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Review Article

A Systematic Review of Marine Omega-3 Fatty Acids for the Reduction of Neuroinflammation in Major Depressive Disorder

Shah Nawaz[1], Bilal Safdar[2], Sana Ahmed[3], Arooj Fatima Awan[4], Muhammad Uzair Mazhar[4], Farkhunda Naz[5]

[1] Senior Registrar, Balochistan Institute of Psychiatry and Behavioural Sciences, Quetta, Pakistan
[2] House Officer, CMH Lahore Medical and Dental College, Lahore, Pakistan. ORCID: https://orcid.org/0009-0009-1316-3371
[3] Lecturer in Inorganic Chemistry, Department of Chemistry, Faculty of Basic Sciences, Sardar Bahadur Khan Women's University, Quetta, Pakistan
[4] MBBS, MD, King Edward Medical University, Lahore, Pakistan. Muhammad Uzair Mazhar ORCID: https://orcid.org/0009-0004-0711-0883
[5] Senior House Officer, Al Kharj Armed Forces Hospital, Riyadh, Saudi Arabia.

***Corresponding author: Shah Nawaz, Shahzehri46@gmail.com**

ABSTRACT

Background: Major depressive disorder is increasingly linked with immune-inflammatory dysregulation, and elevated inflammatory biomarkers may contribute to symptom persistence and treatment resistance in selected patients. Marine omega-3 fatty acids, particularly eicosapentaenoic acid and docosahexaenoic acid, have been proposed as adjunctive modulators of inflammatory pathways, but evidence remains inconsistent. Objective: This systematic review evaluated whether marine-derived omega-3 fatty acid supplementation is associated with modulation of inflammatory biomarkers in adults with major depressive disorder. Methods: A systematic review without meta-analysis was conducted using studies identified from PubMed, Scopus, Web of Science, and the Cochrane Library. Eligible studies included randomized controlled trials and observational studies involving adults with major depressive disorder receiving EPA, DHA, or combined marine omega-3 supplementation and reporting inflammatory biomarker outcomes. Risk of bias was assessed according to study design, and findings were synthesized qualitatively because of heterogeneity in interventions, comparators, and outcome reporting. Results: Eight studies comprising 525 participants were included. Five were randomized controlled trials, two were cohort studies, and one was case-control. Omega-3 supplementation showed a consistent qualitative trend toward reductions in IL-6, CRP, and TNF-α, particularly in EPA-containing interventions. However, exact biomarker values, confidence intervals, and effect sizes were incompletely reported. Conclusion: Marine omega-3 fatty acids may modestly improve inflammatory biomarker profiles in major depressive disorder, but current evidence remains suggestive rather than definitive. Larger standardized trials are required. Keywords: Major Depressive Disorder; Omega-3 Fatty Acids; Eicosapentaenoic Acid; Docosahexaenoic Acid; Inflammatory Biomarkers; Systematic Review.

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INTRODUCTION

Major depressive disorder is a prevalent and disabling psychiatric condition with substantial clinical, social, and functional burden. Although traditionally conceptualized through affective, cognitive, and neurochemical mechanisms, increasing evidence indicates that immune-inflammatory dysregulation may contribute to the onset, persistence, severity, and treatment responsiveness of depressive illness. Elevated peripheral inflammatory biomarkers, including interleukin-6, tumor necrosis factor-alpha, and C-reactive protein, have been reported in subsets of patients with depression and may reflect systemic inflammatory activation relevant to neurobiological pathways involved in mood regulation. However, these circulating biomarkers should be interpreted as indirect inflammatory indicators rather than direct measures of central neuroinflammation, making precise terminology essential when evaluating therapeutic effects in major depressive disorder (1,2).

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Marine-derived omega-3 polyunsaturated fatty acids, particularly eicosapentaenoic acid and docosahexaenoic acid, have attracted clinical and research interest because of their roles in neuronal membrane function, lipid mediator signaling, immune regulation, oxidative balance, and inflammation resolution. EPA may be especially relevant to inflammatory modulation because it can compete with arachidonic acid metabolism and contribute to the generation of less pro-inflammatory and pro-resolving mediators, while DHA contributes to neuronal membrane integrity and synaptic function. These biological mechanisms provide a plausible rationale for examining whether EPA- and DHA-containing interventions can modify inflammatory biomarker profiles in adults with major depressive disorder (3,4).

