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Omega-3 Fatty Acids and Curcumin as Non-Pharmacological Strategies for Modulating Post-Exercise Inflammation in Individuals with Autoimmune Diseases: A Scoping Review

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Abstract

Background. Exercise is recommended in many autoimmune diseases, but acute sessions can transiently increase inflammatory and muscle-damage markers. Omega-3 fatty acids and curcumin have anti-inflammatory and inflammation-resolving properties, yet their effects on post-exercise inflammation in autoimmune populations remain uncertain.

Aim. To map human evidence on omega-3 fatty acids and curcumin as non-pharmacological strategies for modulating exercise-associated inflammation in individuals with autoimmune diseases.

Material and Methods. This scoping review followed JBI guidance and PRISMA-ScR, with search reporting informed by PRISMA-S. No publication-date restriction was applied. Evidence was grouped into direct studies combining autoimmune disease, supplementation and exercise; supplementation studies in autoimmune populations; and exercise studies in autoimmune populations.

Results. Direct evidence was scarce. Omega-3 supplementation showed the strongest disease-specific evidence in rheumatoid arthritis and possible modest benefits in systemic lupus erythematosus, whereas findings in multiple sclerosis were less supportive. In healthy exercising adults, omega-3 may reduce selected cytokines, creatine kinase and soreness. Curcumin studies in autoimmune and immune-mediated diseases suggest potential anti-inflammatory effects, but results vary with formulation, dose and bioavailability. Exercise-recovery studies of curcumin remain heterogeneous and of low certainty.

Conclusion. Omega-3 fatty acids and curcumin are biologically plausible adjuncts for physically active individuals with autoimmune diseases, but current evidence does not establish that either reliably modulates post-exercise inflammation in this population. Dedicated factorial trials combining supplementation and standardized exercise are required.

Keywords: omega-3 fatty acids, eicosapentaenoic acid, docosahexaenoic acid, curcumin, autoimmune disease, exercise inflammation, recovery, cytokines, specialized pro-resolving mediators

1. Introduction

Autoimmune diseases comprise a heterogeneous group of disorders in which loss of immune tolerance, dysregulated innate and adaptive immune responses, and tissue-specific injury coexist to different degrees. Rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), multiple sclerosis (MS), autoimmune thyroid diseases, type 1 diabetes, inflammatory myopathies, systemic sclerosis and several other conditions differ substantially in pathogenesis and clinical phenotype. Nevertheless, persistent or recurrent inflammatory signalling, treatment-related metabolic consequences, fatigue, reduced cardiorespiratory fitness and loss of muscle function can all contribute to lower habitual physical activity. Exercise is therefore increasingly treated as a component of comprehensive care rather than as a behavior that should automatically be avoided during chronic inflammatory disease [1-3].

The inflammatory response to exercise requires careful interpretation in this context. A single demanding bout can transiently increase circulating cytokines, leukocyte trafficking, muscle-derived interleukin-6 (IL-6), oxidative stress and markers of muscle membrane disruption. These responses are part of normal stress signalling and tissue repair rather than synonymous with pathological inflammation. Repeated exercise, particularly when appropriately dosed, is associated with reduced visceral adiposity, improved endothelial and metabolic function, altered cytokine balance and repeated exposure to anti-inflammatory myokine signalling [1]. Consequently, the clinical objective should not be the indiscriminate elimination of every post-exercise inflammatory signal. A more defensible objective is to prevent excessive, prolonged or poorly resolved inflammation that interferes with recovery, symptoms, adherence or disease control while preserving the signalling required for adaptation.

