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Exercise Reduces Lipid Peroxidation and Boosts Total Antioxidant Capacity in Humans: A Systematic Review and Meta-Analysis

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Abstract

Purpose: Oxidative stress contributes to many chronic diseases. Although physical activity promotes health, its effects on lipid peroxidation (MDA) and total antioxidant status (TAS) remain unclear. This meta-analysis quantitatively synthesized the effects of physical activity on MDA and TAS in humans. **Method:** This systematic review and meta-analysis was conducted in accordance with the PRISMA 2020 guideline and registered with PROSPERO (CRD420251144711). A systematic literature search was performed across PubMed, Scopus, and Web of Science covering the period from January 2000 to November 2025. Human studies evaluating the impact of either acute or chronic physical activity on MDA and TAS concentrations were included. Pooled effect sizes were estimated using a random-effects model, and standardized mean differences (SMD) with 95% CI were calculated. Heterogeneity was assessed using the I^2 statistic.

Results: Out of 620 initially identified records, 21 studies met the eligibility criteria, of which 10 and 4 were suitable for meta-analysis of MDA and TAS, respectively. Physical activity resulted in a significant reduction in MDA levels (SMD = –1.02; 95% CI: –1.95 to –0.09; $p = 0.032$). Moreover, physical activity led to a highly significant increase in TAS (SMD = +0.76; 95% CI: 0.38 to 1.15; $p < 0.001$). Heterogeneity was substantial for both analyses ($I^2 = 94.6\%$ for each). Subgroup analyses revealed that the decline in MDA was more pronounced among individuals with a body mass index above 25 (BMI > 25) and those younger than 50 years ($p < 0.05$). **Conclusion:** Regular, structured physical activity effectively lowers lipid peroxidation and enhances total antioxidant capacity in humans. These findings strengthen the role of physical activity as an effective non-pharmacological strategy for modulating systemic oxidative stress and reducing the risk of related diseases.

Keywords: Physical activity, Oxidative stress, Malondialdehyde (MDA), Total antioxidant status (TAS), Meta-analysis.

Introduction

Physical activity was widely recognized as a cornerstone of preventive medicine and a critical determinant of overall health and longevity(Kohl III et al., 2025; Nosrati et al., 2022). Its beneficial effects extended across multiple physiological systems, contributing to a reduced risk of cardiovascular disease, metabolic disorders, certain cancers, and neurodegenerative conditions(Taylor et al., 2023). Despite these well-established advantages, the underlying biological mechanisms through which physical activity conferred protection remained an area of active investigation(Shojah-anzabi et al., 2026; Warburton & Bredin, 2017). One of the most important and increasingly studied pathways involved the modulation of oxidative stress, which we now define as a state characterized by an imbalance between reactive oxygen species (ROS) production and the body's antioxidant defenses(Sies, 2015).

Oxidative stress played a central role in the pathogenesis of numerous non-communicable diseases, including atherosclerosis, type 2 diabetes, obesity, and chronic inflammatory conditions(Jones & Sies, 2015). Excessive ROS could damage lipids, proteins, and DNA, leading to cellular dysfunction and contributing to disease progression. Among the various biomarkers used to assess oxidative stress, malondialdehyde (MDA) was one of the most widely studied indicators of lipid peroxidation, as it reflected oxidative damage to cell membranes(Ayala et al., 2014). Conversely, total antioxidant status (TAS) represented the cumulative action of both enzymatic and non-enzymatic antioxidants in neutralizing free radicals, thereby providing a comprehensive measure of the body's antioxidant capacity(Ghiselli et al., 2000). Taken together, MDA and TAS offered valuable insight into the balance between oxidative damage and antioxidant defense.

Physical activity exerted a complex and somewhat dual effect on oxidative stress. Acute bouts of exercise were known to transiently increase ROS production due to elevated oxygen consumption and metabolic activity(Powers & Jackson, 2008). This short-term increase in oxidative stress was traditionally viewed as potentially harmful;

however, emerging evidence suggested that it might play a beneficial signaling role, stimulating adaptive responses that enhanced endogenous antioxidant systems(Radak et al., 2013). Regular, moderate-to-vigorous physical activity was therefore associated with improved oxidative balance, increased expression of antioxidant enzymes, and greater resilience against oxidative damage. This adaptive process, often described as hormesis, underscored the importance of exercise intensity, duration, and frequency in determining its net physiological impact (Radak et al., 2008; ShojaAnzabi et al., 2023).

Despite growing interest in this area, the literature examining the relationship between physical activity and oxidative stress biomarkers remained heterogeneous and, at times, inconsistent. Variability in study design, population characteristics, exercise protocols, and biomarker measurement methods contributed to conflicting findings(Caturano et al., 2023; Kawamura & Muraoka, 2018). Some studies reported significant reductions in MDA levels and improvements in TAS following structured exercise interventions, while others observed minimal or even opposite effects, particularly in response to high-intensity or unaccustomed exercise. Additionally, factors such as age, body mass index (BMI), baseline fitness level, and underlying health conditions influenced individual responses to physical activity, which further complicated interpretation(Gomez-Cabrera et al., 2008; Nosrati et al., 2022).

Accordingly, this systematic review and meta-analysis was designed to address four specific research questions: (1) What is the pooled effect of physical activity on MDA levels as a marker of lipid peroxidation in humans? (2) What is the pooled effect of physical activity on TAS as a marker of non-enzymatic antioxidant capacity in humans? (3) Does age (≤50 years vs. >50 years) moderate the magnitude of exercise-induced changes in MDA levels? and (4) Does BMI (≤25 kg/m² vs. >25 kg/m²) significantly influence the MDA response to exercise training? By addressing these questions, we aim to quantify the overall effect of exercise on oxidative stress biomarkers, identify key sources of

heterogeneity, and provide evidence-based insights that can inform the design of exercise interventions for oxidative stress management in both healthy and clinical populations.

Methods

Study design and protocol registration

This study was conducted as a quantitative systematic review and meta-analysis, strictly following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guideline to ensure transparency, reproducibility, and minimization of bias throughout all research steps(Page et al., 2021). The protocol was registered in the PROSPERO international database (registration number: CRD420251144711) before the literature search and data extraction began. The primary research question was structured according to the PICO framework (Population, Intervention, Comparison, Outcome), which included human populations of any age or sex, structured physical activity or exercise interventions, comparison with sedentary individuals or baseline values, and the primary outcomes of MDA and TAS.

Inclusion and exclusion criteria

Studies were included if they met the following criteria: (1) original peer-reviewed research articles published in English between January 2000 and November 2025; (2) human studies involving any age, sex, health status, or disease condition; (3) investigation of structured physical activity or exercise interventions, including either acute (single session) or chronic (≥2 weeks) protocols such as aerobic/endurance training, resistance training, high-intensity interval training (HIIT), or combined exercise; (4) reporting of at least one oxidative stress biomarker, specifically MDA (as an index of lipid peroxidation) or TAS (as an index of non-enzymatic antioxidant capacity), measured in human biological samples (whole blood, plasma, or serum); (5) provision of quantitative data (mean and standard deviation before and after the intervention, or sufficient statistical information to calculate

effect sizes); and (6) adequate description of exercise intervention characteristics (intensity, duration, and type).

Exclusion criteria were: (1) studies lacking a pure physical activity or exercise stimulus (e.g., supplement-based studies without an independent exercise arm); (2) absence of direct MDA or TAS measurement; (3) exclusive focus on functional or performance outcomes without any link to oxidative stress mechanisms; (4) insufficient reporting of exercise intervention details (intensity, duration, or type) that prevented interpretation of exercise effects; (5) review articles (narrative, systematic, or meta-analyses), editorials, letters to the editor, or case reports; (6) study protocols or methodological papers without primary outcome data; and (7) studies unrelated to exercise-induced oxidative stress in humans.

Information sources and search strategy

The search strategy was designed a priori to comprehensively identify studies examining the effects of physical activity on oxidative stress biomarkers in humans. A systematic search was performed in three major databases, PubMed (Medline), Scopus, and Web of Science, by two independent authors (R. F. and R. A.) covering the period from January 2000 to May 2026. The search strategy combined free-text keywords and, where applicable, Medical Subject Headings (MeSH) terms related to physical activity and oxidative stress biomarkers. The main search concepts included: (1) physical activity or exercise (namely, "exercise", "physical activity", "training", "aerobic training", "resistance training", "treadmill exercise"); (2) oxidative stress and redox biology; and (3) specific oxidative stress biomarkers including "MDA", "malondialdehyde", "TAS", and "total antioxidant status". These terms were combined using Boolean operators ("AND", "OR", and "NOT") and truncation symbols to maximize search sensitivity. The search was restricted to articles published in English. In addition to database searching, backward and forward citation tracking was performed by manually screening the reference lists of all included

studies and relevant review articles, as well as examining articles that had cited the included studies, to identify any additional eligible studies.

Study selection process

All records retrieved from the database searches were imported into EndNote reference management software (version 2025.3.1), and duplicates were removed. The study selection process followed a two-stage approach according to PRISMA guidelines. In the first stage, titles and abstracts were independently screened by two authors (R. F. and R. A.) based on the predefined inclusion and exclusion criteria. Clearly irrelevant studies (those without exercise, animal studies, or those not reporting oxidative stress biomarkers) were removed at this stage. In the second stage, the full texts of all potentially eligible studies were retrieved and independently assessed by the same two authors for final inclusion. Any disagreements during screening or full-text evaluation were resolved through discussion and consensus; if necessary, a third author was consulted. Reasons for exclusion at the full-text stage (inappropriate population, absence of a structured exercise intervention, lack of oxidative stress biomarker measurement, or insufficient reporting of exercise characteristics) were recorded.