Despite this mechanistic rationale, the available clinical evidence remains inconsistent. Existing studies vary in diagnostic criteria, participant inflammatory status, omega-3 formulation, EPA-to-DHA ratio, dosage, duration of supplementation, comparator condition, concomitant antidepressant use, and biomarker measurement procedures. Some studies suggest reductions in inflammatory markers after omega-3 supplementation, particularly with EPA-rich formulations, whereas others report non-significant or heterogeneous findings. This variability limits confidence in clinical interpretation and highlights the need for a systematic synthesis focused specifically on inflammatory biomarker outcomes rather than broad antidepressant efficacy alone (5–8).

Therefore, this systematic review was designed using a PICO framework to evaluate adults with major depressive disorder, receiving marine omega-3 fatty acids containing EPA and/or DHA, compared with placebo, standard care, antidepressant treatment alone, no supplementation, or relevant control conditions, for changes in inflammatory biomarkers such as IL-6, TNF-α, CRP, and related cytokine measures. The objective was to synthesize available human evidence on whether marine omega-3 fatty acid supplementation is associated with measurable modulation of inflammatory biomarkers in major depressive disorder, while identifying methodological limitations, sources of heterogeneity, and evidence gaps requiring further investigation (9).

MATERIAL AND METHODS

This study was conducted as a systematic review without meta-analysis to evaluate the association between marine-derived omega-3 fatty acid supplementation and inflammatory biomarker modulation in adults with major depressive disorder. The review followed PRISMA 2020 principles and used a PICO framework. The population comprised adults diagnosed with major depressive disorder using standardized diagnostic criteria or validated clinical assessment procedures. The intervention included marine-derived omega-3 fatty acids containing eicosapentaenoic acid, docosahexaenoic acid, or combined EPA+DHA formulations. Comparator conditions included placebo, usual care, antidepressant therapy alone, no supplementation, or other non-omega-3 control groups. The primary outcomes were inflammatory biomarkers, including interleukin-6, tumor necrosis factor-alpha, C-reactive protein, cytokine profiles, and other reported markers of inflammatory activity. Depressive symptom outcomes were considered secondary and were interpreted separately from biomarker modulation.

A systematic literature search was performed in PubMed, Scopus, Web of Science, and the Cochrane Library. The search strategy combined terms for depression, omega-3 fatty acids, and inflammatory outcomes using Boolean operators. Core search concepts included “major depressive disorder” OR “depression,” combined with “omega-3 fatty acids” OR “eicosapentaenoic acid” OR “EPA” OR “docosahexaenoic acid” OR “DHA,” and “inflammation” OR “neuroinflammation” OR “inflammatory biomarkers” OR “cytokines” OR “interleukin-6” OR “tumor necrosis factor-alpha” OR “C-reactive protein.” Search syntax was adapted to the indexing structure of each database. Reference lists of relevant articles and review papers were also manually screened to identify additional eligible studies.

Studies were eligible if they involved adult human participants with major depressive disorder, evaluated EPA, DHA, or combined marine omega-3 supplementation, included a comparator or observational

comparison framework, and reported at least one inflammatory biomarker outcome. Randomized controlled trials and observational studies were eligible. Studies were excluded if they were animal studies, pediatric studies, conference abstracts without full text, unpublished reports, studies involving non-depressive psychiatric populations without separable major depressive disorder data, or studies that did not report inflammatory biomarker outcomes. Articles not available in English were excluded.

All retrieved citations were imported into reference management software, and duplicate records were removed before screening. Titles and abstracts were screened independently by two reviewers according to the predefined eligibility criteria. Potentially relevant articles underwent full-text assessment. Disagreements regarding inclusion were resolved through discussion and consensus. The study selection process was documented using a PRISMA flow structure, including records identified, duplicates removed, records screened, records excluded, full-text articles assessed, and final studies included.

Data extraction was performed using a standardized extraction form. Extracted variables included study identifier, design, sample size, participant characteristics, diagnostic criteria for major depressive disorder, intervention composition, EPA and DHA formulation, dosage where reported, duration of supplementation, comparator condition, concomitant antidepressant use, inflammatory biomarkers assessed, timing of biomarker measurement, direction and magnitude of biomarker change where available, statistical significance where reported, depressive symptom outcomes where available, and study-level limitations. Data extraction was performed independently by two reviewers and checked for consistency before synthesis.

