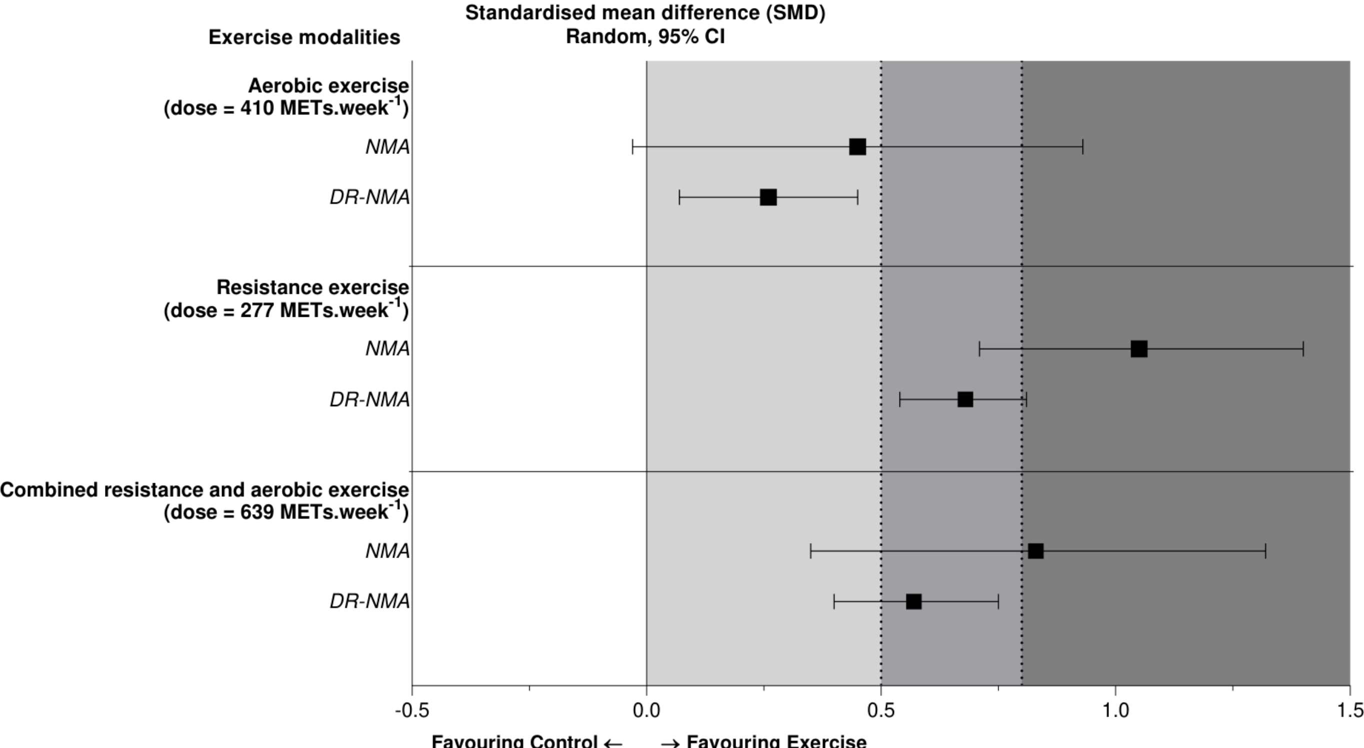

Figure 3 Forest plot demonstrating the NMA and DR-NMA estimates of standardised mean difference and 95% CI for (A) physical function and (B) muscle strength. Predictions were based on the common dose of each exercise modality, defined as the median dose observed across the included studies. DR-NMA, dose–response network meta-analysis; METs.min/week, metabolic equivalents per week; NMA, network meta-analysis.

