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The Role of Omega-3 Fatty Acids in the Pathophysiology and Treatment of Migraine: An Updated Review of Clinical Evidence, Mechanistic Pathways and Nutritional Recommendations

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Abstract

Migraine is a complex neurological disorder in which neuroinflammatory processes, altered neuronal excitability, mitochondrial dysfunction, and changes within the immunological microenvironment play key roles in its pathophysiology. Omega-3 fatty acids, particularly EPA and DHA, exhibit potent anti-inflammatory, pro-resolving, and neuroprotective effects. This article provides a narrative review of the literature describing the mechanisms of action of omega-3 fatty acids and their clinical efficacy in studies related to migraine.

Background: Migraine is one of the most diagnosed neurological disorders and significantly impacts patients' quality of life. Current understanding highlights its neuro-immunological nature, involving complex interactions between neurons, glial cells, and inflammatory mediators. In recent years, increasing attention has been directed toward nutritional factors that may modulate the course of the disorder, including omega-3 fatty acids.

Aim: The aim of this article is to provide an expanded narrative review of the mechanisms of action of omega-3 fatty acids and to evaluate their clinical effectiveness in the context of migraine prevention and treatment.

Material and Methods: This review has a narrative character. The literature was searched in the PubMed, Scopus, and Web of Science databases for the years 2000- 2025, using keywords related to migraine and omega-3 fatty acids (including migraine, omega-3 fatty acids, EPA, DHA, resolvins, CGRP). Experimental studies, clinical trials, and reviews addressing the role

of EPA and DHA in the pathophysiology and treatment of migraine were included. Publications of low methodological quality or lacking complete data were excluded. The aim of this review was to provide a synthetic summary of current knowledge rather than to conduct a systematic review.

Results: The reviewed evidence indicates that omega-3 fatty acids, particularly EPA and DHA, influence several biological pathways relevant to migraine pathophysiology. Mechanistic studies consistently demonstrate their ability to modulate pro-inflammatory lipid mediators, enhance the production of pro-resolving molecules, stabilize neuronal membranes, improve mitochondrial efficiency, and reduce CGRP-related neurovascular activation. Clinical trials show heterogeneous but generally favourable outcomes, with the most pronounced benefits observed in interventions that include increased EPA intake combined with dietary reduction of omega-6 fatty acids and treatment durations of at least 12 weeks. Adjunctive therapies involving omega-3 fatty acids, such as combinations with valproate or metabolic cofactors, also suggest potential synergistic effects. Evidence in paediatric populations remains limited and inconsistent.

Conclusions: Omega-3 fatty acids represent a promising adjunctive strategy in migraine management, supported by mechanistic plausibility and growing clinical evidence. Their therapeutic potential appears strongly context-dependent, particularly on dietary omega-6 intake and treatment duration. EPA-rich formulations may offer enhanced anti-inflammatory and migraine-preventive effects, while DHA contributes to neuroprotection and neuronal stability. Although current findings are encouraging, larger and methodologically rigorous clinical trials are needed to establish optimal dosing, treatment protocols, and biomarkers that could guide personalized omega-3-based interventions in migraine care.

Key words: migraine; omega-3 fatty acids; EPA; DHA; neuroinflammation.

1. Introduction

Migraine is one of the most common neurological disorders worldwide and is a major contributor to reduced quality of life and disability, particularly among women of working age. Despite advances in understanding its biological foundations, migraine remains a condition with complex and multifactorial pathophysiology. Key processes involved include increased neuronal excitability, activation of the trigeminovascular system, chronic neuroinflammation, mitochondrial dysfunction, and alterations within the immunological microenvironment. The interplay of these mechanisms contributes to the initiation and perpetuation of migraine attacks, as well as their progression to chronic forms.

In recent years, increasing attention has been directed toward the role of dietary factors in modulating migraine susceptibility. Interest has focused on omega-3 polyunsaturated fatty acids-especially eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). These compounds exhibit strong anti-inflammatory, pro-resolving, and neuroprotective properties, and experimental evidence suggests that they may influence several key pathways involved in migraine pathophysiology, including modulation of lipid mediators, microglial activity, CGRP signalling, mitochondrial function, and neuronal membrane stability.