Risk of bias was assessed according to study design. Randomized controlled trials were evaluated across domains including randomization, allocation concealment, blinding, incomplete outcome data, selective reporting, and other potential threats to validity. Observational studies were assessed using domains relevant to participant selection, comparability of groups, outcome assessment, follow-up adequacy, and confounding control. Each study was categorized as low, moderate, or high overall risk of bias based on domain-level judgments. Risk-of-bias findings were incorporated into the interpretation of evidence, with greater weight given to randomized studies and studies with lower methodological concern.

A quantitative meta-analysis was not performed because of heterogeneity in study design, omega-3 formulation, EPA-to-DHA ratio, intervention duration, comparator type, biomarker selection, and outcome reporting. Instead, findings were synthesized qualitatively by comparing study characteristics, biomarker coverage, direction of effect, methodological quality, and consistency of findings across studies. No unreported p-values, confidence intervals, effect sizes, or biomarker values were imputed or generated. When statistical details were absent or incomplete, results were described cautiously and interpreted as qualitative evidence only. Ethical approval was not required because this review used previously published data and did not involve direct recruitment of human participants or access to identifiable individual-level information. To strengthen reproducibility and transparency, the review applied predefined eligibility criteria, structured screening procedures, standardized data extraction, risk-of-bias assessment by study type, and cautious synthesis aligned with the available evidence.

RESULTS

The database search identified 120 records from PubMed, Scopus, Web of Science, and the Cochrane Library. After removal of 25 duplicate records, 95 unique records were screened by title and abstract. Of these, 87 records were excluded because they were unrelated to the review question, included unsuitable populations, or did not report inflammatory biomarker outcomes. Eight full-text studies met the eligibility criteria and were included in the final qualitative synthesis. No study was included in quantitative meta-analysis because of heterogeneity in study design, omega-3 formulation, comparator type, intervention duration, and biomarker reporting. The final evidence base comprised 525 participants across eight studies.