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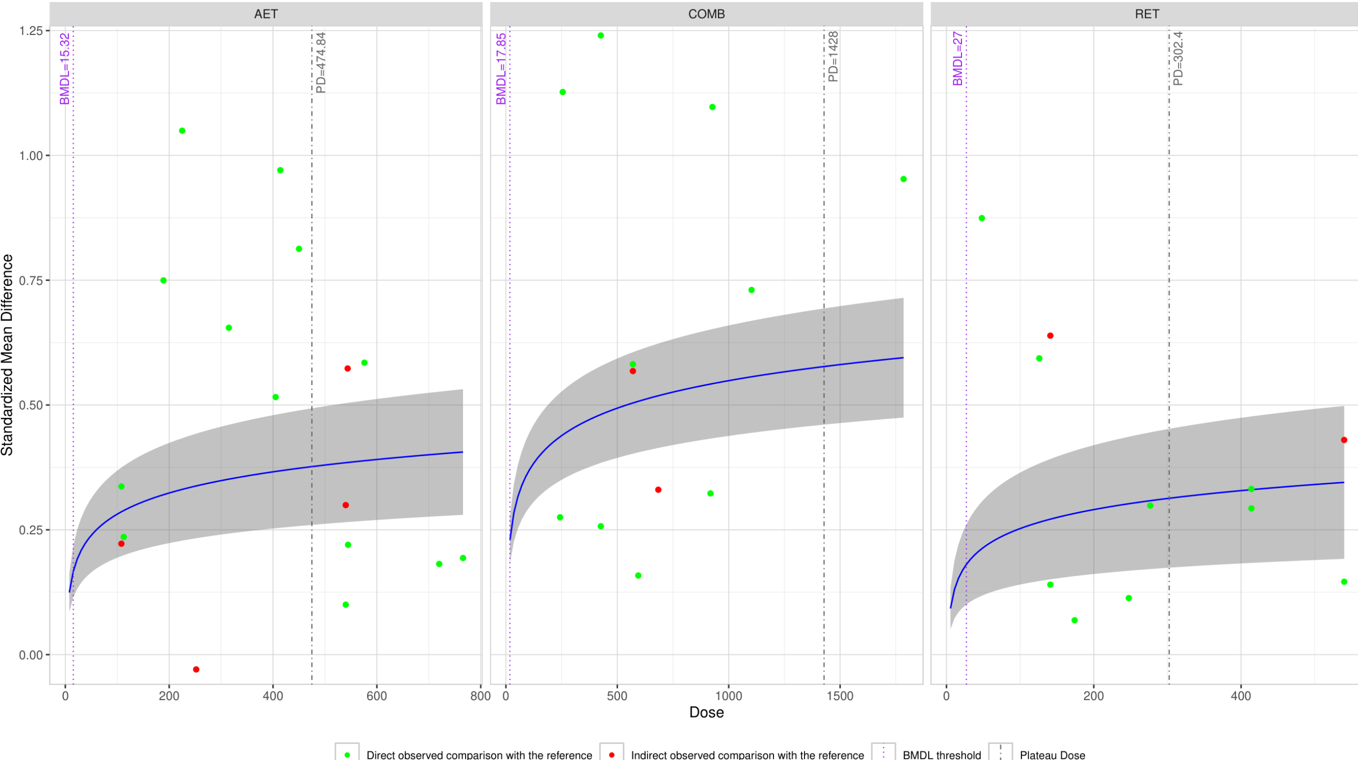

Figure 4 DR-NMA plots for the effects of aerobic, resistance and combined exercise modalities on cancer-related fatigue. AE, aerobic exercise; BMDL, benchmark dose lower confidence limit; COMB, combined exercise; DR-NMA, dose–response network meta-analysis; PD, plateau dose; RE, resistance exercise.

The benefits became more pronounced with increasing dose until reaching a plateau at ~1428 METs.min/week for COMB (SMD 0.58 (95% CI 0.46 to 0.69)), ~475 METs.min/week for AE (SMD 0.38 (95% CI 0.26 to 0.49)) and ~302 METs.min/week for RE (SMD 0.31 (95% CI 0.17 to 0.45)), where additional exercise provided little to no further improvement (figure 4). Sensitivity analyses for the smallest effective dose and the plateau point in the DR-NMA model under different threshold settings are presented in online supplemental table S22.