Data extraction

After finalizing the eligible studies through the two-stage screening process, data extraction was independently performed by two authors (R. F. and R. A.) using a standardized, pre-tested data extraction form. Extracted data were cross-checked to ensure accuracy and completeness. For each included study, the following information was recorded: (1) study characteristics (first author, year of publication, country, and study design); (2) participant characteristics (sample size, age, sex, health or disease status, and baseline oxidative stress biomarker profile when available); (3) intervention or exposure characteristics (exercise type [aerobic, resistance, high-intensity interval], exercise intensity, session duration, sessions per week, and total intervention duration); (4) outcome measures (MDA and TAS

levels, including biological sampling time relative to exercise); and (5) additional notes (reported limitations, potential sources of bias, or methodological considerations relevant to result interpretation).

Statistical analysis

Meta-analysis

All statistical analyses were performed using Comprehensive Meta-Analysis software (version 4). Due to expected heterogeneity between studies, a random-effects model with restricted maximum likelihood (REML) estimation was applied, and the Hartung-Knapp adjustment was used to provide more robust confidence intervals (CI)(Mengersen & Schmid, 2013). Effect sizes were calculated as standardized mean differences (SMD) with 95% CI for the continuous outcomes of MDA and TAS. When numerical data were presented only in graphical form, values were extracted using GetData Graph Digitizer software (version 2.26). Studies that lacked an appropriate comparison group, presented only single-group outcomes without extractable dispersion measures, reported results solely as medians without sufficient variability measures, or provided data that could not be reliably extracted for meta-analysis were excluded from the quantitative synthesis.

2.6.2. Heterogeneity assessment

Between-study heterogeneity was assessed using Cochrane's Q statistic, the I^2 index, and Tau^2 (τ^2). The Q test determined whether effect size dispersion exceeded sampling error alone ($p < 0.05$ indicated potential heterogeneity). Because the Q test is sensitive to the number of studies, it was interpreted alongside other indices (16). I^2 values of 25%, 50%, and 75% were interpreted as low, moderate, and high heterogeneity, respectively. Tau^2, estimated using the REML method within the random-effects model, provided an estimate of the absolute between-study variance(Petitti, 2001).

2.6.3. Subgroup analyses

Subgroup analyses were conducted to explore potential sources of heterogeneity based on intervention and participant characteristics.

Studies were categorized according to mean age (≤30 years vs. >30 years for glutathione and ≤50 years vs. >50 years for other biomarkers), and BMI ≤ 25 vs. >25 kg/m². Pooled effect sizes were estimated using a random-effects model, and subgroup differences were assessed using the between-subgroup Q statistic (Q_between), with $p < 0.05$ considered statistically significant.

Sensitivity analysis and publication bias

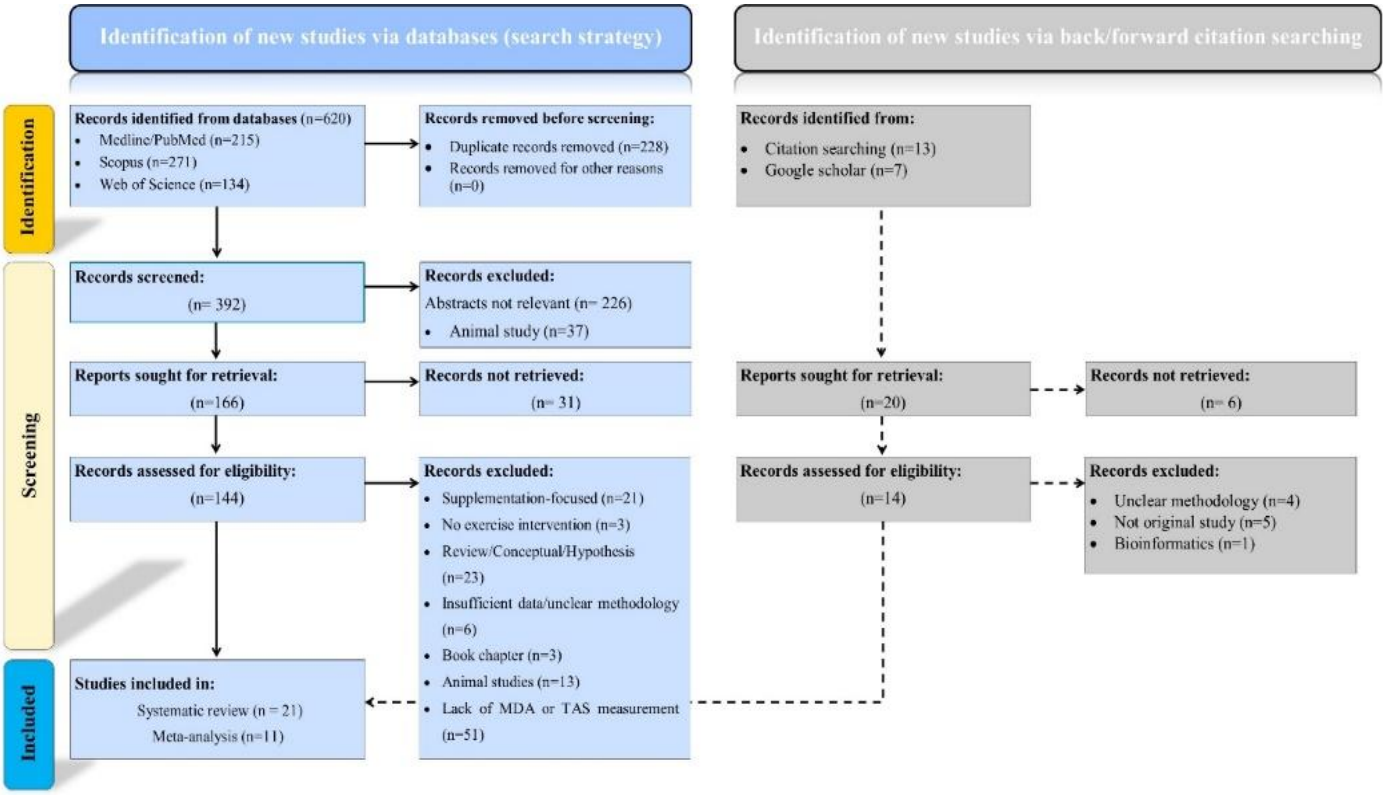
Sensitivity analysis was performed using the leave-one-out method (removing one study at a time) to evaluate the robustness of the pooled estimates and the influence of each individual study on the overall results(Thornton & Lee, 2000). To assess potential publication bias and small-study effects, a funnel plot was used together with Egger's regression test ($p < 0.05$ indicated potential bias). Additionally, the fail-safe N (Rosenthal's method) was calculated to estimate the number of missing studies that would be needed to render the pooled results non-significant(Hattori & Zhou, 2018).

Results

Study selection

The study selection process is illustrated in the PRISMA flow diagram (Fig. 1). Database searches (PubMed, Scopus, Web of Science) yielded 620 records. After removing 228 duplicates, 392 records underwent title and abstract screening, where 229 records (including 37 animal studies) were excluded. Of the 166 reports sought for retrieval, 31 could not be accessed, leaving 144 studies assessed for eligibility. Exclusions included: supplementation-focused (n=21), no exercise intervention (n=3), review/conceptual papers (n=23), insufficient data (n=6), book chapters (n=3), animal studies (n=13), and lack of MDA/TAS measurement (n=51). Finally, 21 studies were included in the systematic review. Additionally, citation searching and Google Scholar identified 20 reports. After removing 6 non-retrieved records, 14 were assessed; 10 were excluded (unclear methodology, not original, bioinformatics). No new studies were added. Ultimately, 21 studies

were included in the systematic review, of which 11 were eligible for



meta-analysis (MDA and TAS).

Fig. 1. PRISMA flow diagram illustrating the study selection process, including identification, screening, eligibility assessment, and final inclusion of studies**.**

Characteristics of included studies

A total of 21 studies met the inclusion criteria for the systematic review, of which 11 provided extractable data for the meta-analysis of MDA and TAS. The characteristics of the included studies are summarized in Table 1. The studies were published between 2016 and 2023 and conducted across 14 countries, including Saudi Arabia, South Korea, Brazil, Taiwan, Italy, Poland, Spain, Slovenia, Egypt, Germany, Portugal, and others. Study designs included randomized controlled trials (n=8, 38%), cross-sectional studies (n=5, 24%), longitudinal

observational studies (n=3, 14%), acute crossover trials (n=3, 14%), and pre-post clinical studies (n=2, 10%). Sample sizes ranged from 11 to 552 participants (mean ≈ 65). Participant ages spanned from 12 to 95 years, encompassing both sexes (male-only: 9 studies; female-only: 6 studies; mixed: 6 studies). Target populations included healthy older adults, children with intellectual disability, patients with heart failure (HFrEF), irritable bowel syndrome (IBS), fibromyalgia, Down syndrome, as well as elite soccer and rugby players. Exercise interventions varied widely: aerobic training (7 studies), combined aerobic-resistance training (3 studies), high-intensity interval training (2 studies), resistance training (2 studies), team sports (3 studies), and acute single-session interventions (4 studies). Intervention durations ranged from acute single bouts to 9 months, with training frequencies of 2–5 sessions per week and intensities from moderate (50–70% HRmax) to maximal.

Table 1. Summary of 21 studies investigating the effects of various exercise modalities on biomarkers, including study design, population characteristics, intervention details, measured outcomes, and key findings related to oxidative stress responses.

Author (ref)	Year	Country	Study design	Sample size & participant characteristics	Study population	Control group	Intervention type	Duration & frequency	Intensity of training	Mode of exercise	Redox biomarkers measured	Numerical results	Key findings	Limitations	Authors' conclusion
Alghadir et al. **(19)**	2016	Saudi Arabia	Randomized controlled trial (RCT)	N=100 healthy older adults; age 65–95 yrs	Healthy older adults	Sedentary control group (n=50)	Moderate-intensity aerobic exercise (walking/treadmill; MAET)	24 weeks; 3 sessions/week	Moderate (60–70% HRmax)	Walking/treadmill	MDA, 8-OHdG, TAC	TAC increased; MDA and 8-OHdG decreased (all $p<0.001$)	Moderate aerobic training improved redox status (↑TAC, ↓MDA, ↓8-OHdG) and cognitive function (MMSE).	No long-term follow-up; restricted to generally healthy older adults.	Moderate aerobic exercise is a safe and effective strategy to enhance antioxidant capacity and cognitive performance in older adults.
Park & Kwak **(20)**	2016	South Korea	Cross-sectional observational study	N=60 healthy young males (20 UT, 20 ET, 20 RT); age 26.6±0.8 yrs	Healthy young men with different training backgrounds	Untrained individuals (UT)	Long-term aerobically trained (ET) and resistance-trained (RT) vs untrained	-	Not applicable (background training only)	Aerobic vs resistance training background	MDA, protein carbonyls (PC), TAC	Resting MDA and PC did not differ; post-exercise MDA and PC increased in UT but not in ET or RT; TAC similar across groups	Chronic aerobic and resistance training blunted the acute exercise-induced rises in lipid and protein oxidation markers.	Cross-sectional design; training volume/intensity not standardized; TAC showed no group differences.	Both aerobic and resistance training enhance redox defence and protect against acute exercise-induced oxidative stress in trained individuals.

