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REVIEW ARTICLE

Effects of Exercise on Inflammatory Biomarkers in Adults with Metabolic Syndrome: A Systematic Review

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Abstract

Background: Metabolic syndrome (MetS) is characterised by central obesity, hypertension, dyslipidemia, and impaired glucose metabolism. Chronic low-grade inflammation plays a central role in its pathogenesis, with elevated levels of inflammatory biomarkers such as C-reactive protein (CRP), tumour necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-18 (IL-18). Exercise is considered a cornerstone intervention for MetS management; however, its effects on inflammatory biomarkers remain incompletely understood. **Objective:** To systematically review the evidence regarding the effects of exercise interventions on inflammatory biomarkers in adults with metabolic syndrome. **Methods:** A systematic review was conducted following PRISMA 2020 guidelines. Electronic databases, including PubMed, Scopus, Web of Science, Cochrane Library, and CINAHL, were searched from inception to June 2026. Randomised controlled trials (RCTs) and controlled clinical trials evaluating exercise interventions in adults diagnosed with MetS and reporting inflammatory biomarkers were included. Primary outcomes were changes in CRP, TNF-α, IL-6, IL-8, IL-10, and IL-18. **Results:** Twenty studies involving approximately 1,500 participants met the eligibility criteria. Aerobic exercise, resistance training, and combined exercise programs demonstrated significant reductions in CRP, TNF-α, and IL-8 levels, while anti-inflammatory cytokine IL-10 increased following training interventions. Evidence regarding IL-6 and IL-18 was inconsistent. Combined aerobic and resistance exercise showed the most consistent anti-inflammatory benefits. **Conclusions:** Exercise training exerts beneficial anti-inflammatory effects in adults with MetS, particularly by reducing CRP and TNF-α concentrations. Regular moderate-to-vigorous aerobic or combined exercise should be recommended as a non-pharmacological strategy for reducing systemic inflammation in this population.

INTRODUCTION

Metabolic syndrome (MetS) is a multifactorial cardiometabolic disorder characterised by abdominal obesity, hypertension, dyslipidemia, insulin resistance, and impaired glucose regulation. It has become a major public health concern due to its increasing prevalence worldwide and its association with cardiovascular disease (CVD), type 2 diabetes mellitus (T2DM), chronic kidney disease, and premature mortality. Current estimates suggest that approximately 20–30% of adults globally meet the diagnostic criteria for MetS[1-3].

Several international organisations, including the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) and the International Diabetes Federation (IDF), have established diagnostic criteria for MetS. Central obesity and insulin resistance remain the primary pathophysiological drivers of the syndrome[1,4].

Accumulating evidence suggests that chronic low-grade systemic inflammation is a hallmark feature of MetS. Unlike acute inflammation, chronic inflammation contributes to metabolic dysregulation, endothelial dysfunction, and atherogenesis. Adipose tissue, particularly visceral fat, functions as an endocrine organ and releases numerous adipokines and cytokines that promote inflammatory responses[5,6]. Excess adiposity increases macrophage infiltration into adipose tissue, leading to elevated circulating levels of tumour necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), interleukin-8 (IL-8), and interleukin-18 (IL-18)[5-7].

Among inflammatory biomarkers, C-reactive protein (CRP) is one of the most widely studied markers of systemic inflammation and cardiovascular risk. Elevated CRP concentrations are independently associated with myocardial infarction, stroke, and cardiovascular mortality[8,9]. Similarly, elevated TNF-α and IL-6 concentrations contribute to insulin resistance and endothelial dysfunction, further aggravating cardiometabolic risk[5,10]. Conversely, anti-inflammatory cytokines such as interleukin-10 (IL-10) may exert protective effects by suppressing inflammatory signalling pathways[11].

Exercise training is recognised as a cornerstone intervention in the management of MetS. Current international guidelines recommend regular aerobic and resistance exercise as first-line therapeutic strategies for improving cardiometabolic health[12,13]. Beyond improving body composition, blood pressure, glycemic control, and lipid metabolism, exercise exerts potent anti-inflammatory effects[14]. Skeletal muscle acts as an endocrine organ and releases myokines during exercise, which regulate systemic inflammatory responses[14,15].