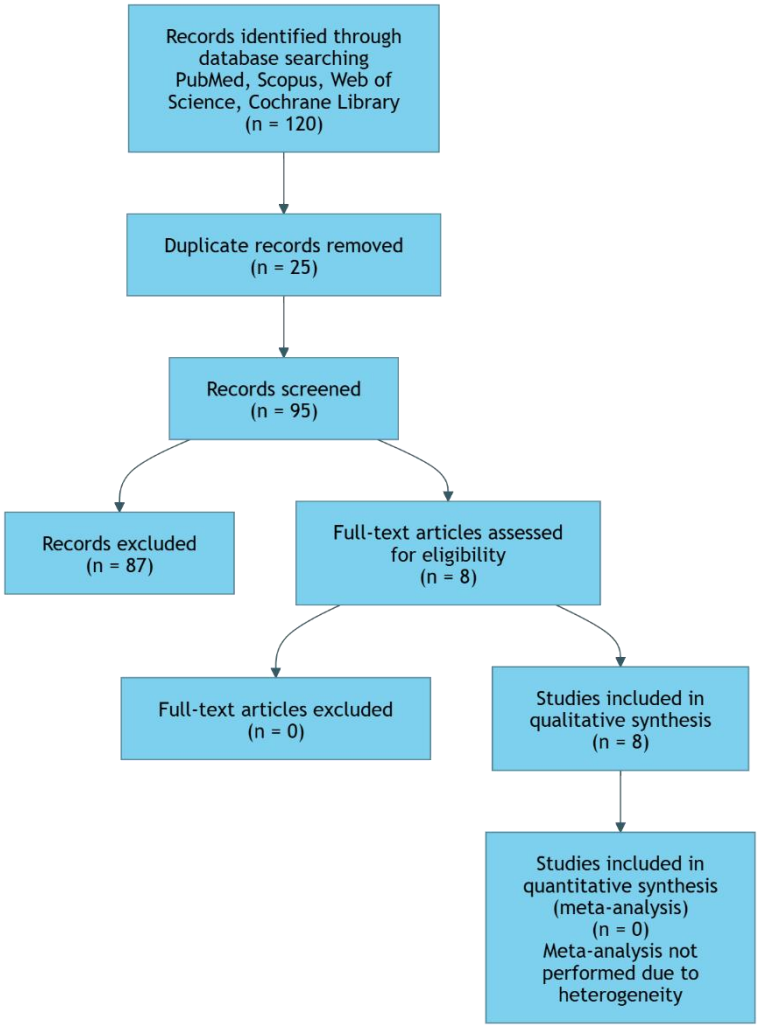

Figure 1 PRISMA Flowchart

The included studies consisted of five randomized controlled trials, two cohort studies, and one case-control study. Sample sizes ranged from 40 to 90 participants, indicating that most included studies were small to moderate in scale. Intervention duration ranged from 6 to 24 weeks. EPA-containing interventions were the most common, appearing either as EPA alone, EPA+DHA, high-EPA formulations, or EPA used adjunctively with antidepressant therapy. DHA alone was evaluated in one cohort study. Comparator conditions included placebo, standard care, antidepressant therapy alone, no supplementation, and controls. The most frequently reported biomarkers were interleukin-6 and C-reactive protein, followed by tumor necrosis factor-alpha.

Table 1. Characteristics of Included Studies

Study	Design	Sample Size	Intervention	Comparator	Duration	Biomarkers / Key Outcomes
S1	Randomized controlled trial	60	EPA	Placebo	12 weeks	IL-6, CRP
S2	Randomized controlled trial	75	EPA + DHA	Standard care	16 weeks	TNF-α, IL-6
S3	Cohort study	50	DHA	No comparator supplementation	8 weeks	CRP
S4	Randomized controlled trial	90	High-EPA formulation	Placebo	24 weeks	IL-6, TNF-α
S5	Case-control study	40	Omega-3 supplementation	Controls	10 weeks	Cytokine profile
S6	Randomized controlled trial	85	EPA + antidepressant therapy	Antidepressant therapy alone	12 weeks	CRP, IL-6
S7	Cohort study	55	EPA + DHA	No comparator supplementation	6 weeks	TNF-α

Study	Design	Sample Size	Intervention	Comparator	Duration	Biomarkers / Key Outcomes
S8	Randomized controlled trial	70	EPA	Placebo	12 weeks	IL-6, CRP

Among the 525 participants, randomized controlled trials contributed 380 participants, representing approximately 72.4% of the total evidence base, while observational studies contributed 145 participants, representing 27.6%. The largest study was S4, with 90 participants and the longest intervention duration of 24 weeks. The smallest study was S5, a case-control study with 40 participants. Three studies used placebo comparators, one used standard care, one compared EPA plus antidepressant therapy against antidepressant therapy alone, two cohort studies had no active comparator, and one case-control study used controls. This distribution indicates that although randomized evidence predominated, comparator heterogeneity limited direct quantitative pooling.

Table 3. Distribution of Study Designs and Participants

Study Design	Number of Studies	Total Participants	Percentage of Total Sample
Randomized controlled trials	5	380	72.4%
Cohort studies	2	105	20.0%
Case-control study	1	40	7.6%
Total	8	525	100.0%

Risk-of-bias assessment showed variable methodological quality. Four studies were judged to have low overall risk of bias, three had moderate overall risk, and one had high overall risk. Low-risk studies were mainly randomized controlled trials with stronger control of selection, detection, and reporting bias. Moderate-risk studies commonly had concerns related to performance bias, attrition, or observational design. The highest-risk study was the case-control study, mainly because of high selection and performance bias. Reporting bias was rated low across studies, but this should be interpreted cautiously because protocol availability and selective outcome reporting could not be fully verified from the presented manuscript data.

Table 3. Risk-of-Bias Appraisal of Included Studies

Study	Selection Bias	Performance Bias	Detection Bias	Attrition Bias	Reporting Bias	Overall Risk
S1	Low	Low	Low	Moderate	Low	Low
S2	Low	Moderate	Low	Low	Low	Low
S3	High	Moderate	Moderate	Low	Low	Moderate
S4	Low	Low	Low	Low	Low	Low
S5	High	High	Moderate	Moderate	Low	High
S6	Low	Moderate	Low	Moderate	Low	Moderate
S7	Moderate	Moderate	Moderate	Low	Low	Moderate
S8	Low	Low	Low	Low	Low	Low

Table 4. Overall Risk-of-Bias Distribution

Overall Risk Category	Number of Studies	Percentage
Low risk	4	50.0%
Moderate risk	3	37.5%
High risk	1	12.5%

The most frequently assessed biomarker was IL-6, reported in five studies, followed by CRP in four studies and TNF-α in three studies. One study reported cytokine outcomes without sufficient biomarker-specific detail in the available manuscript text. Across studies, the direction of findings generally favored reduced inflammatory biomarker activity after omega-3 supplementation. However, the manuscript does not provide exact baseline and post-intervention biomarker values, mean changes, confidence intervals, standardized effect sizes, or exact p-values. Therefore, the synthesis supports a qualitative trend rather than a statistically pooled conclusion.