Puga et al. **(21)**	2016	Brazil	Randomized, placebo-controlled crossover trial	N=16 normotensive postmenopausal women; age 57±6 yrs; BMI 27.6±4.6 kg/m²	Normotensive postmenopausal women	PLA+rest and PLA+exercise (within-subject comparators)	L-arginine (3 g) ingestion plus acute aerobic exercise vs other conditions	Acute; single session (4 conditions)	45 min at 50% VO2max (moderate)	Treadmill walking/running	MDA, AOPP, SOD activity, NOx	L-Arg+AE reduced systolic BP vs PLA+rest (P<0.05); MDA decreased after AE alone but not after L-Arg+AE; NOx highest in L-Arg+AE (P<0.05)	L-arginine plus exercise maximized NO bioavailability and BP reduction but blunted the beneficial exercise-induced decline in MDA, suggesting interference with ROS signalling.	Small sample; single acute dose (3 g L-Arg); limited to normotensive women.	Combining acute L-arginine with aerobic exercise is effective for BP lowering in postmenopausal women, but may attenuate beneficial redox adaptations to exercise.
Tsai et al. **(22)**	2016	Taiwan	Clinical pre-post study (single-group)	Number not specified; patients with BPPV	Patients with benign paroxysmal positional vertigo	Pre-maneuver baseline within subjects	Canalith repositioning procedure (CRP)	Acute; single maneuver session	Not applicable (therapeutic maneuver)	CRP maneuver exercise	SIRT1, miR-34a, ROS, MDA, IL-1β, IL-6, IL-8, TNF-α	Post-CRP: SIRT1 increased, miR-34a decreased; ROS, MDA and pro-inflammatory cytokines were attenuated	CRP induced epigenetic shifts (↑SIRT1, ↓miR-34a) associated with reductions in systemic oxidative stress and inflammation.	Sample size and detailed methods not fully specified; systemic markers only.	Therapeutic efficacy of CRP in BPPV is likely mediated by SIRT1/miR-34a-related epigenetic regulation that dampens oxidative stress and inflammation.
Becatti et al. **(23)**	2017	Italy	Observational longitudinal pre-post season trial	N=25 male elite soccer players; age 20.3±2.4 yrs	Professional male soccer players under chronic competitive stress	Within-subject comparison (start vs end of season)	Full competitive soccer season (training + matches)	~9 months; daily training plus weekly matches	High competitive/exhaustive load	Soccer training and matches	RBC thiols, RBC lipid hydroperoxides (LHs), neutrophil total ROS, plasma AOPP, cfDNA, TAS	RBC thiols ↓ (17.5→13.7 nmol/mg, p<0.05); RBC LHs ↑; neutrophil ROS ↑; AOPP ↑; cfDNA ↑; TAS unchanged	Season-long load caused increased oxidative and cellular damage with reduced erythrocyte antioxidant defences, without change in total antioxidant status.	No external control group; focus on peripheral blood markers only.	A demanding competitive season induces marked redox imbalance and cellular damage in elite soccer players, with depletion of erythrocyte antioxidant reserves.

Georgescu et al. **(24)**	2017	Brazil	Randomized, counterbalanced crossover trial	Healthy men and women (exact N not specified here)	Recreationally active adults	Euhydration (exercise with water ingestion)	Exercise leading to dehydration (no fluid) vs euhydrated exercise	~60-min cycling bouts; two sessions with washout	Moderate to high (set to induce ~3% body-mass loss in dehydration trial)	Cycling	MDA, protein carbonyls (PC), TAC (Trolox equivalents)	Dehydration: MDA +~18%, PC +~8.5%, TAC +~21%; euhydration: minimal/no significant changes	Exercise-induced dehydration (3% body-mass loss) increased both oxidative damage (MDA, PC) and antioxidant capacity (TAC), whereas maintaining hydration prevented these changes.	Lab cycling may not reflect real-world exercise conditions fully.	Dehydration during exercise exacerbates oxidative stress and mobilization of antioxidant defences; adequate fluid intake effectively attenuates exercise-induced oxidative stress.
González-Garrido et al. **(25)**	2017	Mexico	Non-randomized pre-post case-control–type study	N=15 male professional soccer players; age 15–19 yrs (median 17.0±1.11)	Young highly trained professional soccer players	Pre-supplementation baseline within subjects	Daily 100% cocoa powder (0.375 g/kg/day; ~25 g/day) in water	7 days; daily intake during usual training (5×/week + 1 match/week)	High-intensity habitual soccer training (80–90% HRmax)	Soccer training; 12-min Cooper test	MDA, protein carbonyls, TAC, GSH-Px, thiol groups (SH), CK, LDH	MDA −32.4%; carbonyls −26.3%; TAC +16%; GSH-Px +26.4%; CK −39.4%; LDH −23%; Cooper distance +4% (all significant)	Short-term cocoa reduced oxidative and muscle damage markers and increased antioxidant defences, accompanied by improved endurance performance.	No concurrent placebo-controlled group; unexpected rise in 4-HNE reported elsewhere; small N.	Daily cocoa consumption may benefit athletes by improving redox status, reducing muscle damage and enhancing endurance performance.
Hajizadeh Maleki et al. **(26)**	2018	Iran	Randomized controlled trial	N=60 sedentary women with IBS (EX n=30, SED n=30); age 34.8±4.5 yrs	Sedentary women with moderate–severe IBS (Rome III)	Sedentary control instructed to maintain usual lifestyle	Supervised aerobic training (treadmill walking/running)	8 weeks; 3 sessions/week; 45 min/session	Low-to-moderate rate (50–70% HRR, progressed)	Treadmill aerobic exercise	IBS-SS, TNF-α, IL-6, IL-10, TAC, MDA	EX: IBS-SS ~−30%; MDA ~−20%; TAC ~+15%; TNF-α decreased (all $p<0.05$ vs SED)	Low–moderate aerobic training reduced IBS severity, improved antioxidant status and lowered pro-inflammatory cytokines.	Women with IBS only; relied on systemic markers, no gut tissue measures.	Low-to-moderate aerobic exercise is a safe, effective non-pharmacological therapy for women with IBS via restoration of redox balance and reduction of inflammation.

Jamurtas et al. **(27)**	2018	Greece	Acute randomized crossover study (HIIT vs CET)	N=12 healthy young men; age 22.1±1.2 yrs	Healthy physically active young males	Each mode served as comparator (HIIT vs CET)	Low-volume HIIT vs continuous endurance training (CET)	Single acute bout of each mode	HIIT: 4×30 s all-out with 4-min recovery; CET: 45 min at 60% VO2peak	Cycling	Leukocyte counts, PC, MDA, TAC	Both HIIT and CET ↑ leukocytes; CET caused greater PC and MDA rises; HIIT did not significantly increase PC; TAC increased similarly in both	Low-volume HIIT elicited smaller oxidative and inflammatory stress than longer CET while similarly enhancing antioxidant capacity.	Acute responses only; total work and duration differed between modes.	Low-volume HIIT may be a less metabolically stressful alternative to CET, achieving antioxidant benefits with less oxidative damage.
Sielski et al. **(28)**	2018	Poland	Acute within-subject crossover (100% vs 70% BW)	N=15 young amateur female athletes; age 22.0±2.1 yrs; BMI 21.1±1.8	Physically active young women	100% body-weight running as reference	3-min submaximal AlterG treadmill test at 100% vs 70% BW	Single acute 3-min test per condition	60% HRmax (submaximal)	AlterG treadmill running	LOOH, TBARS, PC, TAS, SOD, CAT, IL-6, hs-CRP	LOOH increased only at 100% BW; TAS rose significantly after 70% BW; TBARS, PC similar; IL-6 and hs-CRP unchanged	Reduced weight-bearing (70% BW) lowered oxidative damage and enhanced TAS versus full BW at matched relative intensity.	Very short test; plasma markers only; no longer or harder protocols.	Anti-gravity treadmill running at 70% BW yields a more favourable acute redox profile than full BW exercise in young female athletes.
De la Rosa et al. **(29)**	2019	Spain	Cross-sectional comparative study (trained vs sedentary)	N=86 men (4 groups; middle-aged trained MTG n=24, sedentary MSG n=25)	Healthy young and middle-aged men; MTG are amateur rugby players (54.3±6.6 yrs, 5.1±2.6 h/week)	Middle-aged sedentary group (MSG)	Long-term habitual team-sport training vs sedentary lifestyle	Long-term (MTG ~35±15 yrs of practice)	MTG ~5.1 h/week	Amateur rugby (team sport)	Serum BDNF, plasma CTSB, MDA, protein carbonyls	MTG had better immediate memory; lower MDA and protein carbonyls; BDNF and CTSB inversely correlated with weekly exercise hours	Long-term training associated with better memory and lower neurotrophic and oxidative-stress markers in middle-aged men.	Cross-sectional ; trained group all rugby players.	Long-term exercise appears protective against age-related memory loss and neurodegeneration via favourable modulation of peripheral neurotrophic and redox markers.