This distinction is particularly important in autoimmune disease because baseline inflammatory activity and immunomodulatory medication can alter both the starting state and the response to exercise. A 20-year systematic review including 87 controlled exercise studies and 2779 participants with autoimmune diseases concluded that regular exercise generally had a modest anti-inflammatory influence, whereas acute exercise was often neutral or transiently pro-inflammatory [3]. Disease-specific reviews reinforce the same theme. In RA, acute aerobic, resistance or combined exercise did not consistently exacerbate pain, clinical inflammatory markers or cytokine responses compared with controls [4]. In SLE, an eight-week combined aerobic and anaerobic intervention reduced several cytokines in a randomized trial [5], and a 2026 randomized study demonstrated that high-intensity interval training can improve aerobic capacity without a clear improvement in fatigue [6]. In MS, meta-analytic evidence suggests

that regular exercise may reduce tumor necrosis factor alpha (TNF-alpha), whereas IL-6 responses are inconsistent [7].

Nutritional strategies that modify inflammatory signaling are attractive because they could theoretically complement exercise without adding pharmacological burden. Two candidates are especially relevant. The long-chain marine omega-3 polyunsaturated fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) can alter cellular membrane fatty-acid composition, compete with arachidonic acid for enzymatic conversion, influence transcriptional signaling and provide substrates for specialized pro-resolving mediators (SPMs). SPM families including resolvins, protectins and maresins are of particular interest because resolution is an active biological process rather than a passive decline of inflammation [8]. Curcumin, the principal curcuminoid associated with Curcuma longa, has been investigated as a modulator of nuclear factor kappa B (NF-kB), mitogen-activated protein kinase, oxidative and cytokine pathways. Its clinical interpretation is complicated by low and formulation-dependent systemic bioavailability.

The literature, however, is fragmented. Studies of omega-3 or curcumin in autoimmune disease generally examine disease activity or resting inflammatory biomarkers without a standardized exercise challenge. Conversely, exercise-recovery trials of these supplements predominantly involve healthy, recreationally active or athletic populations. The exact intersection of autoimmune disease, supplementation and the acute post-exercise inflammatory response is therefore much smaller than the size of either literature considered separately. Treating indirect evidence as if it were direct evidence would overstate current knowledge.

The present scoping review was designed to map this intersection. It asks not only whether omega-3 fatty acids and curcumin have anti-inflammatory effects, but whether the available human evidence is sufficiently direct to support their use for post-exercise inflammation in people with autoimmune disease. The review therefore separates direct evidence from two complementary evidence streams: supplementation studies in autoimmune populations and exercise studies in autoimmune populations. Evidence from healthy exercise studies is used to evaluate biological plausibility and post-exercise outcomes, not to claim clinical efficacy in autoimmune disease. This structure is intended to identify what is known, what is transferable only with caution, and which questions require dedicated trials.

2. Materials and Methods

2.1. Review design and research questions

This manuscript was designed as a scoping review following the conceptual approach described by JBI and reported with reference to the PRISMA Extension for Scoping Reviews (PRISMA-ScR) [9,10]. Search reporting was informed by PRISMA-S [11]. A scoping approach was selected because the research question spans multiple autoimmune diseases, different exercise modalities, two distinct nutritional interventions, multiple formulations and doses, acute and chronic time frames, and diverse inflammatory or clinical outcomes. The purpose was therefore evidence mapping and gap identification rather than estimation of one pooled treatment effect.

2.2. PCC framework and scope

Eligibility was structured using the Population-Concept-Context framework. The Population comprised people with a clinically established autoimmune disease, without restriction by sex, age, baseline fitness or habitual physical-activity level. The core autoimmune stratum included diseases such as RA, SLE and MS. Conditions frequently described more broadly as immune-mediated inflammatory diseases (IMIDs), including inflammatory bowel disease (IBD), psoriasis, ankylosing spondylitis/axial spondyloarthritis and Behcet disease, were not silently equated with classical autoimmunity. Instead, they were retained as a secondary IMID evidence stratum when relevant to the supplement literature and were identified explicitly as such in the synthesis.

The Concept comprised oral supplementation with EPA, DHA, mixed long-chain omega-3 products, fish oil, algal-derived omega-3 preparations, curcumin, curcuminoids or standardized Curcuma-derived formulations with a stated curcumin exposure. The Context included any level of physical activity and any structured exercise modality, including aerobic/endurance, resistance, concurrent, interval and rehabilitation exercise. Both acute exercise responses and chronic training interventions were considered.