Table 5. Biomarker Outcomes Across Included Studies

Biomarker	Number of Studies Reporting Biomarker	Studies Reporting Outcome
IL-6	5	S1, S2, S4, S6, S8

Biomarker	Number of Studies Reporting Biomarker	Studies Reporting Outcome
CRP	4	S1, S3, S6, S8
TNF-α	3	S2, S4, S7
Unspecified cytokines	1	S5

The findings suggest that EPA-containing interventions were most commonly associated with favorable biomarker trends. EPA alone was evaluated in two randomized controlled trials, high-EPA supplementation in one randomized controlled trial, and EPA combined with antidepressant therapy in one randomized controlled trial. Combined EPA+DHA was assessed in one randomized controlled trial and one cohort study, while DHA alone was assessed in one cohort study. Although the manuscript suggests stronger effects in studies using higher EPA concentrations, this statement cannot be confirmed quantitatively without EPA dose, EPA:DHA ratio, biomarker change values, and exact inferential statistics.

Table 6. Intervention Pattern and Biomarker Coverage

Intervention Type	Number of Studies	Study Designs	Main Biomarkers Reported
EPA alone	2	RCTs	IL-6, CRP
EPA + DHA	2	RCT, cohort	TNF-α, IL-6
DHA alone	1	Cohort	CRP
High-EPA formulation	1	RCT	IL-6, TNF-α
EPA + antidepressant therapy	1	RCT	CRP, IL-6
General omega-3	1	Case-control	Cytokines

Overall, the qualitative synthesis indicates that marine omega-3 fatty acid supplementation may be associated with reductions in inflammatory biomarkers among adults with major depressive disorder, especially where EPA-containing formulations were used. Nevertheless, the certainty of this finding is limited by small sample sizes, inconsistent comparator conditions, heterogeneous intervention duration, incomplete dose reporting, and lack of extractable numerical outcome data. The evidence is therefore best interpreted as suggestive of potential anti-inflammatory biomarker modulation rather than definitive proof of clinical efficacy.