Lean mass

The results indicated that COMB at 639 METs.min/week (SMD 0.80 (95% CI 0.58 to 1.03)) was the most effective intervention for improving lean mass, followed by RE at 274 METs.min/week (SMD 0.43 (95% CI 0.25 to 0.60)) and AE at 410 METs.min/week (SMD 0.37 (95% CI 0.17 to 0.56)) (figure 2). Subgroup NMA did not indicate differences based on treatment status (p=0.228), age (p=0.807) and BMI (p=0.457; online supplemental table S23). Analyses based on stage III–IV and metastasis were not conducted due to an insufficient number of studies.

The smallest effective dose associated with significant changes in lean mass was estimated at ~11 METs.min/week for COMB (SMD 0.30 (95% CI 0.22 to 0.38)), ~15 METs.min/week for RE (SMD 0.21 (95% CI 0.12 to 0.29)) and ~34 METs.min/week for AE (SMD 0.22 (95% CI 0.10 to 0.33)). The plateau was estimated at ~589 METs.min/week for RE (SMD 0.48 (95% CI 0.28 to 0.68)) and ~424 METs.min/week for AE (SMD 0.37 (95% CI 0.18 to 0.56)).

No plateau was observed for COMB across the evaluated dose range, as the benefits continued to increase progressively throughout the available dose range (figure 5). Sensitivity analyses for the smallest effective dose and the plateau point in the DR-NMA model under different threshold settings are presented in online supplemental table S24.

Physical function

The results indicated that RE at ~277 METs.min/week (SMD 0.66 (95% CI 0.45 to 0.88)) was the most effective intervention for improving physical function, followed by COMB at ~639 METs.min/week (SMD 0.48 (95% CI 0.25 to 0.71)) and AE at ~410 METs.min/week (SMD 0.41 (95% CI 0.23 to 0.60)) (figure 3A). Subgroup DR-NMA did not indicate differences based on treatment status (p=0.314), age (p=0.722), BMI (p=0.636), stage III–IV (p=0.418) and metastasis (p=0.379; online supplemental table S25).

The smallest effective dose associated with significant changes in physical function was estimated at ~7 METs.min/week for RE (SMD 0.23 (95% CI 0.15 to 0.30)), ~22 METs.min/week for COMB (SMD 0.23 (95% CI 0.12 to 0.34)) and ~19 METs.min/week for AE (SMD 0.20 (95% CI 0.11 to 0.29)).

The plateau was estimated at ~848 METs.min/week for COMB (SMD 0.51 (95% CI 0.27 to 0.74)) and ~661 METs.min/week for AE (SMD 0.45 (95% CI 0.25 to 0.65)), while no plateau was observed for RE, as the benefits continued to increase progressively across the available dose range (figure 6A). The smallest effective dose and the plateau point in the DR-NMA model under different threshold settings are presented in online supplemental table S26.

Muscle strength

The results indicated that RE at ~277 METs.min/week (SMD 0.68 (95% CI 0.54 to 0.81)) was the most effective intervention