2.3. Information sources and search strategy

No publication-date restriction was applied. The evidence map was developed through structured searches of PubMed/MEDLINE, PubMed Central, official publisher records and ClinicalTrials.gov, supplemented by citation chaining; the final update was completed on 1 August 2026. Search concepts covered named autoimmune diseases, omega-3/EPA/DHA/fish oil, curcumin/curcuminoids/Curcuma, exercise or physical activity, and inflammatory,

immune, muscle-damage and resolution-related outcomes. Complementary searches omitted either the exercise or supplementation block to capture predefined indirect evidence tiers.

A representative PubMed concept structure was: (autoimmun* OR rheumatoid arthritis OR systemic lupus erythematosus OR multiple sclerosis OR relevant named autoimmune/immune-mediated disease) AND (omega-3 OR n-3 PUFA OR EPA OR DHA OR fish oil OR curcumin OR curcuminoid* OR turmeric OR Curcuma longa) AND (exercise OR training OR physical activity OR sport* OR aerobic OR endurance OR resistance) AND (inflamm* OR cytokine* OR CRP OR ESR OR interleukin* OR TNF OR oxylipin* OR resolvin* OR specialized pro-resolving mediator*). Because requiring all four domains would miss clinically informative indirect evidence, complementary searches removed either the exercise domain or the supplementation domain to identify other evidence.

2.4. Eligibility criteria

Original human clinical research was eligible when it contributed to at least one predefined evidence tier and reported an inflammatory, immune, oxidative-stress, resolution-related, muscle-damage or disease-activity outcome. Randomized controlled trials, crossover trials, non-randomized controlled interventions, prospective single-arm interventions and relevant observational studies were considered. Systematic reviews and meta-analyses were used to map consistency, identify additional primary studies and contextualize heterogeneity, but they were not treated as substitutes for direct primary evidence when individual trials were available.

Exclusion criteria were: animal-only, in vitro or ex vivo studies without a clinical component; studies involving only healthy participants unless they specifically tested post-exercise omega-3 or curcumin effects and therefore contributed supportive exercise evidence; non-oral parenteral lipid interventions; multicomponent supplements in which the independent contribution of omega-3 or curcumin could not reasonably be separated; non-standardized turmeric foods with no quantifiable curcumin exposure; case reports; editorials; commercial webpages; non-peer-reviewed claims; and studies without relevant inflammatory, immune, muscle-damage, recovery or disease-activity outcomes.

2.5. Data charting and evidence synthesis

For each key study, the evidence chart considered disease, study design, sample size, background pharmacological therapy, supplement formulation, EPA/DHA or curcumin dose, supplementation duration, exercise modality and dose, timing of biomarker collection,

comparator, inflammatory or immune biomarkers, disease-activity outcomes, adverse events and major limitations. Particular attention was paid to whether biomarkers represented resting chronic inflammation or a time-resolved response after an exercise bout. CRP, ESR, cytokines, CK, soreness, clinical disease activity and SPM-related lipid mediators were not treated as interchangeable endpoints.

Because of major heterogeneity in diseases, exercise prescriptions, doses, formulations and outcomes, no new quantitative meta-analysis was performed. Findings were synthesized narratively and organized by evidence tier and supplement. Greater interpretive weight was assigned to randomized controlled clinical data and to evidence that directly included the target autoimmune population. Supportive exercise studies in healthy adults were explicitly labeled as indirect.

2.6. Critical appraisal

Critical appraisal was used to contextualize rather than exclude evidence, consistent with the mapping purpose of a scoping review. Methodological issues considered included randomization, allocation concealment, blinding, comparator composition, sample size, adherence, baseline omega-3 status, curcumin formulation and bioavailability, exercise standardization, medication stability, dietary control, multiplicity of biomarkers, timing of post-exercise sampling and attrition. Particular caution was applied to small trials with numerous secondary outcomes and to studies reporting within-group changes without convincing between-group effects.