Debevec et al. **(30)**	2019	Slovenia	Cross-sectional/intervention with three hypoxia/exercise tests	N=35 men (21 pre-term, 14 full-term)	Healthy active young men born pre-term vs full-term	Full-term men as controls	Hypoxia chemosensitivity test, graded exercise in normoxia and hypoxia	Three visits; graded test until exhaustion (40 W every 2 min)	Moderate to maximal, depending on stage	Cycle ergometer	AOPP, MDA, nitrotyrosine, SOD, FRAP, GPx, catalase	Pre-terms had lower resting hypoxic ventilatory response and greater desaturation; oxidative-stress markers did not differ between groups pre- or post-tests	Pre-term birth altered ventilatory responses but did not translate into different oxidative-stress profiles during hypoxic exercise.	No redox differences detected; limited to young active males.	Active adults born prematurely tolerate hypoxic exercise similarly to full-term peers, though blunted resting HVR suggests caution during prolonged hypoxia.
Alghadir & Gabr **(31)**	2020	Egypt	Pre–post intervention study	N=65 schoolchildren with mild–moderate ID; age 12–18 yrs; IQ 35–70	Children with intellectual disability	Pre-training baseline	Moderate-intensity aerobic training	12 weeks; 3 sessions/week; 45–60 min	65–75% training HR (~30–45% VO2max)	Treadmill walking/running	MDA, 8-OHdG, TAC, NO, total oxidative stress	Training improved IQ and motor scores; TAC increased; MDA, 8-OHdG, NO and total-OS decreased; redox changes explained ~78–93% of ID variation in regression models	Moderate aerobic training enhanced cognitive and motor performance via improved redox balance.	No sedentary control arm; limited to children with ID.	Twelve weeks of moderate aerobic training benefits intellectual and motor function in schoolchildren with ID by improving systemic redox status.
Cho et al. **(32)**	2020	South Korea	Randomized acute exhaustive-exercise trial stratified by CYBA genotype and fitness	N=32 young adults; 8 per genotype–fitness group	Healthy students in their 20s	Baseline (IBE) within groups	Single exhaustive VO2max test	Single bout	Exhaustive to VO2max	Likely treadmill or cycling	Plasma SOD, MDA, lymphocyte DNA damage (TD, TL, TM)	CC+MF group had higher MDA and DNA damage than T+HF at IAE/30 MAE; only HF groups showed MDA recovery at 30 MAE	CYBA C242T genotype and aerobic fitness jointly influenced susceptibility to exercise-induced oxidative stress and DNA damage.	Small n=8 per group; acute responses only.	High fitness and T allele confer protection against redox imbalance and DNA damage after exhaustive exercise.

Mooren et al. **(33)**	2020	Germany	Single-group test–retest RCT with probiotic	N=19 untrained adults, 18–35 yrs	Untrained men and women	Pre-supplement exhaustive-exercise test	4 weeks of E. coli Nissle 1917 (EcN) probiotic	4 weeks; 5 mL EcN daily	Exhaustive treadmill bout (60–80% VO2max) pre- and post-intervention	Treadmill running	Zonulin, claudin-3, LPS, I-FABP, TAC, MDA, IL-6, CRP	Pre-test: zonulin and TAC increased/decreased post-exercise; post-EcN: no zonulin rise, attenuated TAC drop; I-FABP and inflammation unchanged	EcN prevented exercise-induced increase in intestinal permeability marker zonulin and partially preserved TAC.	No placebo-exercise arm; interpretability limited.	Four weeks of EcN probiotic can protect against exercise-induced "leaky gut" by maintaining tight-junction integrity.
Lazar-Poloczek et al. **(34)**	2021	Poland	Cross-sectional clinical study	N=552 HFrEF patients, Weber classes A–D	HFrEF with reduced EF	Between-class comparisons (A–D)	Usual care; severity stratified by CPET	Not applicable	Not applicable	CPET + 6-min walk	CER, TOS, TAC, MDA, SH	CER and MDA increased, SH decreased from class A→D; LVEF, 6-min WT declined; NT-proBNP rose; CER predicted by TOS, MDA, ALP (R^2=0.14)	Worsening HF severity associated with higher oxidative stress (↑CER, MDA) and lower antioxidant SH, paralleling poorer function.	Observational; no intervention.	Systemic oxidative-stress markers track functional severity in HFrEF and may help risk-stratify patients.
Leite-Almeida et al. **(35)**	2021	Portugal	Cross-sectional PA–BMI analysis	Sample not specified here; prepubertal NW and OW/OB children	Prepubertal children, NW vs OW/OB	Lower-active peers within BMI strata	Habitual physical activity (frequency index)	Chronic habitual PA	Not prescribed	General PA	Plasma TAS, urinary NOx, HOMA-IR	In NW, higher PA linked to ↑U-NOx; in OW/OB, higher PA linked to ↑P-TAS and ↓HOMA-IR	PA improved NO bioavailability in NW and antioxidant capacity/insulin sensitivity in OW/OB.	Observational; self-reported PA.	Regular PA is protective in children, especially OW/OB, by enhancing antioxidant status and insulin sensitivity.

Rodríguez et al. **(36)**	2021	Spain	Randomized controlled trial	N=36 sedentary men with Down syndrome (IG 18, CG 18); age 28.4±3.3 yrs; BMI 31.4 kg/m²	Sedentary adult males with DS	Non-training DS controls	Supervised circuit resistance training	12 weeks; 3×/week	40%→50% 8RM; 90 s rest	Circuit RT (6 machines)	TAS, GSH, GR, MDA, carbonyls, vit E, vit C, strength	TAS and GR increased; MDA and carbonyls decreased; handgrip strength improved	RT enhanced antioxidant defences, reduced oxidative damage and increased strength in men with DS.	Small DS cohort.	Circuit RT is effective for improving redox balance and muscle strength in sedentary adults with Down syndrome.
Póvoas et al. **(37)**	2022	Portugal	Observational tournament study	N=48; analysis on 13 who played all matches (7 high-rank, 6 low-rank)	Elite female footballers in FIFA tournament	High-rank vs low-rank teams and time course	Match play across 4 official games	8-day tournament; 4 matches, 48–72 h apart	High-intensity match demands	Football matches	CK, CRP, TAS, uric acid, match-load metrics	CK and CRP varied post-match; baseline TAS and UA higher in high-rank players; low-rank players accumulated more minutes	Better-ranked team had higher baseline antioxidant capacity; load stayed stable but lower-rank players had higher cumulative exposure.	Very small final sample; observational.	Higher chronic antioxidant protection (TAS/UA) may characterize higher-rank elite teams during congested match schedules.
dos Santos et al. **(38)**	2023	Brazil	Randomized controlled trial	N=40 women with FM (20 WBVT, 20 untrained)	Women with fibromyalgia	Untrained control	Whole-body vibration training (WBVT)	6 weeks; 3×/week; 10 min	30 Hz, 2 mm amplitude	WBV platform	TBARS, irisin, visceral adipose tissue (VAT)	WBVT: irisin ↑ (~317→478 mg·dL⁻³), TBARS ↓ (0.24 vs 0.39 nmol MDA/mg protein vs UN), VAT ↓ (p=0.001)	WBVT improved redox status (lower TBARS), increased irisin and reduced VAT in FM patients.	Short duration; only women with FM.	WBVT is a promising adjunct therapy in FM, improving adiposity and oxidative-stress markers while increasing irisin.

Lisi et al. **(39)**	2023	Italy	Experimental proof-of-principle	N=19 young men (trained vs untrained)	Healthy young males; VO2max-based groups	Untrained controls; within-subject pre/post	Short-term aerobic training in untrained	4 weeks	Not specified	Aerobic exercise	EV number/size, EV TAS, EV protein carbonyls	Trained vs untrained: lower EV carbonyls and higher TAS/EV; after 4 weeks training, untrained EV profile shifted toward trained (↓carbonyls, ↑TAS/EV, ↓EV count)	Circulating EV redox cargo reflects fitness level and improves with short-term training.	Small N; only males.	EV redox status is a sensitive systemic fitness biomarker, normalized toward "trained" profile after 4 weeks of aerobic training.

Abbreviations: RCT, randomized controlled trial; MDA, malondialdehyde; TAS, total antioxidant status; TAC, total antioxidant capacity (method-dependent); TBARS, thiobarbituric acid reactive substances (equivalent to MDA); HRmax, maximum heart rate; HRR, heart rate reserve; BMI, body mass index; ID, intellectual disability; IBS, irritable bowel syndrome; HFrEF, heart failure with reduced ejection fraction; BPPV, benign paroxysmal positional vertigo; CRP, canalith repositioning procedure; HIIT, high-intensity interval training; CET, continuous endurance training; BW, body weight; OW/OB, overweight/obese; EV, extracellular vesicle; PA, physical activity.

Meta-analysis findings

Effect of physical activity on MDA levels

A pooled analysis of 10 eligible studies was performed using a random effects model to estimate the overall effect of physical activity on MDA levels. The results of this analysis were presented in a forest plot (Fig. 2). The findings showed that physical activity led to a statistically significant, moderate to large reduction in MDA levels compared to control groups. The pooled effect size, calculated as the SMD, was –1.02 (95% CI: –1.95 to –0.09). The p value for this effect was 0.032, indicating statistical significance at the 5% error level. In other words, the mean MDA level in the physical activity intervention groups was approximately 1.02 standard deviations lower than that in the control groups, which was considered both statistically and clinically meaningful.