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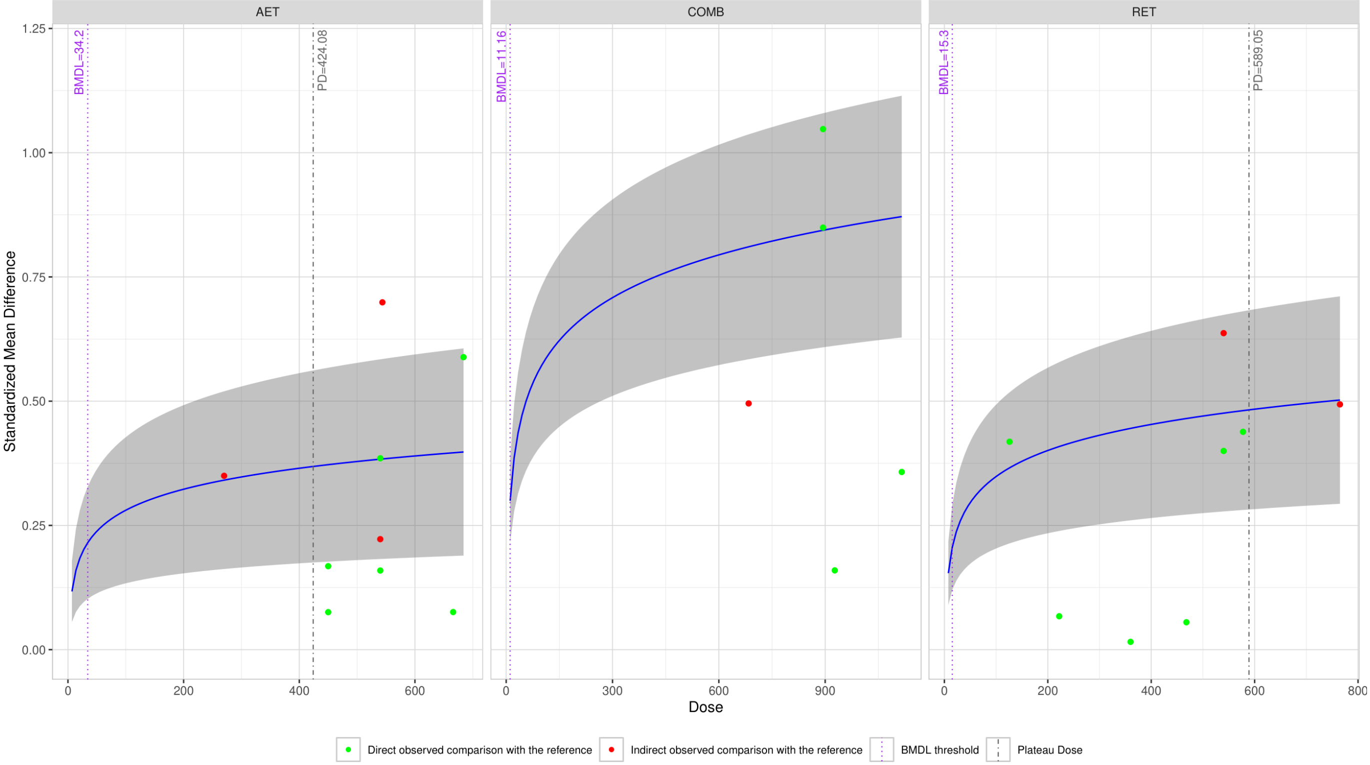

Figure 5 DR-NMA plots for the effects of aerobic, resistance and combined modalities on lean mass. AE, aerobic exercise; BMDL, benchmark dose lower confidence limit; COMB, combined exercises; DR-NMA, dose–response network meta-analysis; PD, plateau dose; RE, resistance exercise.

to improve muscle strength, followed by COMB at ~639 METs.min/week (SMD 0.57 (95% CI 0.40 to 0.75)) and AE at ~410 METs.min/week (SMD 0.26 (95% CI 0.07 to 0.45)) (figure 3). Subgroup NMA based on BMI (p=0.004) is presented in online supplemental table S27. Analyses based on treatment status, age, stage III–IV and metastasis were not conducted due to the insufficient number of studies.

The smallest effective dose associated with significant changes in muscle strength was estimated at ~8 METs.min/week for RE (SMD 0.24 (95% CI 0.20 to 0.29)) and ~18 METs.min/week for COMB (SMD 0.26 (95% CI 0.18 to 0.33)) in muscle strength. The plateau was estimated at ~1607 METs.min/week for COMB (SMD 0.66 (95% CI 0.46 to 0.85)) and ~239 METs.min/week for AE (SMD 0.24 (95% CI 0.06 to 0.41)). The smallest effective dose could not be estimated for AE, as the response reached a plateau relatively early compared with the other interventions (figure 6B).