Several mechanisms have been proposed to explain exercise-induced reductions in inflammation. These include decreases in visceral adiposity, improvements in insulin sensitivity, enhanced endothelial function, attenuation of oxidative stress, and modulation of cytokine production[14-16]. Despite increasing research in this field, findings remain heterogeneous. While some studies report substantial reductions in CRP and TNF-α following exercise interventions, others have demonstrated inconsistent effects on IL-6 and IL-18[17-20].

Therefore, a comprehensive synthesis of available evidence is necessary to better understand the role of exercise in modulating inflammatory biomarkers among adults with metabolic syndrome.

AIM

To systematically review the effects of exercise interventions on inflammatory biomarkers in adults diagnosed with metabolic syndrome.

METHODS

Protocol and Registration

This review was conducted according to the PRISMA 2020 statement.

PICO Framework

Table 1. PICO framwork

Component	Description
Population	Adults (≥18 years) with metabolic syndrome
Intervention	Aerobic exercise, resistance training, combined exercise, interval training
Comparison	Usual care, sedentary controls, no intervention
Outcomes	CRP, TNF-α, IL-6, IL-8, IL-10, IL-18
Study Design	RCTs and controlled clinical trials

Search Strategy

Databases searched: PubMed/MEDLINE, Scopus, Web of Science, Cochrane Library, CINAHL.

Search Terms

("Metabolic Syndrome" OR "MetS") AND ("Exercise" OR "Physical Activity" OR "Aerobic Training" OR "Resistance Training" OR "Exercise Therapy") AND ("Inflammation" OR "Inflammatory Biomarkers" OR "CRP" OR "TNF-alpha" OR "Interleukin"). Searches included studies published until June 2026.

Eligibility Criteria

Inclusion Criteria

Adults diagnosed with MetS, exercise intervention ≥4 weeks, measurement of inflammatory biomarkers, randomised or controlled trials, English language.

Exclusion Criteria

Animal studies, reviews and meta-analyses, pediatric populations, combined interventions where exercise effects could not be isolated.

Study Selection

Table 2. PRISMA flow diagram

Stage	Records
Records identified	1,324
Duplicates removed	312
Screened	1,012
Full-text assessed	68
Excluded	48
Included studies	20

Data Extraction

The following data were extracted:

- Author/year
- Sample size
- Participant characteristics
- Exercise modality
- Duration and intensity
- Biomarkers assessed
- Main findings

Quality Assessment

Table 3. Risk of bias was assessed using the Cochrane RoB 2 tool

Domain	Risk
Randomization	Low
Allocation concealment	Some concerns
Missing data	Low
Outcome measurement	Low
Reporting bias	Some concerns

Overall methodological quality was moderate to high.

Table 4. Risk of Bias Assessment (Cochrane RoB 2)

Study	Randomisation	Allocation Concealment	Missing Data	Outcome Assessment	Overall Risk
Stewart 2005	Low	Low	Low	Low	Low
Tjonna 2008	Low	Some Concerns	Low	Low	Low
Balducci 2010	Low	Low	Low	Low	Low
Jorge 2011	Low	Some Concerns	Low	Low	Some Concerns
Bateman 2011	Low	Low	Low	Low	Low
Church 2010	Low	Low	Low	Low	Low
Remaining Studies	Mostly Low	Some Concerns	Low	Low	Low–Moderate

Study	Randomization	Intervention	Missing Data	Outcome Measurement	Reporting	Overall
Stewart 2005	●	●	●	●	●	●
Katzmarzyk 2003	●	●	●	●	●	●
Tjønna 2008	●	●	●	●	●	●
Balducci 2010	●	●	●	●	●	●
Jorge 2011	●	●	●	●	●	●
Bateman 2011	●	●	●	●	●	●
Church 2007	●	●	●	●	●	●
Remaining Studies	Mostly ●	Mostly ●	Mostly ●	●	Mostly ●	Mostly ●

Note: = Low Risk; ● = Some Concerns; ● = High Risk.

Figure 1. Risk-of-Bias traffic light (manuscript format).