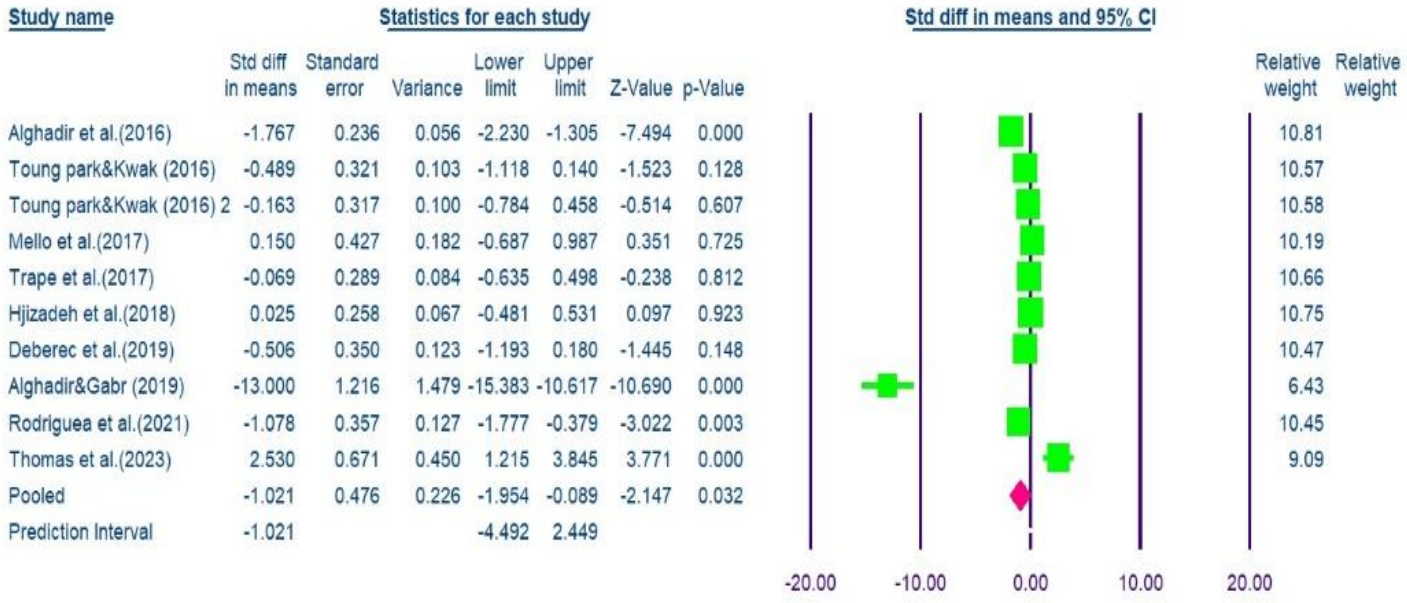


Fig. 2. Forest plots representing the pooled effect of physical activity on malondialdehyde (MDA) levels as a marker of lipid peroxidation.

The assessment of between study heterogeneity using the I^2 statistic revealed very high heterogeneity, with an I^2 value of 94.6% ($p < 0.001$ for the heterogeneity test). Additionally, the Tau^2 value was calculated as 2.039, and the prediction interval ranged from –4.493 to 2.449, indicating a wide range of possible effect sizes in future studies (Fig. 3).

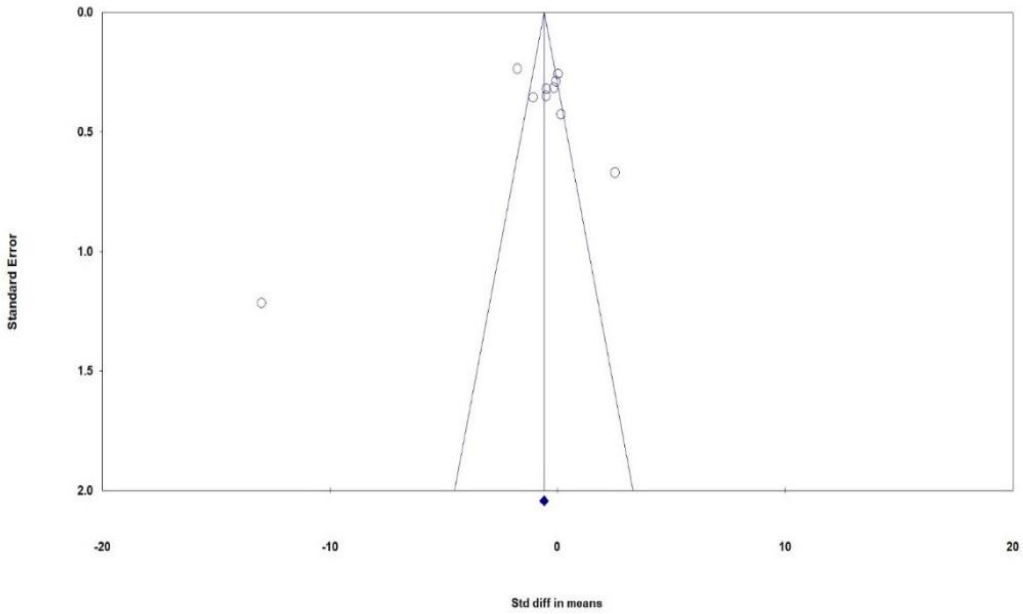


Fig. 3. Funnel plot assessing publication bias among studies evaluating the effect of the intervention on MDA levels.

Effect of physical activity on TAS

A pooled analysis of 4 eligible studies examining the effect of physical activity on TAS was performed using a random effects model. The results of this analysis were presented in a forest plot (Fig. 4). The findings demonstrated that physical activity induced a highly significant, large magnitude increase in TAS levels compared to control groups. The pooled effect size (SMD) was +0.76 (95% CI: 0.38 to 1.15).

The p value for this effect was $p < 0.001$, indicating statistical significance at the highest level. This finding suggested that physical activity significantly enhanced systemic non enzymatic antioxidant capacity.

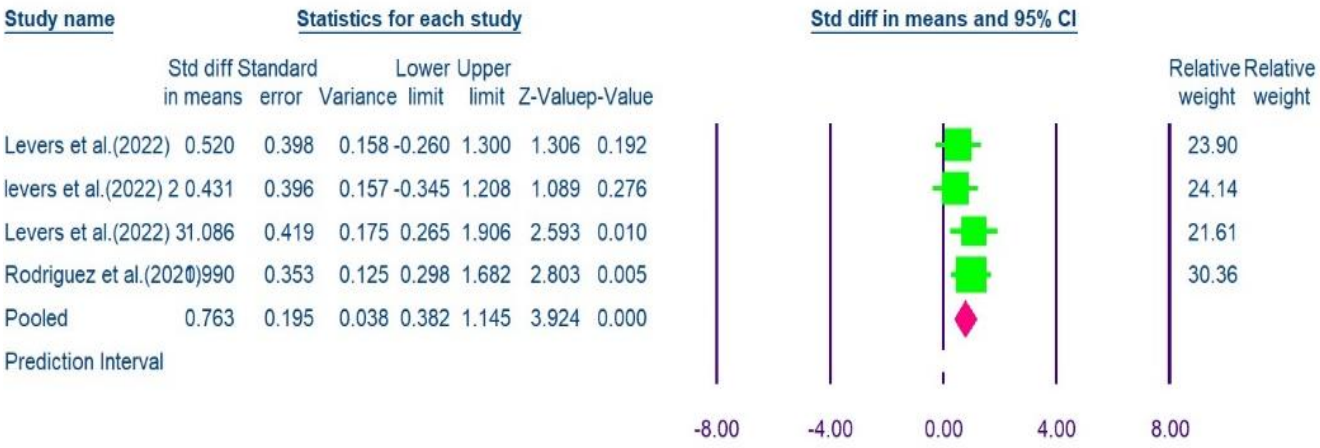


Fig. 4. Forest plots showing the effect of physical activity on total antioxidant status (TAS) in human studies.

The assessment of between study heterogeneity using the I^2 statistic indicated substantial heterogeneity, with an I^2 value of 94.59% ($p < 0.001$ for the heterogeneity test). Despite this high heterogeneity, sensitivity analysis using the leave one out method confirmed that the pooled estimate was appropriately robust, and the removal of any single study did not substantially change the results (Fig. 5).

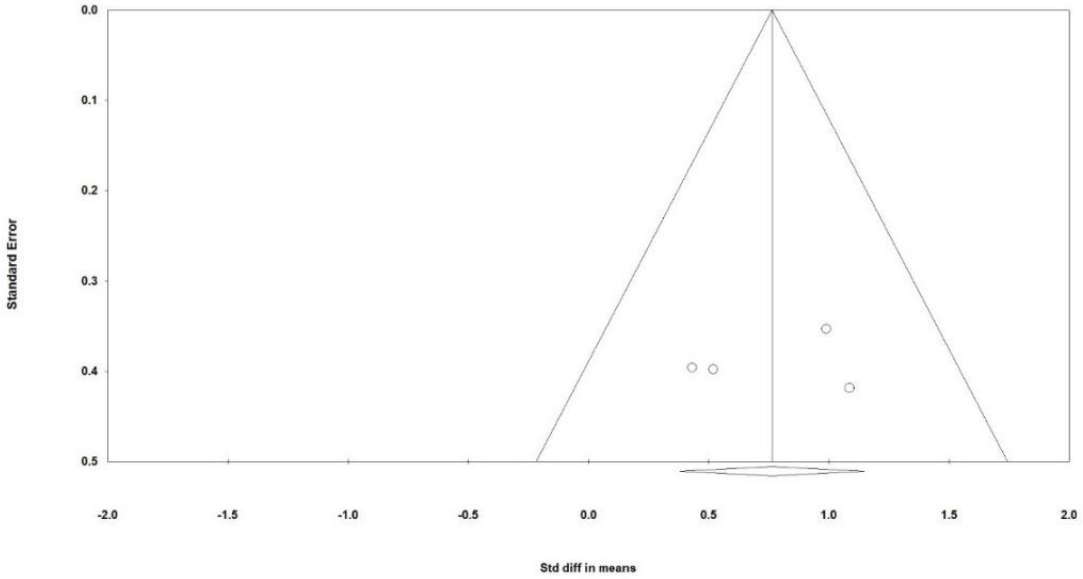


Fig. 5. Funnel plot assessing publication bias among studies included in the meta analysis evaluating the effect of the intervention on TAS levels. Symmetry around the pooled effect suggests a low risk of publication bias.

Subgroup analysis findings for MDA

To identify potential sources of heterogeneity and to examine the role of moderator variables on the effect of physical activity on MDA levels, subgroup analyses were performed based on two key variables: BMI and age.