In contrast, no plateau was observed for RE across the evaluated dose range, with the effect increasing progressively throughout the available dose range (figure 6B). Sensitivity analyses for the smallest effective dose and the plateau point in DR-NMA model under different threshold settings are presented in online supplemental table S28.

DISCUSSION

We examined the effects of different doses and exercise modalities on fatigue, lean mass, physical function and muscle strength in women with breast cancer. We observed a non-linear association between exercise dose and outcomes, indicating a potential therapeutic window characterised by a minimum effective exercise dose (11–22 METs.min/week for COMB, 8–27 METs.min/week for RE and 15–34 METs.min/week for AE), representing ~10–40 min of exercise per week. The plateau was estimated at higher volumes (848–1607 METs.min/week for COMB, 302–589 METs.min/week for RE and 239–661 METs.min/week for AE), indicating that performing more than 40–320 min per week, depending on the exercise modality, did not confer additional benefits. RE and COMB were the most effective exercise modalities. Collectively, these findings emphasise the importance of identifying an effective yet feasible exercise dose, avoiding unnecessarily high exercise doses that may be impractical or burdensome for women undergoing or recovering from breast cancer treatment.

Although numerous epidemiological studies have investigated the association between physical activity and clinical outcomes,[181,182] self-reported physical activity metrics often overestimate the actual range of exercise doses. Previous experimental[99,117,183,184] and meta-analytic[17,185–187] studies have examined structured exercise programmes in patients with cancer, linking exercise prescription characteristics with changes in objectively measured and patient-reported outcomes. These approaches have typically relied on linear assumptions and may not adequately capture the complexity of exercise dose prescription and its effects. Additionally, our findings are broadly consistent with those of our previous NMA,[20] which showed that resistance and COMB programmes were generally more effective for reducing fatigue and improving muscle strength, lean mass and physical function in women with breast cancer. However, some differences were observed, likely due to the smaller number of studies included in the present DR analysis, as only trials reporting detailed exercise prescription were eligible.[52,56–59,61,64–66,68–71,73–76,78,82,84,87,89,90,97–99,103–105,107,109–115,117,118,124,126,128–133,139,140,142,146,149,151–154,156,158,166–169,171–173,176] In addition, the current study extends previous work by modelling DR relationships, which may influence the magnitude and shape of the observed effects. Therefore, we applied a range of DR models

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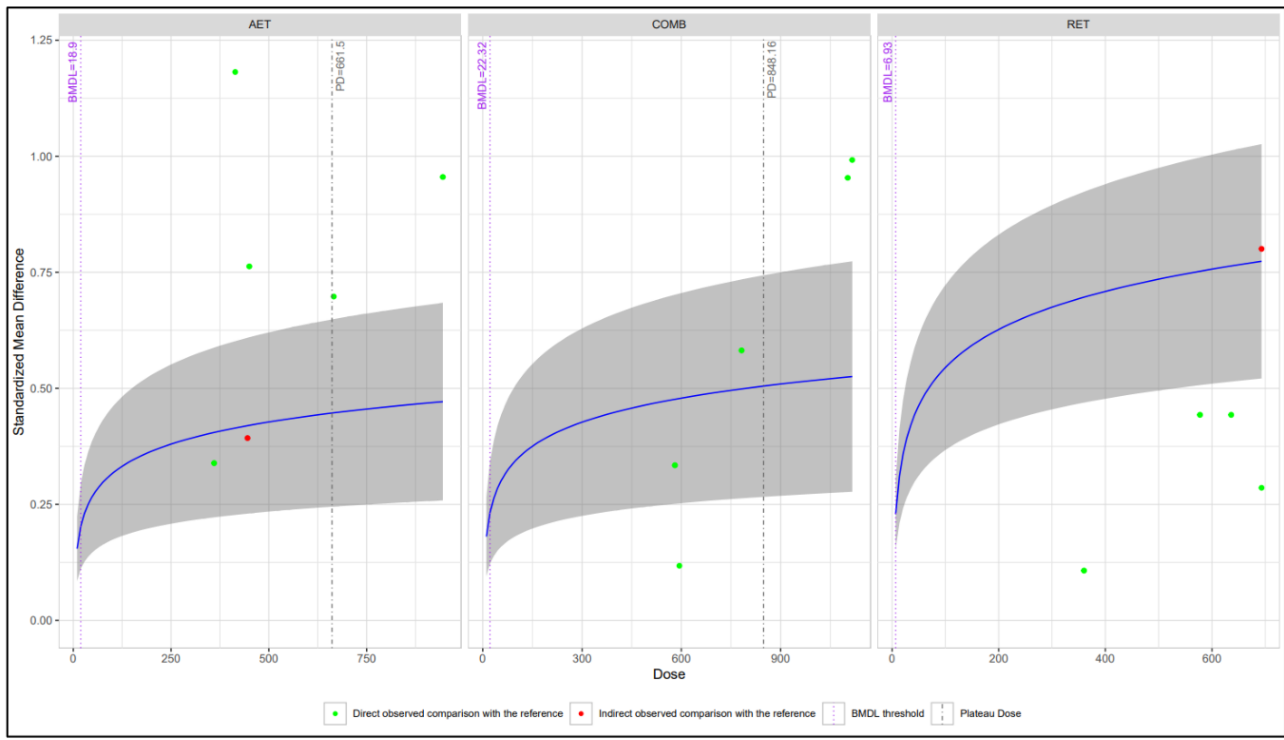