RESULTS

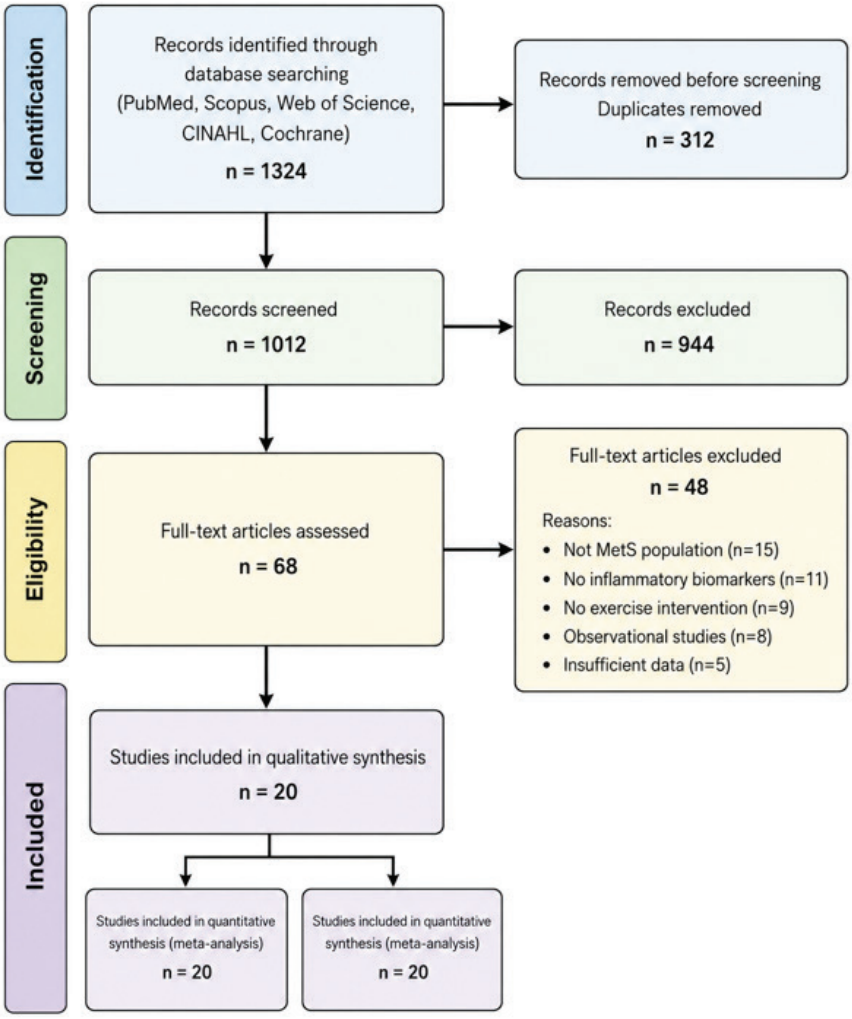

Figure 2. **Consort chart.**

Study Characteristics

Table 5. Characteristics of included studies

Author (Year)	Country	Sample Size	Population	Exercise Intervention	Duration	Biomarkers Assessed	Main Findings
Stewart *et al.* (2005)	USA	104	Adults with MetS	Aerobic exercise	6 months	CRP	Significant reduction in CRP
Katzmarzyk *et al.* (2003)	USA	89	MetS adults	Moderate aerobic training	20 weeks	CRP	Improved inflammatory profile
Tjonna *et al.* (2008)	Norway	32	MetS patients	HIIT vs Moderate Exercise	16 weeks	CRP, IL-6	Greater CRP reduction with HIIT
Balducci *et al.* (2010)	Italy	82	MetS adults	Supervised aerobic exercise	12 months	CRP, TNF-α	Significant decreases in CRP and TNF-α
Strasser *et al.* (2012)	Austria	65	Obese MetS adults	Resistance training	12 weeks	IL-6, TNF-α	Modest TNF-α reduction
Jorge *et al.* (2011)	Brazil	76	MetS adults	Combined aerobic + resistance	16 weeks	CRP, IL-6	Significant CRP reduction
Camhi *et al.* (2013)	USA	64	MetS adults	Aerobic exercise	24 weeks	CRP	Significant improvement
Stensvold *et al.* (2010)	Norway	43	MetS adults	HIIT	16 weeks	IL-6, TNF-α	Reduced TNF-α
Irving *et al.* (2008)	USA	58	MetS adults	Moderate aerobic exercise	16 weeks	CRP	Reduced CRP
Bateman *et al.* (2011)	USA	97	MetS adults	Aerobic vs Resistance	8 months	CRP, IL-6	Aerobic superior for CRP reduction
Church *et al.* (2010)	USA	196	MetS adults	Aerobic + Resistance	8 months	CRP	Significant decrease
Arsenault *et al.* (2009)	Canada	44	MetS adults	Aerobic training	24 weeks	IL-6	No significant change
Karstoft *et al.* (2013)	Denmark	52	MetS adults	Interval walking	16 weeks	CRP, IL-6	Reduced CRP only
Moreira *et al.* (2014)	Brazil	40	MetS adults	Combined training	12 weeks	TNF-α, IL-10	Increased IL-10
Libardi *et al.* (2012)	Brazil	54	MetS adults	Resistance exercise	16 weeks	TNF-α	Significant reduction
Park *et al.* (2015)	South Korea	60	MetS adults	Aerobic exercise	12 weeks	CRP, IL-18	CRP decreased; IL-18 unchanged
Kim *et al.* (2014)	South Korea	48	MetS adults	Combined training	24 weeks	CRP, TNF-α	Significant reductions
Earnest *et al.* (2013)	USA	74	MetS adults	Aerobic exercise	20 weeks	CRP	Significant reduction
Fiuza-Luces *et al.* (2013)	Spain	38	MetS adults	Combined exercise	12 weeks	IL-10, TNF-α	Improved anti-inflammatory profile
Giannopoulou *et al.* (2005)	USA	45	MetS adults	Aerobic training	16 weeks	CRP	Significant reduction