Given the growing interest in individualized therapeutic approaches and nutritional strategies in migraine prevention, an updated synthesis of the available scientific evidence is warranted. This article provides a narrative review of the mechanisms of action of omega-3 fatty acids and their clinical effectiveness and discusses the potential role of EPA and DHA as adjuncts in the prevention and treatment of migraine.

2.Research materials and methods

This review was narrative in nature. The literature search was conducted in the PubMed, Scopus, and Web of Science databases from January 2000 to December 2025. A combination of the following keywords and MeSH terms was used: “migraine”, “omega-3 fatty acids”, “EPA”, “DHA”, “resolvins”, “inflammation”, “CGRP”, “dietary modulation”, and “nutritional intervention”.

The analysis included original research articles, randomized clinical trials, observational studies, experimental studies, and systematic reviews addressing the role of omega-3 fatty acids in the

pathophysiology or treatment of migraine. Publications lacking clinical or mechanistic data, papers without full-text access, and studies deemed to be of low methodological quality were excluded.

The goal of this review was not to replicate the procedures of a systematic review but to provide a concise synthesis of current knowledge based on the most relevant and well-documented scientific sources.

3. Research results

The mechanisms through which omega-3 fatty acids exert their effects in the context of migraine are multifaceted and encompass modulation of inflammatory processes, alterations in membrane composition, stabilization of neuronal excitability, and regulation of neurotransmission. EPA and DHA display distinct yet complementary profiles of action, making them particularly relevant from the perspective of pain neurobiology.

3.1. Modulation of lipid mediators

One of the best-described mechanisms involves the competition between eicosapentaenoic acid (EPA) and arachidonic acid (AA) for cyclooxygenase (COX) and lipoxygenase (LOX) enzymes. AA serves as a substrate for the synthesis of pro-inflammatory eicosanoids, such as prostaglandin E2 (PGE2) and leukotriene B4 (LTB4), which intensify the neuroinflammatory milieu associated with migraine. Substituting AA with EPA leads to the formation of analogues with significantly weaker pro-inflammatory activity (e.g., PGE3, LTB5), thereby reducing sensitization of the trigeminal system.

3.2. Synthesis of pro-resolving mediators

EPA and DHA are precursors of specialized pro-resolving mediators (SPMs), including resolvins, protectins, and maresins. These compounds actively terminate inflammation and restore tissue homeostasis. In migraine, resolvins of the E and D series are of relevance, as they reduce microglial activation, decrease the release of pro-inflammatory cytokines, and limit the secretion of neuropeptides such as CGRP. This mechanism plays an essential role in extinguishing processes that initiate migraine attacks.

3.3. Stabilization of neuronal cell membranes

DHA is one of the most important structural lipids of neuronal membranes. Increased availability of DHA enhances membrane fluidity and flexibility, influencing the function of receptors, ion channels, and synaptic transmission. Improved membrane stability contributes to reduced neuronal hyperexcitability- one of the fundamental features of migraine pathophysiology, including the mechanism underlying cortical spreading depression (CSD).

3.4. Regulation of neuropeptides, including CGRP

Calcitonin Gene-Related Peptide (CGRP) is a key neuropeptide involved in vascular and inflammatory processes in migraine. Studies indicate that EPA and DHA supplementation may reduce CGRP levels both through direct modulation of neuronal membranes and through pro-resolving actions that mitigate neuroinflammation. This mechanism positions omega-3 fatty acids as a potentially useful adjunct to therapies targeting the CGRP pathway.

3.5. Effects on mitochondrial function

Mitochondrial dysfunction- particularly impairments in oxidative phosphorylation- is a crucial component of migraine. Omega-3 fatty acids, especially EPA, improve electron transport chain function, enhance ATP production, and reduce oxidative stress. DHA, in turn, stabilizes cardiolipin, a lipid essential for the activity of mitochondrial enzymes. These mechanisms reduce neuronal susceptibility to depolarization and lower the likelihood of triggering a migraine attack.