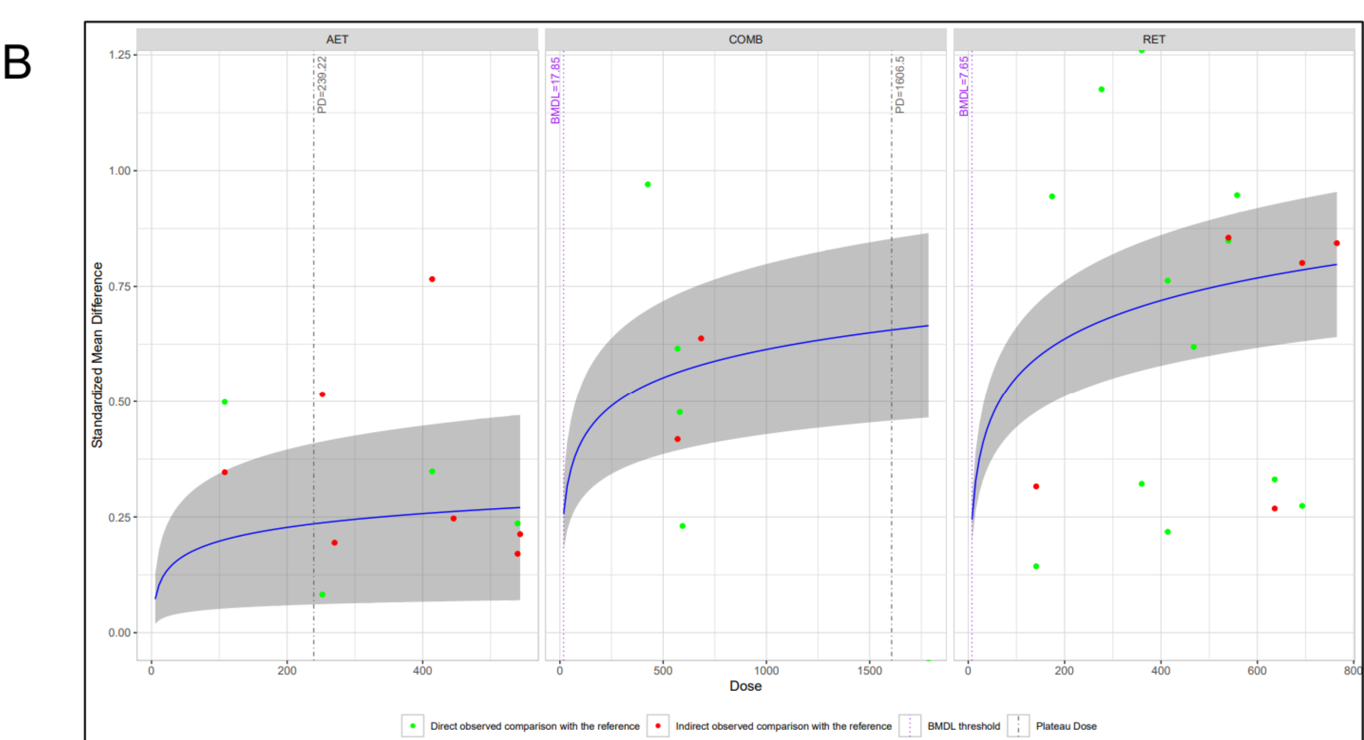

Figure 6 DR-NMA plots for the effects of aerobic, resistance and combined exercise modalities on (A) physical function and (B) muscle strength. AE, aerobic exercise; BMDL, benchmark dose lower confidence limit; COMB, combined exercises; DR-NMA, dose–response network meta-analysis; PD, plateau dose; RE, resistance exercise.

within a DR-NMA framework[49–51] to evaluate dose effects and identify minimum effective doses as well as potential plateaus across different exercise modalities. This approach enables the simultaneous comparison of multiple exercise modalities across a continuum of doses while accounting for potential non-linear DR relationships that traditional NMA or linear methods may fail to detect.

Contrary to our previous hypotheses,[17 183 185 186] benefits from exercise were observed in a non-linear fashion, commencing at very low doses but maximised at moderate doses, beyond which

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the DR curves plateaued. Interestingly, the minimal dose for COMB and RE was set at 8–27 METs.min/week, which may correspond to 10–30 min of moderate-intensity exercise per week. This is highly achievable for patients undergoing primary treatment, considering that exercise and health professionals can accommodate exercise preferences, health status and deconditioning, cyclical variation in symptoms and respect patients' readiness to exercise within a given dose.[188 189] The effectiveness of such relatively small doses may be related to the deconditioning state and lack of prior exercise exposure commonly observed in this population,[190] resulting in rapid early adaptations in the neuromuscular systems[191] and improved metabolic function.