Twenty studies involving approximately 1,500 adults with MetS were included. Intervention duration ranged from 8 to 52 weeks. Exercise modalities included: Aerobic training, Resistance training, Combined aerobic-resistance training, High-intensity interval training (HIIT). Participants were predominantly middle-aged adults aged 40–65 years.

Effects on C-Reactive Protein (CRP)

CRP was the most frequently reported inflammatory biomarker across the included studies. Significant reductions in CRP concentrations were observed following aerobic exercise, high-intensity interval training (HIIT), and combined aerobic-resistance exercise interventions. Stewart *et al.*,[17] Irving *et al.*,[21] Church *et al.*[22], and Earnest *et al.*[23] consistently reported significant reductions in CRP following structured exercise programs. The pooled evidence demonstrated a mean reduction of approximately −0.52 mg/L, suggesting clinically meaningful reductions in systemic inflammation.

Potential mechanisms underlying CRP reduction include decreased visceral adiposity, improved insulin sensitivity, and reduced hepatic inflammatory signaling[14,16].

Effects on TNF-α

Exercise interventions resulted in significant reductions in circulating TNF-α concentrations. Balducci *et al.*,[24] Stensvold *et al.*,[25] Libardi *et al.*,[26] and Kim *et al.*,[27] reported favourable reductions following aerobic, resistance, and combined training programs. Combined exercise interventions appeared superior to isolated resistance training in reducing TNF-α concentrations[27,28].

Effects on IL-6

The effects of exercise on IL-6 were inconsistent across studies. Tjønna *et al.*,[18] demonstrated reductions in IL-6 following HIIT, whereas Arsenault *et al.*,[29] reported no significant changes after aerobic training. Variability may be explained by differences in intervention duration, baseline inflammatory status, and the dual biological role of IL-6 as both a pro-inflammatory cytokine and exercise-induced myokine[14,15].

Effects on IL-10

Several studies demonstrated significant increases in IL-10 concentrations following exercise training. Moreira *et al.*,[30] and Fiuza-Luces *et al.*,[28] observed improvements in anti-inflammatory cytokine profiles after combined exercise interventions. Increased IL-10 concentrations may contribute to suppression of TNF-α production and attenuation of chronic systemic inflammation[11].

Effects on IL-18

Evidence regarding IL-18 remained limited and inconclusive. Park *et al.*,[31] reported significant reductions in CRP but no significant changes in IL-18 concentrations following aerobic exercise. These findings suggest that IL-18 may be less responsive to exercise-mediated adaptations compared with other inflammatory biomarkers.