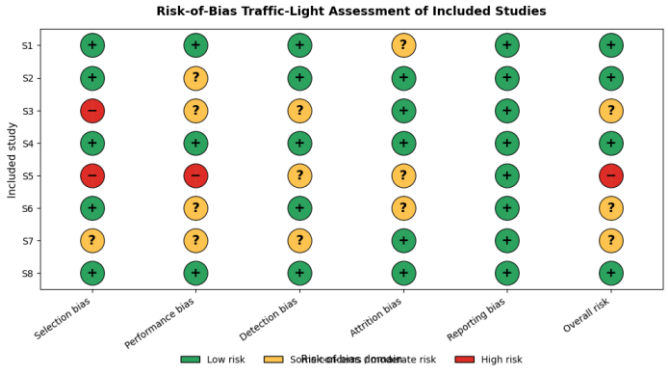

Figure 2 Risk-of-Bias Traffic-Light Assessment of Included Studies

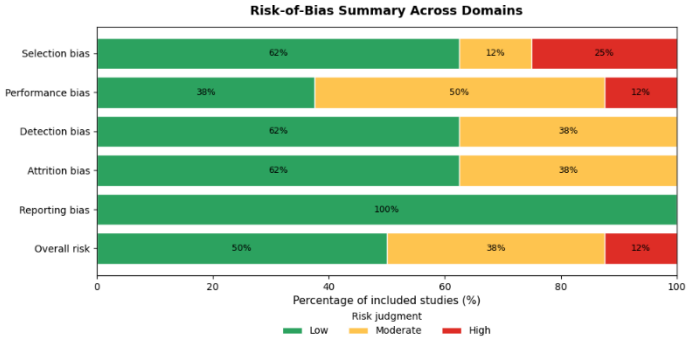

Figure 3 Risk-of-Bias Summary Across Domains

DISCUSSION

This systematic review synthesized evidence from eight studies involving 525 participants to evaluate whether marine-derived omega-3 fatty acids are associated with modulation of inflammatory biomarkers in adults with major depressive disorder. The available evidence suggests a generally favorable direction of effect, particularly for IL-6, CRP, and TNF-α, with EPA-containing interventions appearing most frequently among studies reporting reductions in inflammatory markers. However, these findings should be interpreted cautiously because the included studies varied in design, comparator condition, omega-3 formulation, intervention duration, and biomarker reporting. The absence of extractable baseline and post-intervention biomarker values, exact p-values, confidence intervals, and standardized effect estimates limits the ability to determine the magnitude and precision of the observed effects. Therefore, the current synthesis supports a qualitative trend toward inflammatory biomarker reduction rather than definitive quantitative evidence of efficacy.

The findings are biologically plausible because EPA and DHA participate in inflammatory regulation, cell membrane signaling, and the generation of lipid mediators involved in inflammation resolution. EPA-rich formulations may be particularly relevant because EPA can compete with arachidonic-acid-derived inflammatory pathways and may reduce production of pro-inflammatory mediators. Nevertheless, the manuscript's findings should not be interpreted as direct proof of reduced central neuroinflammation, because the reported outcomes are mainly peripheral inflammatory biomarkers such as IL-6, CRP, and TNF-α. These biomarkers may reflect systemic inflammatory burden and may be clinically relevant in depressive subgroups with inflammatory activation, but they remain indirect indicators rather than direct measures of neuroimmune activity within the central nervous system.

The risk-of-bias appraisal further moderates confidence in the review findings. Four of the eight studies were rated as low overall risk of bias, three as moderate risk, and one as high risk. Performance bias and selection bias were the most frequent concerns, especially in observational designs and smaller studies. Although randomized controlled trials contributed most of the participant pool, the small-to-moderate sample sizes and heterogeneity of interventions reduce the certainty of inference. The inclusion of cohort and case-control studies broadens the evidence base but also increases susceptibility to confounding, particularly where antidepressant use, baseline inflammatory status, diet, comorbid metabolic disease, and severity of depression were not clearly controlled.

The review has several important limitations. First, the included studies were not identified by author and year in the available synthesis table, limiting transparency and independent verification. Second, the lack of exact numerical biomarker outcomes prevented meta-analysis and restricted interpretation to narrative synthesis. Third, variation in EPA:DHA ratio, supplementation dose, duration, and comparator condition makes it difficult to determine which omega-3 formulation is most clinically relevant. Fourth, the current evidence does not establish whether biomarker improvements translate into meaningful improvement in depressive symptoms. Future studies should use standardized MDD diagnostic criteria, prespecified inflammatory biomarker endpoints, adequate blinding and allocation concealment, clear reporting of EPA and DHA doses, and complete presentation of effect estimates with 95% confidence intervals. Stratified analyses by baseline inflammatory status, depression severity, antidepressant use, and metabolic comorbidity would help clarify which patients are most likely to benefit.

Overall, marine omega-3 fatty acids remain a promising adjunctive intervention for inflammatory biomarker modulation in major depressive disorder, particularly in EPA-containing formulations. However, the present evidence is suggestive rather than conclusive. Stronger recommendations will require larger, well-designed randomized controlled trials with standardized biomarker measurement and transparent reporting of clinically interpretable outcomes.

CONCLUSION

Marine-derived omega-3 fatty acids, especially EPA-containing formulations, show a consistent qualitative trend toward reducing inflammatory biomarkers such as IL-6, CRP, and TNF-α in adults with major depressive disorder. However, the evidence remains limited by small study samples, heterogeneous intervention protocols, incomplete numerical reporting, and variable methodological quality. Current findings support omega-3 supplementation as a biologically plausible adjunctive strategy for further investigation rather than as a definitive biomarker-targeted treatment recommendation. Future trials should apply standardized dosing, prespecified inflammatory endpoints, rigorous risk-of-bias control, and complete effect-size reporting to clarify clinical utility.

REFERENCES

1. Malau IA, Chang JP-C, Lin Y-W, Chang C-C, Chiu W-C, Su K-P. Omega-3 fatty acids and neuroinflammation in depression: targeting damage-associated molecular patterns and neural biomarkers. 2024;13(21):1791.