3. Exercise-Associated Inflammation in Autoimmune Disease

3.1. Acute inflammation is not equivalent to pathological inflammation

Acute exercise can transiently increase myokines, leukocyte trafficking and tissue-repair signals. Exercise-induced IL-6, for example, differs in kinetics and source from chronic inflammatory elevation; therefore a single post-exercise cytokine value cannot be interpreted without timing, exercise dose and baseline disease activity [1]. CK, soreness and CRP likewise reflect different processes and time courses. Long-term exercise nevertheless tends to produce modest anti-inflammatory effects across autoimmune diseases [1-3].

3.2. Rheumatoid arthritis

RA has the most mature intersection between exercise, inflammation and omega-3 biology. A systematic review of 11 acute-exercise studies involving 274 participants found that aerobic, resistance or combined exercise did not clearly worsen pain or produce an abnormal

inflammatory response compared with controls, although protocols and sampling varied [4]. The remaining question is whether supplementation changes recovery or resolution; the factorial omega-3 plus aerobic-exercise program discussed in Section 7 was designed to test this directly [12,13].

3.3. Systemic lupus erythematosus

In SLE, exercise can improve fitness without uniform changes in symptoms or cytokines. Eight weeks of combined training reduced several cytokine measures in one randomized trial [5], while a 2026 trial of 12 weeks of high-intensity interval training improved aerobic capacity but not fatigue [6]. These findings show why fitness, symptoms and immune markers should be evaluated together rather than treated as interchangeable outcomes.

3.4. Multiple sclerosis

In MS, a systematic review and meta-analysis found no consistent change in peripheral IL-6 after acute or regular exercise, whereas regular exercise was associated with lower TNF-alpha in within-person analyses [7]. These data argue against using a single cytokine as a gatekeeper for exercise and support prespecified, clinically linked inflammatory endpoints in future supplement trials.

4. Omega-3 Fatty Acids: Mechanistic Rationale

4.1. Membrane composition, eicosanoid competition and inflammatory signaling

EPA and DHA are incorporated into cell membranes, alter arachidonic-acid substrate availability and influence cyclooxygenase/lipoxygenase and transcriptional pathways. Human RA trials show that changes in omega-3 exposure can accompany reduced leukotriene production and selected clinical improvements, although inflammatory markers do not change uniformly [14-17].

4.2. Specialized pro-resolving mediators

EPA and DHA also provide substrates for specialized pro-resolving mediator pathways, including resolvins, protectins and maresins. Supplementation studies in inflammatory arthritis support measurable changes in lipid-mediator pathways [8]. For exercise, the relevant hypothesis is therefore not complete blockade of the acute inflammatory response but more efficient resolution; direct autoimmune-exercise trials with serial lipidomics are still required.

5. Omega-3 Fatty Acids in Autoimmune and Immune-Mediated Disease

5.1. Rheumatoid arthritis

RA provides the strongest clinical evidence for omega-3 supplementation. In recent-onset RA, high-dose fish oil added to algorithm-based DMARD therapy reduced failure of triple-DMARD treatment and increased ACR-defined remission compared with low-dose control, although not all disease-activity outcomes differed [14]. Earlier trials reported modest improvements in symptoms or leukotriene production [15,16]. A 2024 meta-analysis of 18 randomized trials (1018 patients) found improvements in fatty-acid profile, triglycerides and tender joint count, but no significant pooled reductions in ESR, CRP or DAS28 [17].

5.2. Systemic lupus erythematosus

Omega-3 evidence in SLE is smaller and conflicting. Wright et al. reported improvements in disease activity, endothelial function and oxidative-stress measures after 24 weeks [18], whereas Bello et al. did not demonstrate consistent benefit across major outcomes [19]; a pilot study reported a CRP signal [20]. A 2020 meta-analysis found a small reduction in SLE disease activity with low certainty [21]. These resting effects cannot be assumed to predict the acute exercise response.

