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Effect of Omega-3 Fatty Acids on the Alveolar Bone – Literature Review

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Abstract

Objective

The aim of this review was to comprehensively evaluate the effects of omega-3 polyunsaturated fatty acids (ω-3 PUFAs), including eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), on alveolar bone metabolism across different dental conditions. Special emphasis was placed on their role in periodontitis, peri-implantitis, apical periodontitis, and orthodontic tooth movement.

Methods

A systematic search of PubMed and Scopus databases was conducted for publications from 2015 to 2025 using the keywords *"omega 3 alveolar bone"* and *"omega 3 orthodontic tooth movement"*. Out of 41 initially identified studies, 27 publications were included after applying inclusion and exclusion criteria. The selected studies consisted of in vivo and in vitro experimental models, clinical trials, and systematic reviews evaluating the effects of ω-3 PUFAs on alveolar bone remodeling, inflammation, and host immune responses.

Results

Omega-3 PUFAs demonstrated significant anti-inflammatory, antioxidant, and bone-preserving properties. In periodontitis, supplementation reduced pro-inflammatory cytokines (e.g., TNF-α, IL-1β, IL-6), increased IL-10 levels, inhibited osteoclastogenesis, and limited alveolar bone loss. Resolvin derivatives derived from ω-3 further enhanced periodontal healing and tissue regeneration. In peri-implantitis, resolvin D2 and DHA-loaded nanostructured lipid carriers effectively reduced inflammatory infiltration, modulated RANKL/OPG balance, and preserved peri-implant bone. In apical periodontitis, combined omega-3 supplementation and physical exercise decreased IL-17 and TNF-α expression, reduced osteoclast numbers, and improved collagen formation, suggesting enhanced periapical healing. In orthodontic contexts, ω-3 slowed tooth movement by decreasing osteoclastic activity and oxidative stress, while promoting osteoblastic activity and bone quality.

Conclusion

Omega-3 fatty acids represent promising adjunctive agents for preserving alveolar bone and modulating inflammatory responses in various dental pathologies. However, the majority of existing evidence is derived from preclinical studies. Well-designed, high-quality clinical trials are urgently needed to confirm efficacy, establish optimal dosages, and assess long-term safety and therapeutic potential in human populations.

***Keywords*— omega-3 fatty acids, alveolar bone, periodontitis, peri-implantitis, bone resorption**

1. INTRODUCTION

Alveolar bone plays a crucial role in maintaining dental structure, supporting the teeth, and facilitating functions such as mastication and speech. Bone integrity in this region is continuously challenged by local and systemic factors, particularly in the context of inflammatory oral diseases such as periodontitis, peri-implantitis, and apical periodontitis, as well as mechanical influences like orthodontic tooth movement. In these conditions, chronic inflammation and dysregulated bone remodeling lead to progressive bone loss, often resulting in tooth or implant failure.

Recent advances in nutritional and immunological sciences have drawn attention to the therapeutic potential of omega-3 polyunsaturated fatty acids (PUFAs), which can be found in dietary products such as fish and flaxoil [1], including eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), which exhibit notable anti-inflammatory, antioxidant, and pro-resolving properties [2, 3]. These fatty acids are precursors to specialized pro-resolving lipid mediators (SPMs) such as resolvins, protectins, and maresins, which have been shown to actively regulate the resolution phase of inflammation and facilitate tissue regeneration [4, 5]. [fig.1]

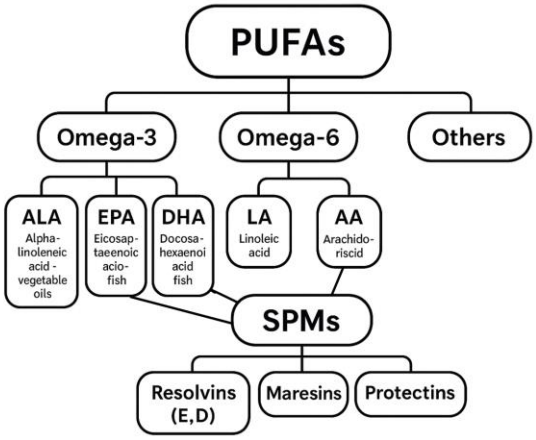

Figure 1 – Polyunsaturated Fatty Acids and Specialized Pro-resolving Lipid Mediators

In dental research, omega-3 PUFAs have demonstrated the ability to modulate host immune responses, inhibit osteoclastogenesis, and reduce inflammatory cytokine expression in periodontal and periapical tissues. Their role in

preserving alveolar bone has also been explored in the context of orthodontic treatment, where controlled bone resorption is essential.