2. Raza ML, Hassan ST, Jamil S, Fatima W, Fatima M. Nutritional interventions in depression: the role of vitamin D and omega-3 fatty acids in neuropsychiatric health. 2025;45:270-80.

3. Lastretti M, Campedelli L, Scarparo T, Spagna S, Cicoli A, Faa G, et al. Omega-3 fatty acids and mood disorders: a critical narrative review. 2026;3(1):2.

4. Simon MS, Arteaga-Henriquez G, Algendy AF, Siepmann T, Illigens BM. Anti-inflammatory treatment efficacy in major depressive disorder: a systematic review of meta-analyses. 2023:1-25.

5. Saadh MJ, Ghnim ZS, Mahdi MS, Baldaniya L, Karim SA, Srivastava M, et al. The effect of omega-3 supplementation on metabolic, inflammatory and oxidative stress biomarkers in pregnant women: a systematic review and meta-analysis. 2025;12:1639906.

6. Ge Z, Hu Y, Kan W, Li L, Xu J, Zhang Y, et al. Lipid metabolic dysregulation-induced neuroinflammation in the pathophysiology of major depressive disorder. 2025;16:1625087.

7. Kim KY, Shin KY, Chang K-A. Potential inflammatory biomarkers for major depressive disorder related to suicidal behaviors: a systematic review. Int J Mol Sci. 2023;24(18):13907.

8. Nave CB, Pereira P, Silva ML. The effect of polyunsaturated fatty acid supplementation on clinical manifestations and inflammatory parameters in individuals with Sjögren's syndrome: a literature review of randomized controlled clinical trials. Nutrients. 2024;16(21):3786.

9. Ilavská L, Morvová M Jr, Paduchová Z, Muchová J, Garaiova I, Ďuračková Z, et al. The kynurenine and serotonin pathway, neopterin and biopterin in depressed children and adolescents: an impact of omega-3 fatty acids, and association with markers related to depressive disorder. A randomized, blinded, prospective study. 2024;15:1347178.

10. Mozaffarian D, Freeman MW, Swenson S. Fish oil: physiologic effects and administration. 2024.

11. Yılmaz Çakan GC, Yılmaz Küsbeci Ö. Dietary approaches to support mental health in chronic pain and opioid use disorders. In: Feeding the Mind: The Connection Between Diet, Drugs, and Mental Health. Volume 1. Springer; 2025. p. 377-403.

12. Maes M, Almulla A, Drozdstoj S, Zhang Y. Hallmarks of major depression: neuroimmune-metabolic-oxidative pathways. 2025;10.

13. Velasco G, Guevara ML, Triana-Vidal ML. Classical biomarkers and omics analysis in bipolar disorder. 2026:100906.

14. Charneca S, Hernando A, Costa-Reis P, Guerreiro CS. Beyond seasoning—the role of herbs and spices in rheumatic diseases. Nutrients. 2023;15(12):2812.

15. Zafrilla Rentero P, Ballester Navarro P, Victoria Montesinos D, Cerdá Martínez-Pujalte B, Marhuenda Hernández J, Arcusa Saura R, et al. Dietary bioactive compounds and their role in allergy prevention: a comprehensive review. 2025.

16. Qureshi U, Bajwa A, Aslam Z, Aggrey A, Nawaz UH, Ul Ain Q. Gut microbiota modulation in type 2 diabetes and cardiometabolic risk: a systematic review. Cureus. 2025;17(9):e92020.

17. Dymek A, Czerwonogrodzka-Senczyna A, Wyszyńska J, Wójcik M, Tarkowski B, Zalewska-Janowska AM, et al. Multidisciplinary management of psoriasis: integrating diet, exercise, psychological support, and sleep interventions. 2025:2951-70.

18. Gao H, He C, Xin S, Hua R, Du Y, Wang B, et al. In-depth analysis beneficial effect of probiotics and fatty acids in anesis of depression. 2023.

19. Saini A, Sharma B, Panghal A, Malik P. Eat to heal: the powerful connection between nutrition and mental health. J Altern Integr Med. 2026;26(1):176-91.

20. Raad T. Diet and rheumatoid arthritis: investigating the impact of a Mediterranean diet on patient-reported outcome measures. 2023.