Regarding BMI, studies were divided into two subgroups: BMI ≤ 25 (4 studies) and BMI > 25 (4 studies). In the subgroup with BMI ≤ 25, the effect size was unexpectedly positive (SMD = +5.08; 95% CI: 0.49 to 9.67; p = 0.03), indicating an increase in MDA levels in this group. This finding contradicted the overall direction of the pooled effect and was likely driven by very high heterogeneity within this subgroup (I^2 = 99.28%). In contrast, in the subgroup with BMI > 25, the effect size was SMD = –0.004 (95% CI: –0.04 to 0.03; p = 0.83), indicating a very

small, non significant reduction in MDA levels among overweight or obese individuals.

Regarding age, studies were classified into two subgroups: age ≤ 50 years (7 studies) and age > 50 years (3 studies). In the subgroup aged ≤ 50 years, the effect size was SMD = –1.39 (95% CI: –2.84 to 0.05; p = 0.06), indicating a reduction that approached but did not reach conventional statistical significance (borderline significant). In contrast, in the subgroup aged > 50 years, the effect size was SMD = –0.61 (95% CI: –1.80 to 0.58; p = 0.31), indicating a non significant reduction in MDA levels among older individuals. Overall, the subgroup analysis results suggested that the MDA lowering effect of physical activity was more pronounced in younger individuals (under 50 years), whereas the moderating role of BMI required further investigation with more homogeneous studies.

Meta regression of age as a moderator of MDA responses to physical activity

The meta regression analysis showed no statistically significant linear relationship between age and the standardized mean difference in MDA (p = 0.48), indicating that age did not substantially contribute to the observed heterogeneity across studies. The intercept was –0.33, but its biological interpretation is limited due to the non significant slope. Collectively, these findings suggest that age does not act as a linear moderator of MDA responses in this dataset. Biologically, this implies that MDA levels are more likely influenced by factors such as metabolic state, oxidative burden, study level differences, or nonlinear regulatory processes rather than by a consistent age related trend (Fig. 6).

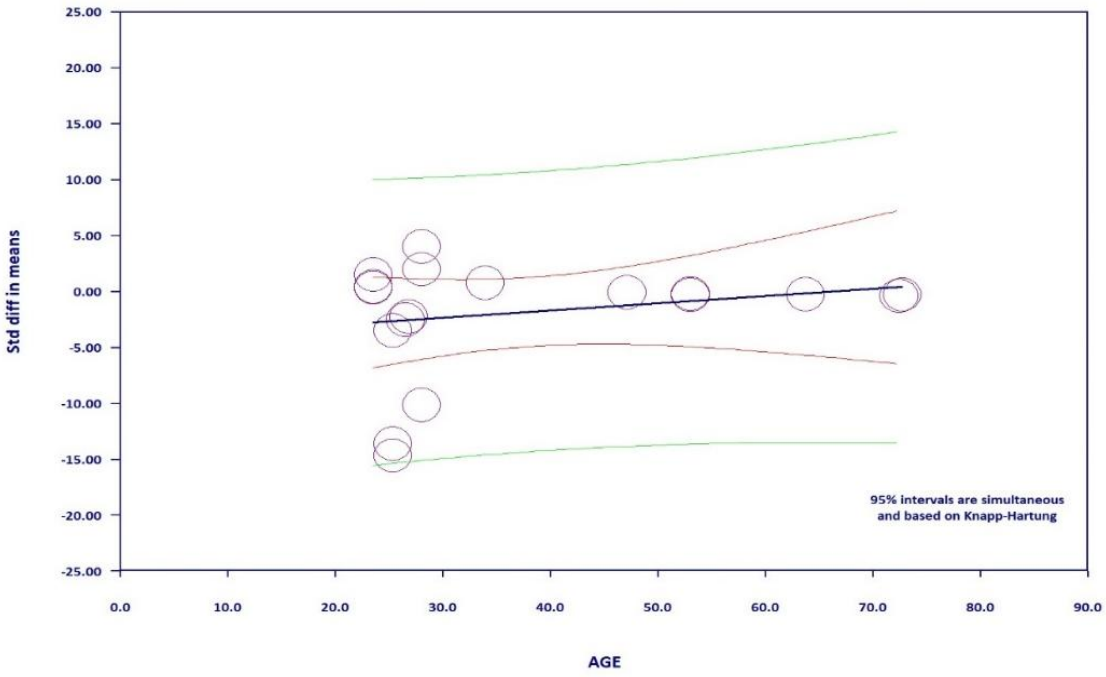


Fig. 6. Meta regression evaluating the effect of age on mean differences in MDA levels. The model revealed no statistically significant linear trend (p = 0.48), and the near zero slope indicates that age did not meaningfully influence MDA effect size estimates. The wide confidence bounds further confirm the lack of age dependent modulation.

Sensitivity analysis and publication bias findings

Sensitivity analysis using the leave one out method was performed to assess the influence of each individual study on the pooled estimates (Figs. 7-8). The results of this analysis confirmed that the pooled estimates for both MDA and TAS were appropriately robust, as the stepwise removal of any single study did not significantly alter the pooled effect size or CI. To evaluate potential publication bias, funnel plots were used together with Egger's regression test. The Egger's test results for MDA showed no clear evidence of publication bias (p = 0.53), and similarly, no publication bias was confirmed for TAS (p =

0.49). The corresponding funnel plots were presented in Figures S2d and S2e. Additionally, Rosenthal's fail safe N was calculated to estimate the number of missing studies needed to render the pooled results non significant.

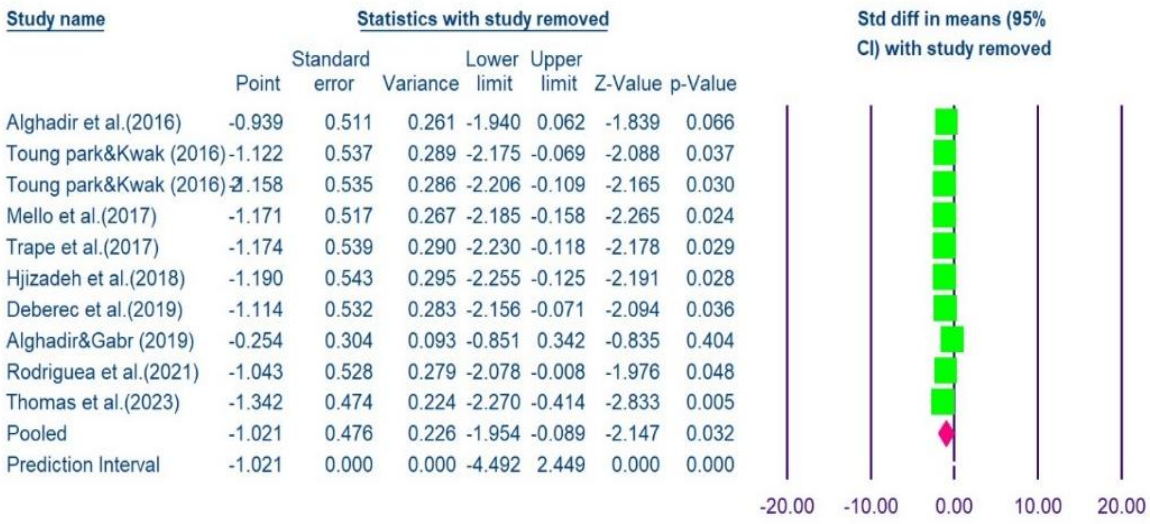


Fig. 7. Forest plot of the sensitivity analysis examining the robustness of the pooled effect of the intervention on MDA levels. The analysis evaluates the influence of individual studies on the overall effect estimate. Squares represent study specific effect estimates with 95% confidence intervals, and the diamond indicates the pooled effect.

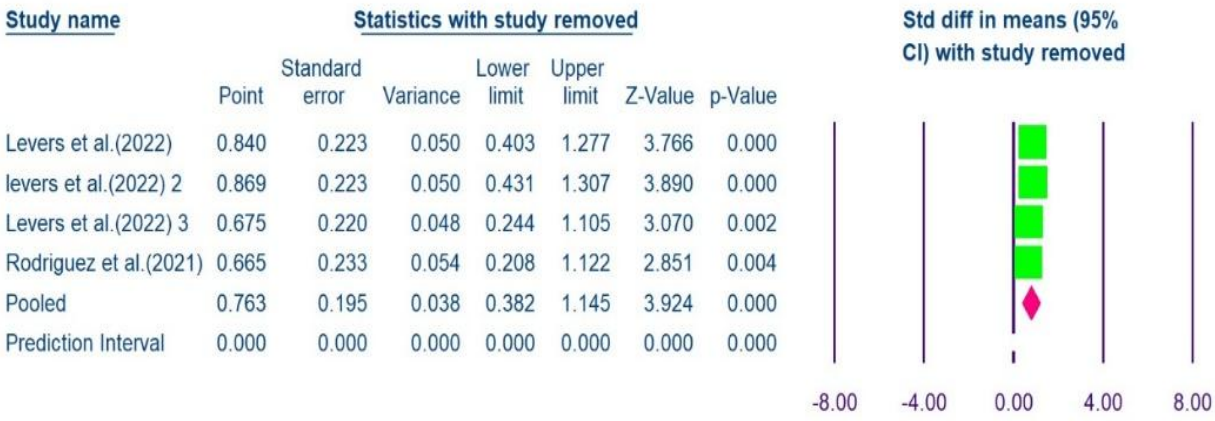


Fig. 8. Forest plot of the sensitivity analysis evaluating the robustness of the pooled effect of the intervention on TAS levels. Squares indicate individual study effects with 95% confidence intervals, and the diamond shows the pooled effect**.**

Discussion

Main findings

The present systematic review and meta analysis, which pooled data from 21 human studies (including 1,235 participants in the MDA and TAS analysis), provided acceptable quantitative evidence that physical activity significantly reduces lipid peroxidation (MDA) and significantly increases total antioxidant capacity (TAS) in humans. These findings were biologically plausible and consistent with the fundamental principles of hormetic adaptation to exercise.