3.6. Regulation of the serotonergic system and neuroplasticity

Omega-3 fatty acids also influence serotonin metabolism, its transport, and the expression of serotonin receptors (e.g., 5-HT1B/1D). This is particularly important, as many antimigraine medications- such as triptans- act on this pathway. Additionally, DHA increases the expression of BDNF, supporting neuroplastic processes that may enhance neuronal resilience to migraine-related stressors and reduce attack frequency.

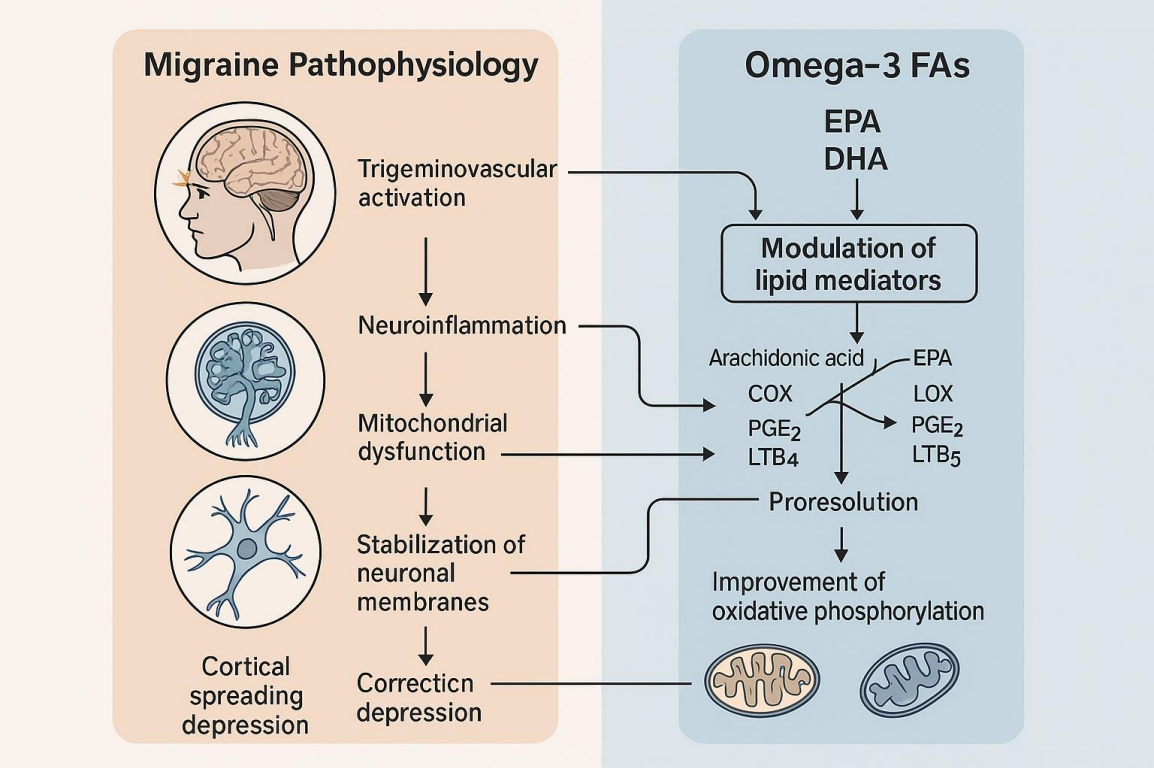

Figure 1. Schematic representation of the main mechanisms of migraine pathophysiology and the action points of omega-3 fatty acids, including modulation of lipid mediators, pro-resolution action, stabilization of neuronal membranes and improvement of mitochondrial function.

3.7. Review of Clinical Studies

Clinical studies examining the effects of omega-3 fatty acids on migraine include both early supplementation trials and more recent dietary intervention studies with high methodological rigor. Their findings are heterogeneous, reflecting differences in study design, EPA and DHA dosing, duration of the intervention, and the degree of dietary control among participants.