Despite a growing number of studies in this field, there remains a need for a comprehensive synthesis of the evidence on how omega-3 fatty acids influence alveolar bone across various dental conditions. This review aims to consolidate current knowledge derived from preclinical and clinical studies, focusing on the effects of omega-3 PUFAs on alveolar bone remodeling in periodontitis, peri-implantitis, apical periodontitis, and orthodontic tooth movement.

2. METHODS

This review was conducted using the PubMed and Scopus databases. The search covered the years 2015–2025 and included the keywords: “omega 3 alveolar bone” and “omega 3 orthodontic tooth movement.” The first search returned 32 results, of which 7 were excluded due to irrelevance. The second yielded 9 articles, with 4 excluded for the same reason. Three of the remaining articles overlapped with the first group. In total, 27 publications were included in the review. These comprised experimental in vivo and in vitro studies, clinical trials, and systematic reviews focused on the effects of omega-3 fatty acids on alveolar bone metabolism in the context of periodontitis, peri-implantitis, and orthodontic tooth movement.

3. RESULTS

A. *The effect of omega-3 acids on periodontitis*

A substantial body of evidence supports the therapeutic benefits of omega-3 polyunsaturated fatty acids (PUFAs) in the prevention and management of periodontitis, a chronic inflammatory disease marked by progressive alveolar bone loss. Multiple preclinical and clinical studies have established the capacity of omega-3 PUFAs to attenuate inflammation, inhibit osteoclastic bone resorption [6, 7, 8] , and promote periodontal tissue homeostasis.

In rodent models of ligature-induced periodontitis, omega-3 supplementation consistently resulted in reduced alveolar bone loss, attributed to a marked suppression of pro-inflammatory cytokines such as TNF-α, IL-1β, IL-6 [9] and IL-17 [4], and an elevation of anti-inflammatory mediators like IL-10 [10, 11, 12, 13,14]. Doğan et al. demonstrated that combining omega-3 fatty acids with probiotics exerted a synergistic effect, leading to the most significant reduction in alveolar bone loss and favorable modulation of systemic inflammatory markers [11]. Similarly, González-Alva et al. reported that omega-3 PUFAs decreased tissue expression of MMP-2 and MMP-9—proteolytic enzymes that contribute to extracellular matrix degradation in periodontitis—thereby mitigating connective tissue breakdown [13].

Studies employing dietary interventions further supported these findings. Antona et al. found that rats with hypercholesterolemia subjected to periodontal disease exhibited substantial preservation of alveolar bone volume when their diet was modified to include n-3 PUFAs, highlighting the dual metabolic and periodontal benefits of such supplementation [15]. These results are aligned with earlier observations by Montalvany-Antonucci et al., who noted that omega-3-rich diets protect alveolar bone by positively influencing bone microarchitecture through anti-inflammatory and osteoanabolic effects [16].

Furthermore, specialized pro-resolving lipid mediators (SPMs), such as resolvins derived from omega-3 fatty acids, have emerged as key effectors in restoring periodontal homeostasis [17]. Studies have demonstrated that topical or systemic administration of RvE1 and RvD2 significantly reduced inflammatory cell infiltration, modulated cytokine profiles, and inhibited osteoclastogenesis in experimental models of periodontitis [18, 19, 20, 21, 1]. Resolvin D2 was shown to suppress Th1-driven immune responses and preserve osteoprotegerin levels while reducing RANKL expression, thus maintaining a bone-protective microenvironment [14]. Moreover, RvE1 enhanced regulatory T-cell populations in gingival tissues, promoting immune tolerance and long-term periodontal stability [20].