[192] As exercise dose increases, these adaptations may approach a ceiling effect, whereby further physiological gains may require disproportionately greater stimulus, which may be limited by treatment-related fatigue, inflammation and reduced recovery capacity. Together, these mechanisms may underpin the non-linear pattern observed across outcomes.[179 193]

As musculoskeletal, neural and cardiovascular systems adapt through repeated exposure to exercise, progression towards higher doses may be recommended. However, this may not always be achievable, as previously observed, particularly in patients with advanced disease, where exercise compliance was around 60%.[58 194] Despite the limited number of studies reporting exercise compliance in women with breast cancer, the observed dose range supports significant benefits in fatigue, lean mass, physical function and muscle strength even when patients are unable to fully achieve the prescribed dose. This is important for patients who frequently experience reduced motivation, symptom burden and treatment-related toxicities[195] that may limit their exercise capacity.

For those who tolerate treatment-related symptoms well during and after primary treatment, the plateau corresponded to 1–2 weekly RE sessions, with or without 3–4 AE sessions, aligning with the internationally recommended 150–300 min of moderate-intensity physical activity per week.[196] Nevertheless, the exercise progression in both dose and complexity should be planned according to individual preferences, goals and physical capacity, ensuring feasibility, compliance, engagement and safety. Collectively, these are major components highlighted by international organisations to support effective exercise prescription and long-term participation among individuals living with and beyond cancer.[8–13]