Author (Year)	Population / N	Intervention	Duration	Main Findings	Key Limitations
Ramsden et al., 2021	182 adults	Diet ↑omega-3 + ↓omega-6	16 weeks	Significant reduction in migraine days; decreased	Dietary changes difficult to fully control; use

			of acute medications	
Pradalier et al., 2001	56 adults	6 g fish oil	12 weeks	No significant effect	Lack of omega-6 intake control; low EPA-to-DHA ratio
Wang et al., 2024	88 adults	EPA 1.8 g/day	12 weeks	Reduction in pain and inflammatory markers	EPA monotherapy; no comparison with EPA/DHA combination
Ibrahim et al., 2025	60 adults	EPA + DHA + ALCAR + vitamin E	+12 weeks	40-50% reduction in attacks; improved oxidative stress markers	Combination therapy-difficult to isolate omega-3 effects
Fayyazi et al., 2016	45 children	Omega-3 valproate	+8 weeks	Mild improvement	Small sample size; no data on EPA/DHA levels
Tajmirriahi et al., 2012	80 adults	Omega-3 valproate	+12 weeks	Faster clinical response vs. monotherapy	Individual variability in drug and lipid metabolism
IJCN Study, 2021	100 adults	Omega-3 g/day	1.28 weeks	Moderate reduction in attack frequency	No detailed dietary information

Ramsden et al. (2021, BMJ)

The study by Ramsden is one of the most significant dietary intervention trials in the history of migraine research. It included 182 patients and involved simultaneous modification of fatty acid intake: increasing dietary EPA and DHA, and within one group, reducing omega-6 intake. The group with increased omega-3 intake and reduced omega-6 consumption showed the greatest reduction in migraine days, decreased use of acute medications, and improved quality of life. The findings clearly indicate that the effectiveness of omega-3 fatty acids is closely linked to changes in the omega-6/omega-3 ratio.

Pradalier et al. (2001)

One of the early supplementation trials, in which participants received 6 g of fish oil daily. The study did not demonstrate a significant difference between the supplemented group and placebo. However, methodological analysis reveals major limitations: lack of control over omega-6 intake, a low proportion of EPA relative to DHA, short supplementation duration, and insufficient dietary monitoring. Although frequently cited as evidence against the efficacy of omega-3s, the study's limitations substantially weaken its conclusions.

Wang et al. (2024)

In this more recent study, Wang and colleagues evaluated the effects of pure EPA at a dose of approximately 1.8 g per day. A clear reduction in pain intensity, inflammatory markers, and migraine frequency was observed. The results suggest that EPA may be the more biologically active omega-3 fatty acid in the context of migraine compared with DHA.

Combination therapies with valproate

Several studies, including Tajmirriahi (2012) and Fayyazi (2016), examined the combination of omega-3 fatty acids with valproate. In adults, this approach resulted in faster clinical response and reduced attack frequency, while findings in paediatric populations were less consistent. These discrepancies may reflect differences in metabolic profiles and the use of insufficient EPA doses in children.

Interventions involving antioxidants

In the study by Ibrahim et al. (2025), a combined therapeutic approach was used: EPA + DHA, acetyl-L-carnitine (ALCAR), and vitamin E. A 40-50% reduction in migraine attacks and improvements in oxidative stress markers were observed. These findings suggest that migraine may be strongly linked to neuronal energetic dysfunction, and metabolic interventions may hold therapeutic potential.

Paediatric studies

Research in children is limited and often yields inconclusive results. Contributing factors include short supplementation periods, small sample sizes, and inadequate monitoring of dietary fat intake. Optimal EPA and DHA doses for paediatric migraine remain unclear.

Systematic reviews

The most comprehensive reviews, such as Kumar et al. (2020), indicate moderate but consistent evidence supporting the benefits of EPA and DHA in migraine management. However, the conclusions are constrained by study heterogeneity, varying dosages, differing durations, and insufficient dietary control across trials.

Summary of findings

Overall, the analysis shows that the greatest therapeutic effects- supported by Ramsden (2021) and Wang (2024)- are achieved with interventions that:

1. increase EPA intake,
2. simultaneously reduce omega-6 intake,
3. last at least 12 weeks,
4. incorporate additional metabolic interventions.

This indicates that the effectiveness of omega-3 fatty acids in migraine is strongly context-dependent, shaped by dietary background and individual biological factors.