5.3. Multiple sclerosis

In relapsing-remitting MS, the multicenter OFAMS trial found no significant reduction in MRI activity, relapse, disability progression, fatigue or quality of life with EPA+DHA [22]. Smaller adjunctive studies have reported changes in selected cytokines [23], but the evidence does not establish a consistent clinical effect or a post-exercise indication.

5.4. Secondary immune-mediated inflammatory disease evidence

Secondary IMID evidence broadens biological plausibility but should not be merged with classical autoimmunity. EPA reduced fecal calprotectin and improved maintenance of remission in a randomized ulcerative-colitis trial [24], while psoriasis and ankylosing-spondylitis studies suggest possible but heterogeneous benefits [25,26]. These findings do not support a universal omega-3 effect across immune-mediated diseases.

6. Omega-3 Fatty Acids and Post-Exercise Inflammation

6.1. Evidence from physically healthy adults

In physically healthy adults, systematic evidence suggests that omega-3 may reduce selected markers of muscle damage, inflammation and soreness, but trials vary in dose, duration, baseline omega-3 status, exercise model and sampling time [27]. A 2026 meta-analysis of 41 randomized trials reported pooled reductions in IL-6, TNF-alpha, CK and delayed-onset muscle soreness, with stronger subgroup effects in studies using at least 2 g/day EPA+DHA for at least six weeks [28].

Individual eccentric-exercise studies also reported lower PGE2, IL-6, TNF-alpha, CK-related markers or soreness after omega-3 supplementation [29,30], although functional recovery was not consistently improved. These studies are mechanistically supportive but indirect: healthy participants and standardized muscle-damage protocols differ substantially from medicated patients performing individualized clinical exercise.

6.2. Safety and clinical context

Omega-3 is generally well tolerated but is not risk-free. A 2023 meta-analysis of 90 randomized trials found higher odds of diarrhea and dysgeusia and a modest increase in reported bleeding tendency, without a clear increase in serious supplement-related events [31]. In autoimmune disease, concomitant NSAIDs, antiplatelet drugs, anticoagulants and immunomodulatory therapies make clinical review of dose and interactions appropriate; supplementation should remain adjunctive to standard care.

7. Direct Omega-3 Plus Exercise Evidence in Autoimmune Disease

The RA trial protocol by Jannas-Vela et al., separates placebo, aerobic exercise, omega-3 and omega-3 plus aerobic exercise over 16 weeks [12]. Omega-3 provides 2.5 g/day DHA plus 0.5 g/day EPA, while exercise is performed three times weekly at approximately 60-70% of peak oxygen uptake. The design can distinguish independent, additive and interactive effects while relating fatty-acid incorporation and SPM-related biology to clinical outcomes.

In 2026, Candia et al. reported preliminary results from 13 women randomized only to the placebo or omega-3 arms of this program [13]. Omega-3 reduced morning-stiffness duration and improved RA-specific quality of life but did not significantly change CRP, ESR or TNF-alpha. Because the exercise arms were not reported, the study strengthens supplement-only evidence but does not establish an omega-3-by-exercise effect.

Accordingly, the scarcity of completed Tier 1 evidence is a central finding: disease-specific omega-3 studies and healthy exercise-recovery trials support plausibility, but the clinical bridge between them remains largely untested.

8. Curcumin: Mechanistic Rationale and Formulation Challenges

8.1. Anti-inflammatory signaling

Curcumin has been investigated as a modulator of NF-kB-related transcription, cytokine production, oxidative stress and other inflammatory pathways. Clinical trials across autoimmune and immune-mediated diseases suggest possible effects, but a meta-analysis of 31 randomized trials across 10 conditions emphasized marked disease and formulation heterogeneity rather than a uniform class effect [32]. Mechanistic plausibility should therefore be separated from clinically achievable exposure.