Reduction in MDA: Protection of cell membrane integrity against oxidative damage

The key finding of this study was the moderate to large reduction in MDA levels (SMD = –1.02; 95% CI: –1.95 to –0.09; p = 0.032) in response to physical activity. MDA, widely regarded as a gold standard indicator of lipid peroxidation, is the end product of oxidative

degradation of polyunsaturated fatty acids in cell membranes. Elevated MDA levels have been linked to the pathogenesis of atherosclerosis (through oxidation of low density lipoproteins), systemic inflammation (via activation of NF-κB signaling pathways), and ultimately apoptosis and cell death. The significant reduction in MDA following physical activity observed in this meta analysis suggested that regular exercise effectively protected membrane lipid integrity against free radical attack, even in the absence of major changes in classic antioxidant enzymes such as superoxide dismutase (SOD) and catalase (CAT).

This finding was fully consistent with results from prominent primary studies. (Alghadir et al., 2016) reported a significant decrease in MDA following 24 weeks of moderate aerobic training in healthy older adults. Similarly, (Hajizadeh Maleki et al., 2018) observed a 20% reduction in MDA in sedentary women with irritable bowel syndrome after 8 weeks of supervised aerobic training. (González Garrido et al., 2017) demonstrated a 32.4% reduction in MDA in young professional soccer players following 7 days of cocoa supplementation combined with high intensity training. Furthermore, (Rosety-Rodriguez et al., 2021) showed decreased MDA levels in sedentary men with Down syndrome after 12 weeks of circuit resistance training. (Tsai et al., 2016) also reported reduced MDA following canalith repositioning procedure in patients with benign paroxysmal positional vertigo, suggesting that even non traditional physical maneuvers can positively modulate oxidative stress. Additionally, (Rosa et al., 2019) found lower MDA levels in middle aged men with long term team sport training compared to sedentary controls, and (Cho et al., 2020) demonstrated that MDA responses to exhaustive exercise were modulated by aerobic fitness and genetic factors (CYBA C242T genotype).

Mechanistically, the significant reduction in MDA following physical activity is mediated through the interaction of several cellular and molecular pathways:

1. Activation of the Nrf2/ARE Signaling Pathway: Regular exercise, through mild and transient increases in reactive oxygen species (ROS), activates the transcription factor Nrf2 (Nuclear factor erythroid 2-

related factor 2). Upon activation, Nrf2 translocates to the nucleus and binds to Antioxidant Response Elements (ARE), upregulating the expression of phase II detoxifying enzymes such as heme oxygenase-1 (HO-1), catalase (CAT), and superoxide dismutase (SOD). These enzymes directly neutralize free radicals and inhibit the propagation of lipid peroxidation chain reactions, thereby reducing MDA formation. This Nrf2-mediated adaptive response has been established as a primary mechanism through which endurance exercise training increases the abundance of key antioxidant enzymes in skeletal muscles (Alghadir et al., 2016).

2. Improved Mitochondrial Efficiency: Exercise-induced adaptations, including increased mitochondrial biogenesis and enhanced electron transport chain efficiency, result in reduced electron leakage and consequently lower basal ROS production. This decrease in mitochondrial ROS generation reduces the primary substrate available for initiating lipid peroxidation of polyunsaturated fatty acids in cell membranes. Studies have demonstrated that chronic exercise modulates redox homeostasis in various tissues, improving mitochondrial function and reducing the ROS/ATP ratio.

3. Downregulation of ROS-Generating Enzymes: Chronic exercise can downregulate the expression of pro-oxidant enzymes such as NADPH oxidase (Nox2), a major source of superoxide production in endothelial and immune cells. Research has shown that exercise training decreases NADPH oxidase activity and expression in vascular tissues while increasing antioxidant enzyme expression, thereby reducing the overall oxidative burden on cellular membranes (Cho et al., 2020).

4. Enhanced Non-Enzymatic Antioxidant Capacity: As discussed in the following section (4.3), exercise increases systemic levels of non-enzymatic antioxidants such as uric acid, albumin, and bilirubin, which collectively contribute to the scavenging of lipid peroxyl radicals and prevention of MDA formation (Tsai et al., 2016).

Despite these positive adaptations, it is crucial to emphasize that the oxidative stress response to exercise is not a linear or uniform phenomenon and is highly dependent on timing, intensity, and tissue type. For example, a single session of acute high-intensity exercise can transiently increase MDA levels (Georgescu et al., 2017; Jamurtas et al., 2018). However, this transient increase serves as a hormetic signal that triggers long-term adaptive responses, including the upregulation of antioxidant defenses. The hormesis concept provides an interpretative framework: low or transient increases in ROS from moderate-intensity and regular exercise activate signaling pathways that induce adaptive and protective responses, whereas inactivity or sporadic strenuous exercise can lead to a ROS load that overwhelms antioxidant defenses.

Therefore, the "net" MDA level measured in peripheral blood represents a dynamic balance between ROS production in working muscles, liver, and blood cells on one hand, and the plasma antioxidant buffering capacity on the other (Lisi et al., 2023). This balance is influenced by factors such as exercise modality, fitness level, nutritional status, and the timing of blood sampling relative to the last exercise session, which collectively explain the substantial heterogeneity observed in our meta-analysis.

Furthermore, tissue-specific responses must be considered, as skeletal muscle, cardiac tissue, hepatic tissue, and adipose tissue may exhibit different redox dynamics in response to the same exercise stimulus. Research has demonstrated that responses to oxidative stress induced by exercise differ markedly depending on the organ tissue type and its endogenous antioxidant levels. Circulating biomarkers may not fully capture these tissue-specific adaptations, and compartmentalized redox signaling within distinct cellular organelles further complicates the interpretation of systemic measurements.

Increase in TAS: enhancement of systemic non enzymatic antioxidant buffering capacity

The second major finding of this study was the highly significant, large magnitude increase in TAS (SMD = +0.76; 95% CI: 0.38 to 1.15; $p < 0.001$). TAS is a cumulative indicator reflecting the total capacity of a biological sample (blood, plasma, or serum) to neutralize free radicals, and it is primarily determined by water-soluble non-enzymatic antioxidants such as uric acid, albumin, bilirubin, ascorbic acid (vitamin C), and tocopherols (vitamin E). The increase in TAS following physical activity suggested that exercise induced a systemic adaptation toward enhanced antioxidant buffering capacity.

This finding was supported by multiple studies in our review. (Alghadir et al., 2016) reported a significant increase in TAC (total antioxidant capacity) following moderate aerobic training in healthy older adults. (Hajizadeh Maleki et al., 2018) observed a 15% increase in TAC in women with irritable bowel syndrome after 8 weeks of aerobic exercise. (González-Garrido et al., 2017) demonstrated a 16% increase in TAC following cocoa supplementation combined with training in young soccer players. (Sielski et al., 2018) found that TAS increased significantly after anti-gravity treadmill running at 70% body weight in young female athletes. (Rosety-Rodríguez et al., 2021) reported increased TAS following circuit resistance training in men with Down syndrome. (Almeida et al., 2021) showed that higher physical activity levels were associated with increased TAS in overweight/obese prepubertal children. (Póvoas et al., 2022) demonstrated that higher-rank elite female football teams had higher baseline TAS and uric acid levels. (Lisi et al., 2023) found that extracellular vesicle TAS improved with short-term aerobic training and was higher in trained compared to untrained young males.

Mechanistically, the increase in TAS was likely explained by several pathways: (1) increased uric acid production resulting from purine breakdown during intense physical activity; (2) elevated albumin levels, which act as effective antioxidants with metal-binding capabilities; (3) increased expression and activity of hepatic antioxidant

enzymes leading to enhanced production of antioxidant compounds; and (4) reduced consumption of non-enzymatic antioxidants due to improved efficiency of the cellular defense system. This finding carried clinical significance, as individuals with higher baseline TAS have demonstrated greater resistance to acute oxidative stressors and a lower risk of chronic diseases associated with oxidative stress.

Comparison with evidence from previous studies

The findings of this study regarding reduced MDA and increased TAS were consistent with many prominent human studies but also showed interesting differences from animal studies in terms of the antioxidant response pattern. In human studies, multiple investigations have supported the idea that the protective effect of exercise on MDA is a reproducible finding across different populations. (Park & Kwak., 2016) (Shoja Anzabi et al., 2026) demonstrated that chronic aerobic and resistance training blunted acute exercise induced rises in MDA in trained young males. (Georgescu et al., 2017) showed that dehydration during exercise exacerbated MDA increases, whereas adequate hydration prevented this effect, highlighting the importance of fluid intake. (Jamurtas et al., 2018) reported that continuous endurance training caused greater MDA rises than low volume HIIT, suggesting that exercise modality modulates oxidative stress responses. (Lazar Poloczek et al., 2021) demonstrated that MDA levels progressively increased with heart failure severity, while TAS decreased, indicating that oxidative stress markers track disease progression.

However, comparison of our findings with meta analyses conducted on animal models revealed interesting differences. Animal studies typically reported significant increases in antioxidant enzymes such as SOD, CAT, and GPx in response to training, whereas the present study (like previous human meta analyses) found non significant effects of exercise on these enzymes in humans. This cross species difference could be attributed to several factors: (1) differences in baseline oxidative status, laboratory animals typically live in standard, stress free conditions and therefore have greater potential for upregulation in

response to exercise, whereas humans face multiple oxidative stressors and may have antioxidant enzymes operating near maximum capacity (a ceiling effect); (2) differences in training protocols, animal studies often use forced exercise protocols that may induce different stress responses compared to voluntary exercise in humans; (3) differences in sampling timing, the peak activation of the Nrf2 pathway typically occurs 2–24 hours after exercise, whereas many human studies sample at standardized times that may miss the peak enzymatic response. Despite these differences, the main common finding across human and animal studies was the reliable reduction in MDA/TBARS, establishing this biomarker as a dependable indicator of the exercise training response.

Subgroup analysis: moderating roles of age and BMI

Subgroup analyses revealed that the MDA response to physical activity was modulated by age and BMI. Regarding age, the reduction in MDA was more pronounced in younger individuals (under 50 years; SMD = –1.39; p = 0.06) compared to older individuals (over 50 years; SMD = –0.61; p = 0.31), consistent with previous studies (19, 31). This attenuated response in older individuals may be explained by age related declines in cellular repair, Nrf2 pathway activity, and mitochondrial biogenesis. Clinically, although exercise is beneficial at all ages, older individuals may require higher doses or longer training to achieve the same protective effect.