Human evidence, although still emerging, lends support to these preclinical observations. A systematic review by Wadia et al. revealed that the adjunctive use of omega-3 fatty acids with standard non-surgical periodontal therapy (scaling and root planing) led to greater reductions in probing pocket depth (PPD) and improvements in clinical attachment level (CAL), with a mean gain of 0.41 mm in CAL [22]. This is corroborated by data from Saleh et al., who conducted a retrospective analysis in a large dental data repository and found weak, though statistically significant, correlations between fish oil supplementation and reduced prevalence of periodontitis [23].

It is also worth noting that omega-3 PUFAs may counteract diet-induced exacerbation of periodontal disease. Choowong et al. emphasized that diets rich in saturated fats and refined carbohydrates exacerbate periodontal inflammation, whereas diets with a high omega-3 to omega-6 ratio confer a protective effect by reducing tissue destruction and inflammation [24]. Collectively, the evidence underscores the multifaceted protective roles of omega-3 fatty acids in modulating periodontal inflammation, supporting tissue regeneration, and preventing alveolar bone resorption.

B. The Effect of Omega-3 Fatty Acids on Peri-Implantitis

Peri-implantitis, characterized by inflammation of the peri-implant mucosa and progressive bone loss, shares pathological similarities with periodontitis but may exhibit a more aggressive progression due to distinct immunological responses and anatomical features. In murine models, resolvin D2 (RvD2) has been investigated as a therapeutic agent capable of modulating the excessive immune response associated with peri-implantitis. Experimental peri-implant infections induced by repeated oral inoculations with Porphyromonas gingivalis resulted in significant bone resorption and increased local infiltration of leukocytes. However, RvD2 administration effectively mitigated these effects, as evidenced by downregulation of IFN-α, IL-1β, and a favorable shift in the RANKL/OPG expression ratio [25].

Heyman et al. extended these findings by demonstrating that RvD2 also reduced leukocyte infiltration and local inflammatory burden in peri-implant tissues, supporting its role in resolving inflammation and preserving bone architecture [23]. Of note, these effects appeared to be more pronounced in peri-implant tissues compared to natural teeth, suggesting a heightened susceptibility of implant-associated bone to inflammatory destruction and a stronger response to therapeutic intervention.

An innovative approach to improve the bioavailability and therapeutic efficacy of DHA was explored through the use of nanostructured lipid carriers (NLCs). In a rat model of peri-implantitis, DHA-loaded NLCs demonstrated superior antioxidant and anti-inflammatory effects compared to unmodified DHA. This formulation inhibited the nuclear translocation of NF-κB p65 and effectively suppressed downstream inflammatory signaling in macrophages, resulting in reduced alveolar bone resorption [23]. These findings support the development of targeted omega-3 delivery systems as promising adjuncts for managing peri-implantitis.

C. The Effect of Omega-3 Fatty Acids on Apical Periodontitis

Apical periodontitis (AP) is a localized inflammatory condition arising from pulpal infection, leading to periapical tissue destruction and bone loss. The disease trajectory is largely determined by the host's immune and inflammatory response. In this context, omega-3 fatty acids have been shown to exert beneficial effects on bone preservation and inflammatory regulation.

In a rat model of AP, Ribeiro et al. demonstrated that the combination of physical exercise and omega-3 supplementation significantly modulated inflammatory markers and reduced osteoclastic activity. Omega-3 treatment led to decreased IL-17 and TNF-α expression, a reduction in the number of TRAP-positive osteoclasts, and enhanced formation of birefringent immature collagen fibers, which are indicative of early tissue repair [26]. Microtomographic analysis confirmed that alveolar bone loss was significantly lower in the omega-3 supplemented group.

Importantly, the combination of omega-3 and physical exercise appeared to exert a synergistic effect, suggesting that lifestyle factors may enhance the efficacy of nutritional interventions in AP management. These findings highlight the role of omega-3 fatty acids in modulating not only the inflammatory milieu but also promoting tissue regeneration in the periapical region.