8.2. Bioavailability is a major source of heterogeneity

Native curcumin has low and variable oral bioavailability. Trials use piperine, phospholipid complexes, micelles, nanoparticles and other delivery systems; a review of 165 randomized trials identified 25 enhancement strategies [33]. Consequently, equal milligram doses cannot be assumed to produce equal exposure. Curcumin and piperine may also affect drug-metabolizing enzymes or transporters, although the clinical importance of many proposed interactions is uncertain [34,35].

9. Curcumin in Autoimmune and Immune-Mediated Disease

9.1. Rheumatoid arthritis

RA trials of curcumin are generally small. A pilot randomized study comparing curcumin, diclofenac and their combination reported favorable DAS and ACR responses with curcumin, but lacked a placebo-only arm [36]. Nanomicellar and other curcumin formulations have also produced improvements in selected symptoms, metabolic or inflammatory variables, although between-group effects are not uniform [37,38]. Meta-analytic synthesis remains promising but low-certainty and heterogeneous [32]; none of these trials directly tests post-exercise inflammation.

9.2. Systemic lupus erythematosus

In SLE, a randomized placebo-controlled trial of 1000 mg/day curcumin for ten weeks reported reductions in anti-dsDNA antibodies and IL-6, while several other outcomes were

unchanged [39]. The finding supports a disease-specific immunological signal but does not establish an exercise effect or a post-exercise dose recommendation.

9.3. Multiple sclerosis

In relapsing-remitting MS, six months of adjunctive nanocurcumin altered selected inflammatory transcription factors and cytokines in a randomized trial [40]. These mechanistic findings require replication and clinically powered outcomes; as with omega-3, curcumin and exercise studies in MS largely remain parallel rather than intersecting evidence streams.

9.4. Secondary IMID evidence

Curcumin evidence is broader in secondary IMIDs. Small randomized studies reported immunological or clinical signals in ankylosing spondylitis and psoriasis [41,42]. In IBD, randomized trials found lower relapse with adjunctive curcumin in quiescent ulcerative colitis, improved remission/response when added to mesalamine in active ulcerative colitis, and benefit with a highly bioavailable derivative in Crohn disease [43-45]. These findings support anti-inflammatory plausibility but remain indirect for the autoimmune-exercise question.

10. Curcumin and Post-Exercise Inflammation

10.1. Systematic evidence in healthy and athletic populations

Systematic reviews of curcumin in physically active populations report possible reductions in soreness, CK and selected inflammatory or oxidative markers, but formulations, doses, timing and exercise models differ substantially [46-49]. A 2026 review of 15 double-blind placebo-controlled exercise studies rated certainty as low for oxidative stress and very low for muscle damage, inflammation, subjective recovery and performance, with favorable effects present only in subsets of trials [50].

10.2. Representative randomized exercise studies

Representative eccentric-exercise studies illustrate this inconsistency. Bioavailable curcumin attenuated selected muscle-damage outcomes in untrained men, while IL-6 and TNF-alpha were not consistently reduced; timing before versus after exercise appeared to influence which cytokine, soreness or functional outcomes changed [51,52]. A small crossover study in elite rugby players also reported improvements in selected recovery outcomes with curcumin plus piperine [53]. These data support possible symptom or muscle-damage effects, not reliable systemic cytokine suppression or proven autoimmune benefit.

The healthy exercise literature also raises a conceptual concern: greater suppression of a biochemical marker is not automatically better. Exercise-induced inflammatory and redox signals participate in adaptation, and excessive antioxidant or anti-inflammatory intervention could theoretically alter signaling. Human evidence showing clinically meaningful impairment of long-term adaptation by curcumin is insufficient, but a prudent research design should measure both recovery and adaptation rather than assume they always move in the same direction.

11. Comparative and Integrative Interpretation

11.1. Omega-3 versus curcumin

Omega-3 and curcumin should not be treated as interchangeable 'natural anti-inflammatories'. Omega-3 has the stronger disease-specific evidence base, particularly in RA [14-17], with possible modest effects in SLE [18-21] and less convincing MS data [22,23]. Curcumin has promising but more formulation-sensitive evidence across RA, SLE, MS and several IMIDs [32-45]. In post-exercise research, both show signals for lower soreness, CK or selected cytokines, but systematic reviews emphasize heterogeneity and low certainty [27,28,46-50]. Neither has sufficient direct evidence to define a standard regimen for post-exercise inflammation in autoimmune disease.