Regarding BMI, the results were complex. In overweight/obese individuals (BMI > 25), MDA reduction was very small and non significant (SMD = –0.004; p = 0.83), supported by Almeida et al. (2021) (35). This may be due to high heterogeneity (I^2 = 89.54%), insufficient exercise intensity or duration, or chronic inflammation modulating the response. In contrast, the normal weight subgroup (BMI ≤ 25) showed a paradoxical increase in MDA (SMD = +5.08; p = 0.03) with extremely high heterogeneity (I^2 = 99.28%), likely a statistical artifact from an outlier study in a small subgroup (only 4 studies). Thus, definitive conclusions about BMI require further standardized studies.

Heterogeneity and publication bias: challenges and strengths

One of the main challenges of this study was the very high heterogeneity between the included studies (I^2 = 94.6% for both MDA and TAS). Although this level of heterogeneity appeared high at first glance, it was entirely expected in meta analyses within the field of exercise and oxidative stress. (Becatti et al., 2017) demonstrated that a full competitive soccer season increased oxidative damage without changing TAS, while (Póvoas et al., 2022) showed that baseline TAS differed between higher rank and lower rank teams during a tournament. (Puga et al., 2016) showed that L arginine supplementation blunted the exercise induced decline in MDA, while (Mooren et al. 2020) demonstrated that probiotic supplementation partially preserved TAC following exhaustive exercise. These findings highlight the variability in responses due to supplementation, population, and exercise modality.

The main sources of this heterogeneity included: (1) variability in exercise protocols, acute vs. chronic, aerobic vs. resistance, low vs. high intensity, and durations ranging from 2 to 52 weeks; (2) variability in study populations from young elite athletes to middle aged obese men, older adults, and clinical populations such as heart failure patients and fibromyalgia; (3) variability in biomarker measurement methods, different assays for MDA and TAS have different sensitivities and specificities; and (4) variability in sampling timing, sampling at different times after the last exercise session could produce different results.

Despite this high heterogeneity, several findings indicated that our main results were appropriately robust. First, sensitivity analysis (leave one out) confirmed that removing any single study did not significantly alter the pooled estimate. Second, the fail safe N for the MDA effect showed that at least 28 null studies would be required to negate the current significant finding, far exceeding the number of included studies (10 studies), indicating high robustness against publication bias. Third, statistical tests for publication bias (Egger's test) were non significant

for both MDA and TAS ($p = 0.53$ and $p = 0.49$, respectively), indicating no clear evidence of publication bias.

Justification for combining acute and chronic Exercise studies

One of the primary methodological considerations of this meta-analysis was the combination of studies with acute (single session) and chronic (≥2 weeks) exercise interventions in the overall analysis. This decision was made deliberately and is justified by three main arguments:

First, shared physiological basis: Both acute and chronic exercise affect the "oxidative stress–antioxidant response" axis, albeit through different mechanisms. Acute exercise generates a transient pulse of ROS production, which serves as a signaling trigger for adaptive pathways (hormesis), while chronic exercise represents the cumulative effect of repeated pulses, leading to sustained adaptations in antioxidant capacity. However, both types can produce measurable and significant changes in MDA and TAS levels (Lisi et al., 2023). The primary aim of this meta-analysis was to evaluate the overall effect of physical activity on these biomarkers, not to distinguish between immediate and long-term effects. This approach provides a global estimate that reflects the totality of evidence, which is valuable for public health messaging and clinical decision-making.

Second, complementary nature: In clinical practice and real-world settings, physical activity is inherently a dual-faceted phenomenon. Each exercise session represents an "acute event" embedded within a "chronic program." Therefore, combining these two study types provides a more realistic and holistic picture of the total impact of physical activity on the biological system, as individuals in the real world experience both the immediate effects of individual sessions and the cumulative effects of regular participation (Tsai et al., 2016).

Third, statistical constraints: Conducting separate meta-analyses for acute and chronic studies would have severely reduced the number of studies in each subgroup, thereby substantially weakening the statistical

power of the analyses and increasing the risk of type II errors. Importantly, our sensitivity analysis (leave-one-out method) confirmed that removing acute studies did not alter the direction or statistical significance of the overall effect, indicating that the pooled estimate is robust to the inclusion of these studies.

However, to maintain appropriate scientific caution, we note the following: (1) We have acknowledged the potential contribution of combining acute and chronic studies to the observed high heterogeneity in Section 4.7 (Limitations). (2) The differing nature of these two intervention types underscores the need for future studies with standardized, randomized designs to separately examine acute versus chronic responses. (3) We explicitly advise that findings related to acute exercise effects should not be equated with chronic adaptations, and this distinction must be considered in clinical applications. For instance, a single bout of high-intensity exercise may transiently increase oxidative stress markers, whereas chronic training consistently lowers them. These contrasting effects should guide clinical decision-making regarding exercise prescription in different patient populations.

Study limitations

Despite several strengths including a comprehensive systematic search, a PROSPERO registered protocol, use of a random effects model with Hartung Knapp adjustment, and comprehensive sensitivity analyses, this study had several limitations that should be considered when interpreting the findings. First, the very high heterogeneity ($I^2 \approx 95\%$) remained largely unexplained despite our subgroup analyses, which cautions against definitive conclusions regarding causality and generalizability. Second, the small sample sizes of most included studies (11 to 40 participants per study) led to wide prediction intervals and reduced statistical power for subgroup analyses. Third, the limited number of studies in the TAS analysis (only 4 studies) restricted the generalizability of this specific finding. Fourth, the English language restriction may have led to the exclusion of studies published in other

languages, potentially introducing publication bias. Fifth, reliance on blood biomarkers may not accurately reflect redox status in target tissues such as skeletal muscle, liver, or heart. Sixth, there was no control for confounding variables such as nutritional status, antioxidant supplement use, medications, or genetic polymorphisms affecting redox metabolism. Seventh, blinding was not possible in exercise based interventions, which resulted in a high risk of bias in the outcome measurement domain for 56% of the studies.

Clinical applications and practical implications

Despite the limitations mentioned above, the findings of this study had important clinical applications. First, confirmation of the protective effect of exercise on cell membranes: the significant reduction in MDA indicated that regular physical activity could serve as an effective non pharmacological strategy for reducing the risk of atherosclerosis, inflammation, and cardiovascular diseases. This finding provided a molecular mechanism for part of the cardiovascular protective effects of exercise demonstrated in large epidemiological studies. Second, introduction of TAS as a useful biomarker for monitoring the response to training: increases in TAS could serve as an objective marker for evaluating the effectiveness of training programs and an individual's adaptation to exercise load. Sports coaches and clinical physiologists could use TAS measurements before and after training periods to optimize exercise dose and identify poor responders. Third, evidence based exercise recommendations: based on the subgroup analysis findings, the protective effects of exercise on reducing MDA appeared to be more pronounced in younger individuals (under 50 years). This meant that although starting exercise at any age was beneficial, the maximum benefit in reducing oxidative damage was achieved at younger ages. Therefore, emphasizing physical activity from childhood and adolescence is essential for maximum lifetime protection against oxidative stress**.**

Conclusion

This meta-analysis of 21 human studies (1,235 participants) provided acceptable quantitative evidence that physical activity effectively reduces systemic oxidative stress through two complementary pathways: a significant reduction in lipid peroxidation (MDA: SMD = –1.02; 95% CI: –1.95 to –0.09; $p = 0.032$) and a highly significant increase in total antioxidant capacity (TAS: SMD = +0.76; 95% CI: 0.38 to 1.15; $p < 0.001$). These findings support the hormetic adaptation model, in which mild exercise-induced oxidative stress triggers adaptive responses that strengthen endogenous antioxidant defenses.

From a clinical translation perspective, these findings support the integration of regular, structured physical activity into preventive and therapeutic strategies for conditions associated with oxidative stress. The observed changes in MDA and TAS provide a mechanistically plausible explanation for the cardiovascular and metabolic benefits of exercise documented in large epidemiological studies. The potential practical implications of our findings include:

Biomarker Monitoring: Changes in MDA and TAS may serve as objective markers for evaluating the effectiveness of training programs and monitoring individual adaptation to exercise load. Sports coaches and clinical physiologists could consider using these biomarkers to optimize exercise prescription and identify individuals who may be poor responders to training.

Age Considerations: Our subgroup analysis revealed that the oxidative stress-lowering effects of exercise may be more pronounced in younger individuals (under 50 years). This finding, while preliminary, suggests that emphasizing physical activity from childhood and adolescence may be particularly important for maximizing lifetime protection against oxidative damage, although this hypothesis requires confirmation in future studies.

Clinical Trial Design: The observed effect sizes provide a strong hypothesis-generating basis for designing future randomized controlled trials with hard clinical endpoints (e.g., cardiovascular events, mortality, hospitalization rates). Such trials are necessary to definitively

establish whether exercise-induced redox modulation translates into clinically meaningful improvements in patient outcomes.

It must be emphasized that improvements in surrogate biomarkers (MDA and TAS) do not automatically translate into hard clinical outcomes such as reduced cardiovascular mortality, decreased hospitalization rates, or increased lifespan. Our findings should be interpreted as providing mechanistic insights and hypothesis-generating evidence rather than definitive proof of clinical efficacy. The clinical implications outlined above represent potential applications that require validation through well-designed prospective studies with long-term follow-up and clinically meaningful endpoints.

Future high-quality randomized controlled trials with standardized protocols, harmonized biomarker measurements, and mechanistic studies using tissue biopsies and key signaling pathways (Nrf2, PGC-1α, NF-κB) are needed to reduce heterogeneity and better understand the underlying molecular mechanisms. Additionally, future research should investigate the direct relationship between exercise-induced changes in oxidative biomarkers and hard clinical outcomes to establish the clinical utility of these biomarkers as therapeutic targets.

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The author declares no conflict of interest.

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