The relevance of the investigated outcomes is well established for women with breast cancer. Fatigue is one of the most impactful symptoms on activities of daily living and quality of life experienced during and after primary treatment.[2–4] Sarcopenia, frailty and cachexia share similar phenotypic characteristics, including loss of lean mass, physical function and strength,[197] and are significantly associated with shorter overall, cancer-free and progression-free survival.[5 198–200] Additionally, lower levels of physical function[6] and muscle strength[7] are independently associated with reduced overall survival in patients with cancer. We found that RE and COMB were the most effective interventions to improve these outcomes, which may be key determinants of chemotherapy completion and survival benefits.[14 15] Although our review did not directly assess survival, evidence from a large trial in another cancer population, such as the CHALLENGE trial[16] in colon cancer, indicates that exercise may have beneficial effects on survival and recurrence. While these findings cannot be directly extrapolated to breast cancer, they provide a broader clinical rationale supporting the potential role of exercise in improving not only symptom management, activities of daily living and fitness but also long-term disease outcomes in this population.

Strengths of this study are (1) the inclusion of 73 articles comprising 5156 patients with breast cancer, (2) the application of both standard and DR-NMA to compare exercise modalities and prescribed doses and (3) the comparison between the two approaches, indicating that the DR approach produced more precise estimates as reflected by narrower CIs. However, some limitations should be acknowledged. First, there is a potential selection bias in the included studies, which focused on women with early-stage disease who were likely to have fewer complications and treatment-related side effects, limiting generalisability of our findings. Second, we did not examine other clinically relevant outcomes, such as bone health, cardiorespiratory fitness, sleep quality, pain, cognition, distress and quality of life. The exercise dose range identified in this review may not necessarily apply to these outcomes. Third, sensitivity analyses using alternative thresholds for benchmark and plateau in the DR-NMA resulted in variations in the estimated exercise dose range. Therefore, absolute values of minimal exercise doses and plateaus should be interpreted with some caution, particularly when extrapolating to different clinical contexts or outcomes. In addition, the minimum effective doses identified in this review were based on a statistical benchmark threshold (SMD 0.1) which may not necessarily correspond to established minimally clinically important difference values commonly considered meaningful in exercise oncology. Therefore, the clinical relevance of these lower dose thresholds warrants prospective validation. Fourth, several studies with small sample sizes were included, and this may lead to small-sample bias.[201] Fifth, the small number of trials judged to be at low risk of bias for most outcomes precluded sensitivity analyses. Sixth, the DR analyses were based on prescribed exercise doses reported in the included trials rather than actual completed doses. This potential source of measurement error may have contributed additional variability to the estimated DR relationships and plateau points. Seventh, methods for assessing inconsistency, influential studies and P-scores were applied at the study level within the standard NMA framework; however, such methods within DR-NMA are currently limited. Eighth, exploratory subgroup analyses were conducted according to treatment status, age, BMI, disease stage and metastasis. Evidence of subgroup differences was observed only for BMI in the muscle strength outcome; however, this finding should be interpreted cautiously, as only three studies included participants with obesity, and exercise modalities were unevenly distributed across studies. Finally, most included studies were short-term exercise interventions, and it remains unclear whether the adaptations observed at these exercise doses can be maintained over longer periods.

In conclusion, this systematic review and DR-NMA revealed an increasing non-linear DR relationship between exercise dose and improvements in fatigue, lean body mass, physical function and muscle strength among women with breast cancer. The analysis identified that benefits can be achieved with doses as low as ~10–40 min per week. Additionally, exercising more than 40–320 min per week, depending on the exercise modality, may not have additional benefits. COMB exercise programmes were the most effective for improving fatigue and lean mass, whereas RE showed superior effects on physical function and muscle strength. Overall, our findings delineate a potential therapeutic range of exercise modalities and doses that can guide the refinement of exercise medicine towards effective and feasible prescriptions in clinical and community settings. Future studies should aim to confirm these dose thresholds in prospective trials and explore individualised exercise prescriptions based on clinical characteristics, treatment status and patient preferences.

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Author note The results of the study are presented clearly, honestly, without fabrication, falsification or inappropriate data manipulation.

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