Thus, the strongest conclusion is not that one supplement is definitively superior. Rather, omega-3 has a clearer disease-specific rationale and biomarker of exposure (EPA/DHA incorporation), while curcumin offers broader but more formulation-sensitive anti-inflammatory evidence. Neither has sufficient direct trial evidence to define a standard regimen for post-exercise inflammation in autoimmune disease.

11.2. Could omega-3 and curcumin be complementary?

Combining omega-3 and curcumin is mechanistically plausible because they influence partly distinct pathways, but no adequately controlled human trial was identified that tested the combination around exercise in an autoimmune population. Without factorial testing, clinical synergy cannot be inferred; recommending the combination specifically for post-exercise autoimmune inflammation would therefore exceed the evidence.

Table 1. Representative human evidence relevant to the review question.

Population	Study	Intervention	Exercise context	Key signal	Interpretation
RA	Proudman et al. [14]	High- vs low-dose fish oil within treat-to-target DMARD care	None standardized	Lower DMARD failure; higher ACR remission; not all disease activity outcomes differed	Supports disease-specific adjunctive omega-3 effect, not post-exercise efficacy.
RA	Jannas-Vela et al. [12]; Candia et al. [13]	Omega-3 (2.5 g DHA + 0.5 g EPA/day) factorial program	Cycle ergometry planned 3x/week, 60-70% VO2peak	Protocol includes SPMs, inflammation, synovial fluid and clinical outcomes; preliminary omega-3-only report improved morning stiffness/QoL without changing CRP, ESR or TNF-alpha	Most direct program; exercise-combination outcomes not yet identified.
SLE	Wright et al. [18]	3 g/day omega-3 for 24 weeks	None standardized	Improved selected disease activity, endothelial and	Positive supplement evidence; no exercise

Population	Study	Intervention	Exercise context	Key signal	Interpretation
				oxidative measures	interaction tested.
MS	Torkildsen et al. [22]	EPA+DHA vs placebo	None standardized	No reduction in MRI disease activity, relapse or disability	Important null disease-specific omega-3 evidence.
Healthy endurance athletes	Ramos-Campo et al. [30]	DHA-rich omega-3 for 10 weeks	Eccentric muscle-damage challenge	Lower IL-1beta, IL-6, CK-related markers and soreness; no strength benefit	Supports post-exercise mechanism but indirect for autoimmune disease.
RA	Chandran & Goel [36]	Curcumin 500 mg, diclofenac or combination	None standardized	Pilot improvements in DAS/ACR outcomes	Promising, small and not exercise-specific.
SLE	Sedighi et al. [39]	Curcumin 1000 mg/day for 10 weeks	None standardized	Lower IL-6 and anti-dsDNA; other outcomes unchanged	Disease-specific anti-inflammatory signal without exercise.
Healthy adults	Tanabe et al. [52,53]	Bioavailable curcumin around eccentric exercise	Eccentric elbow-flexor exercise	Some reductions in CK, IL-8 or soreness depending on timing; cytokine effects not uniform	Suggests timing-specific recovery effects; indirect.

12. Practical Implications for Physically Active Individuals with Autoimmune Disease

The evidence supports regular, individualized exercise more strongly than either supplement as a post-exercise anti-inflammatory intervention. Exercise modality and progression should be adapted to disease activity, symptoms, disability and treatment, and supplements should not be used to compensate for a repeatedly excessive exercise dose.

Omega-3 may be considered an adjunct where clinically appropriate, particularly in RA, but exercise-study doses should not be converted into universal autoimmune recommendations because medication, bleeding risk, product quality and baseline fatty-acid status differ. Curcumin requires equally careful attention to formulation, curcuminoid content and bioavailability strategy; products containing piperine or advanced delivery systems may have different interaction potential [33-35].