D. The Effect of Omega-3 Fatty Acids on Orthodontic Tooth Movement

The process of orthodontic tooth movement (OTM) necessitates alveolar bone remodeling through tightly regulated osteoclast and osteoblast activity. Recent animal studies have examined the influence of omega-3 fatty acids on this process, focusing on their ability to alter the inflammatory and resorptive phases of bone remodeling.

Gad et al. observed that systemic omega-3 administration in rabbits undergoing OTM resulted in a significantly smaller amount of tooth displacement over a 21-day period compared to control animals. Histological analysis revealed reduced numbers of bone-resorptive lacunae and enhanced osteoblastic activity, indicating a shift toward bone preservation rather than resorption [27].

Similar findings were reported by Ogrenim et al., who demonstrated that omega-3 supplementation reduced the expression of RANKL, IL-1β, and IL-6 at multiple time points during OTM in rats. Additionally, the supplemented group exhibited significantly lower oxidative stress levels and higher antioxidant capacity, providing a favorable environment for controlled bone remodeling [28].

Mechanistic insights from Ma et al. confirmed that DHA suppresses TNF-α-induced osteoclastogenesis via activation of GPR120. In GPR120-deficient mice, the inhibitory effects of DHA on osteoclast formation and tooth movement were abolished, underscoring the pivotal role of this receptor in mediating omega-3's action [9,30]. The same signaling pathway was found to regulate RANKL expression in osteoblasts, further reinforcing omega-3's dual impact on bone cell communication during OTM [29].

While these findings suggest that omega-3 fatty acids could be strategically employed to limit excessive bone resorption and inflammation during orthodontic therapy, clinicians must also consider the potential delay in tooth movement and adjust treatment plans accordingly.

4. DISCUSSION

This review confirms that omega-3 fatty acids, especially EPA and DHA, play a protective and therapeutic role in preserving alveolar bone across a range of oral diseases. In periodontitis, omega-3s consistently reduce inflammation, suppress osteoclast activity, and limit bone resorption, largely via modulation of cytokines and the RANKL/OPG axis [10, 11, 13, 14]. Specialized pro-resolving mediators derived from omega-3s, such as resolvins, further enhance these effects by promoting inflammation resolution and tissue repair [18, 19, 20].

In peri-implantitis models, omega-3 compounds—especially resolvin D2—have demonstrated potential in preventing bone loss and dampening excessive immune responses around implants [25, 23]. Their use in optimized delivery systems may further improve therapeutic outcomes.

For apical periodontitis, although evidence is limited, omega-3 supplementation combined with physical exercise showed reduced inflammation and improved bone healing [26]. In orthodontics, omega-3s inhibited osteoclastogenesis and delayed tooth movement, which could be either a therapeutic benefit or a clinical limitation depending on treatment goals [27, 28, 30].

Despite encouraging findings, most studies are preclinical, with limited human data. Further well-designed clinical trials are needed to confirm efficacy, determine optimal dosing, and assess long-term outcomes. Omega-3 fatty acids hold promise as adjunctive agents in managing inflammatory oral diseases with alveolar bone involvement.

5. CONCLUSION

The current body of evidence strongly supports the role of omega-3 fatty acids as potent modulators of alveolar bone metabolism in a variety of dental pathologies. Through their anti-inflammatory and osteoprotective properties, omega-3 PUFAs contribute to the resolution of inflammation, inhibition of osteoclastogenesis, and promotion of bone regeneration.

In periodontitis, omega-3s demonstrate consistent efficacy in reducing alveolar bone loss, both as monotherapy and in conjunction with standard periodontal treatments. In peri-implantitis, emerging data suggest that resolvin-based therapies may offer a novel, targeted approach to limiting implant-related bone destruction. In apical periodontitis, omega-3s modulate the host response and facilitate periapical healing. In orthodontics, their capacity to decelerate tooth movement underscores a dual potential for harm (treatment delay) and benefit (bone preservation)

While these findings are compelling, they are primarily grounded in preclinical research. Therefore, further clinical investigations are essential to validate these results in human populations, optimize administration protocols, and determine long-term outcomes. If corroborated, omega-3 fatty acids could be integrated as adjunctive or preventive agents in comprehensive oral health management, particularly in inflammation-driven alveolar bone diseases.

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