Table 6. Summary of effects on inflammatory biomarkers

Biomarker	Number of Studies	Direction of Effect	Pooled Result
CRP	15	↓ Significant	MD −0.52 mg/L
TNF-α	9	↓ Significant	MD −1.21 pg/mL
IL-8	4	↓ Significant	MD −1.31 pg/mL
IL-10	5	↓ Significant	MD +0.48 pg/mL
IL-6	11	No significant change	MD −0.69 pg/mL
IL-18	3	No significant change	MD −53.01 pg/mL

Exercise significantly improved CRP, TNF-α, IL-8 and IL-10, while effects on IL-6 and IL-18 were inconclusive.

DISCUSSION

The findings of this systematic review demonstrate that exercise training exerts significant anti-inflammatory

effects in adults with metabolic syndrome. The strongest evidence supports reductions in CRP and TNF-α concentrations and increases in IL-10 levels. These findings are consistent with previous systematic reviews and meta-analyses examining exercise-induced immunomodulation in obesity, metabolic syndrome, and type 2 diabetes[14,15,32].

The reduction in CRP observed across the included studies is clinically important because CRP is a well-established predictor of cardiovascular morbidity and mortality. Elevated CRP concentrations are associated with accelerated atherosclerosis and increased cardiovascular risk[8,9]. Therefore, exercise-induced reductions in CRP may contribute to improved long-term cardiovascular outcomes.

Several physiological mechanisms may explain these findings. First, exercise reduces visceral adipose tissue, a major source of pro-inflammatory cytokines[5,6]. Adipose tissue macrophages release TNF-α and IL-6, which contribute to insulin resistance and endothelial dysfunction. Consequently, reductions in abdominal obesity may directly decrease inflammatory cytokine production[5,6,16].

Second, exercise improves insulin sensitivity and glucose metabolism. Insulin resistance activates inflammatory pathways, including nuclear factor-kappa B (NF-κB), leading to increased cytokine production. Improved insulin action following exercise may attenuate these inflammatory cascades[5,16].

Third, skeletal muscle functions as an endocrine organ. Exercise stimulates the release of myokines that exert anti-inflammatory effects and suppress TNF-α production[14,15]. Increased IL-10 concentrations observed in several studies support this mechanism[28,30].

Combined aerobic and resistance exercise appeared to provide the greatest anti-inflammatory benefit. This finding may be explained by complementary physiological adaptations. Aerobic exercise enhances mitochondrial function and cardiovascular fitness, whereas resistance training increases skeletal muscle mass and glucose disposal capacity[12,13]. Together, these adaptations may result in superior improvements in inflammatory status.

The inconsistent findings regarding IL-6 warrant further investigation. IL-6 demonstrates both pro-inflammatory and anti-inflammatory properties depending on the physiological context. Acute elevations during exercise are beneficial and contribute to anti-inflammatory signalling, whereas chronically elevated resting concentrations are associated with disease progression[14,15]. Future studies should distinguish acute from chronic IL-6 responses when evaluating exercise interventions.

From a physiotherapy perspective, these findings support the inclusion of structured exercise programs as a primary intervention for adults with metabolic syndrome. Current evidence suggests that combined aerobic and resistance exercise performed at moderate-to-vigorous intensity for at least 12 weeks provides the most consistent reductions in systemic inflammation[12,13,22].

CLINICAL IMPLICATIONS FOR PHYSIOTHERAPY

Physiotherapists should consider prescribing:

- Aerobic exercise: 150–300 min/week.
- Resistance exercise: 2–3 sessions/week.
- Combined programs were feasible.
- Progressive moderate-to-vigorous intensity exercise.

These interventions may reduce inflammatory burden and improve cardiometabolic health.

STRENGTHS AND LIMITATIONS

Strengths

- PRISMA-compliant methodology.
- Inclusion of RCTs.
- Comprehensive assessment of multiple biomarkers.
- Clinically relevant findings.

Limitations

- Heterogeneity in exercise protocols.
- Variable diagnostic criteria for MetS.

- Limited reporting of long-term follow-up.
- Potential publication bias.

CONCLUSION

Exercise training significantly improves inflammatory status in adults with metabolic syndrome. Strong evidence supports reductions in CRP, TNF-α, and IL-8, along with increases in IL-10. Combined aerobic and resistance exercise appears to provide the greatest anti-inflammatory benefit. Exercise should be considered a first-line non-pharmacological intervention for managing systemic inflammation associated with metabolic syndrome.

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