4. Discussion

Clinical and experimental data indicate that omega-3 fatty acids play a significant role in modulating pathophysiological processes associated with migraine. The complexity of these mechanisms stems from the involvement of multiple biological systems, including the immune, neuronal, mitochondrial, and vascular systems. EPA and DHA exert their effects through the reduction of neuroinflammatory lipid mediators, improvement of cellular membrane integrity, and stabilization of neuronal excitability. However, the clinical effectiveness of these fatty acids appears to be strongly dependent on dietary context, a finding particularly emphasized in the work of Ramsden and colleagues.

One of the most important observations is that omega-3 supplementation provides the greatest benefits when combined with reduced intake of omega-6 fatty acids. In individuals whose diets are high in linoleic acid- rich vegetable oils, EPA and DHA face limited ability to compete with arachidonic acid for key inflammatory enzymes such as COX and LOX. This explains why

several earlier studies reporting a lack of omega-3 efficacy cannot be interpreted independently of participants' dietary background.

Another crucial factor is the duration of supplementation. The incorporation of EPA and DHA into neuronal phospholipid membranes is a gradual process that may take several months. Consequently, short interventions- lasting only 4 to 8 weeks- may be insufficient to fully assess therapeutic potential. Studies extending to 12 weeks or longer have demonstrated more pronounced clinical effects.

The ratio of EPA to DHA is also an important consideration. Growing evidence suggests that EPA, due to its stronger anti-inflammatory activity, may play a dominant role in migraine prevention. In contrast, DHA contributes to neuroplasticity, neuronal membrane integrity, and neurotransmitter system function. The optimal EPA:DHA ratio in the context of migraine may be approximately 2:1 or even 3:1.

Despite promising findings, current research has several limitations, including small sample sizes, insufficient standardization of dosages, variability in supplement quality, and inconsistent outcome measures. Therefore, further well-designed randomized controlled trials are needed. Such studies should incorporate inflammatory, lipidomic, and mitochondrial biomarkers, as well as assessments of EPA and DHA levels in serum or erythrocyte membranes.

5. Conclusions

Available evidence indicates that omega-3 fatty acids, particularly EPA and DHA, may play an important role as an adjunct in migraine prophylaxis. Their effects are based on several well-documented biological mechanisms, including modulation of inflammatory mediators, synthesis of pro-resolving molecules, stabilization of neuronal membranes, and improvement of mitochondrial function. Clinical findings remain heterogeneous; however, the most promising outcomes are observed in interventions that increase EPA intake while simultaneously reducing omega-6 consumption and that last at least 12 weeks.

Although current results are encouraging, further well-designed clinical trials incorporating dietary control, standardized dosing, and evaluation of metabolic biomarkers are needed. Such studies would allow for more precise determination of optimal supplementation strategies and the potential for personalized omega-3-based therapy in migraine management.

Future research on the role of omega-3 fatty acids in migraine should address several key areas. First, large randomized clinical trials with extended duration and rigorous dietary monitoring are required. Second, biomarker-based approaches- such as measuring resolvin levels, plasma lipidomics, and mitochondrial parameters- should be integrated. Third, personalization of supplementation based on an individual's metabolic profile, including the omega-6/omega-3 ratio and genetic variants affecting fatty acid metabolism, is essential.

Another promising research direction involves the potential synergy between omega-3 therapy and CGRP-targeted medications, as well as the relationship between omega-3 supplementation, gut microbiota composition, and neuroinflammation. Growing evidence suggests that modulation of the microbiome may indirectly influence inflammatory processes within the nervous system, making this an especially compelling area for future investigation.

Disclosure

Author's contribution

Conceptualization: [KW], [PZ]
Methodology: [JM], [EC], [WW]
Check: [PZ], [JB], [KW]
Investigation: [JB], [IZ], [WW]
Data curation: [JM], [IZ], [PZ], [JB]
Writing - rough preparation: [IZ], [EC], [JM]
Writing - review and editing: [PZ], [EC]
Visualization: [JB], [KW], [IZ]
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Conflict Of Interest

The authors declare no conflict of interest.

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