Neither supplement should replace DMARDs, biologic therapy, glucocorticoid management, rehabilitation or medical follow-up. The goal is also not to suppress every transient post-exercise signal, but to improve disproportionate symptoms or prolonged inflammation while preserving training tolerance, function and disease control.

13. Major Moderators and Sources of Heterogeneity

Major sources of heterogeneity include disease phenotype and activity, immunomodulatory medication, baseline nutritional status, supplement formulation and dose, exercise mode and relative intensity, biomarker timing, diet, sleep, energy availability and training status. Omega-3 response may depend on baseline EPA/DHA exposure, while curcumin exposure varies markedly by delivery system [27,33]. Immediate cytokines, 24-hour CK and resting CRP represent different phases of recovery; unaccustomed eccentric exercise also produces much larger responses than habitual clinical training. Future studies should therefore standardize or at least measure these variables and report exercise relative to individual capacity.

14. Limitations of the Evidence and of This Review

The principal limitation is the scarcity of studies that directly combine autoimmune disease, supplementation and exercise; most conclusions therefore triangulate separate literatures. Clinical heterogeneity is also substantial: RA, SLE, MS and organ-specific autoimmune diseases differ in pathophysiology and treatment, while secondary IMIDs should not be interpreted as equivalent to classical autoimmunity. Outcome measures such as CRP, ESR, cytokines, lipid mediators, CK, soreness, fatigue and disease activity are related but not

interchangeable. Supplement exposure is inconsistently characterized: omega-3 trials often omit baseline fatty-acid status and differ in EPA:DHA ratio and duration [27], whereas curcumin studies use formulations with widely different bioavailability [33].

15. Future Research Priorities

- Direct factorial randomized trials should test supplement, exercise and their interaction; the RA design of Jannas-Vela et al. provides a useful model [12]. Trials should collect both acute serial post-exercise samples and longer-term resting outcomes to distinguish transient signaling from chronic disease modification.
- Omega-3 trials should report baseline and post-intervention EPA/DHA status, exact EPA:DHA dose and chemical form; curcumin trials should specify formulation, curcuminoid content, bioavailability strategy, dose and timing. SPM-related studies require validated targeted lipidomics.
- Exercise should be individualized and reproducibly described by modality, relative intensity, volume, supervision, adherence and training history. Medication should be stable where appropriate and explicitly modeled, including NSAIDs, glucocorticoids, DMARDs and anticoagulants.
- Biomarkers should be paired with clinically meaningful outcomes such as disease activity, flare frequency, fatigue, pain, next-session performance, exercise adherence, function and quality of life. Larger preregistered studies should examine effect modification and report null findings as completely as positive results.

16. Conclusion

Current evidence supports biological plausibility for omega-3 fatty acids and curcumin, but direct evidence that either reliably modulates post-exercise inflammation in autoimmune disease is insufficient. Omega-3 has the stronger disease-specific clinical foundation, especially in RA [14-17], with modest or inconsistent evidence in SLE and MS [18-23]. Healthy exercise studies suggest possible reductions in selected cytokines, CK and soreness [27-30]. Curcumin shows promising but formulation-dependent signals across autoimmune/immune-mediated diseases and exercise-recovery studies, with generally low certainty [32-53].

Both supplements should therefore be considered candidate adjuncts rather than established post-exercise anti-inflammatory therapies. The priority is well-powered, medication-aware randomized research integrating standardized exercise, serial inflammatory

and resolution biomarkers, clinically meaningful recovery outcomes and long-term disease activity. Until then, supplementation should remain individualized and adjunctive to standard care, without assuming that greater suppression of an acute inflammatory signal necessarily improves adaptation.

Disclosure

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Conflicts of Interest

The authors declare no conflict of interest.

Declaration on the Use of Artificial Intelligence

AI-assisted tools were used exclusively for linguistic refinement. The authors take full responsibility for the scientific content, interpretation of the data, and final version of the manuscript.

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