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Interleukin-1β–Mediated Orofacial Bone Defects and Alveolar Bone Loss in Periodontal Disease: A Scoping Review

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ABSTRACT: Orofacial bone defects, particularly alveolar bone loss, are hallmark features of periodontal disease and represent a significant clinical challenge affecting oral function and quality of life. Interleukin-1β (IL-1β) has emerged as a key pro-inflammatory cytokine mediating the link between periodontal inflammation and osteoclastic bone resorption. This scoping review using keywords related to orofacial bone defects, alveolar bone loss, and IL-1β, published between 2016 and 2026. The studies included experimental models, animal studies, and clinical observations. Results consistently indicate that ligature induced periodontitis and microbial dysbiosis upregulate IL-1β expression, correlating with enhanced alveolar bone resorption. Interventions such as desipramine, epigallocatechin-3-gallate (EGCG), curcumin, and four-day fermented milk kefir demonstrated reductions in IL-1β levels alongside attenuated alveolar bone loss, highlighting the functional role of IL-1β in periodontal bone destruction. Immune modulation studies, including TLR-activated B10 cell transfer and Ifi204-deficient mouse models, further suggest that IL-1β–mediated osteoclast activity contributes directly to alveolar bone resorption independently of certain bone marrow–derived immune cell responses. Mechanistically, IL-1β promotes osteoclastogenesis via upregulation of RANKL and matrix metalloproteinases while downregulating tissue inhibitors of metalloproteinases. Collectively, the findings underscore IL-1β as a pivotal mediator in periodontitis-related bone loss and as a promising therapeutic target. Understanding these IL-1β mediated pathways informs the development of novel anti-inflammatory and regenerative strategies to improve oral and dental health outcomes.

KEYWORDS: Orofacial bone defect, interleukin-1β, periodontal disease, cytokine

INTRODUCTION

Bone is a complex hard tissue primarily composed of collagen fibrils and biomineral nanoparticles. As the only mineralized tissue capable of continuous remodeling, bone initiates new formation through the development of an initial structure known as the cement line, which serves as the foundation for subsequent bone growth (Grünewald,et al. 2023). Histologically, bone is classified as a highly specialized connective tissue that possesses the ability to adapt and undergo remodeling in response to external and mechanical demands. Bone formation involves specialized cells known as osteoblasts, which are responsible for synthesizing and secreting osteoid, an organic collagen-rich matrix. The osteoid contains a ground substance composed of osteocalcin and chondroitin sulfate (Cowan, et al. 2024). Bone plays essential roles in the body, including supporting movement, protecting and providing structural support to soft tissues, serving as a reservoir for calcium and phosphate, and housing the bone marrow (Florencio-Silva, et al. 2015). Bone loss or damage, referred to as a bone defect, in the dental context denotes the reduction or destruction of the alveolar bone that functions as the supporting structure for teeth. The alveolar bone plays a critical role in maintaining tooth support as well as preserving masticatory function, phonation, and facial aesthetics. Orofacial bone defects may arise from various factors, including periodontal inflammation, dentoalveolar trauma, infection, developmental abnormalities, and surgical interventions. Conditions commonly associated with bone defects include periodontal disease, congenital anomalies such as cleft lip and palate, and clinical procedures such as tooth extraction, removal of odontogenic cysts or tumors, and implant therapy. If not managed appropriately, bone defects may result in tooth mobility, tooth loss, implant treatment failure, and a subsequent decline in patients' quality of life.

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Alveolar bone defect is a hallmark feature of periodontitis and represents one of the most common manifestations of chronic inflammatory disease affecting the oral cavity. In periodontitis, a dysbiotic microbial biofilm accumulates on the tooth root surface, triggering a host-mediated inflammatory response that becomes pathogenic and leads to progressive destruction of the tooth-supporting structures. This process results in alveolar bone resorption, loss of connective tissue attachment, and degradation of tooth-associated cementum, ultimately causing tooth mobility and tooth loss in advanced stage (Ikeuchi, T. and Moutsopoulos, N.M, 2022).

Under healthy conditions, teeth are firmly anchored within the alveolar bone, which provides structural support and stability. The alveolar bone is connected to the tooth root surface, covered by cementum, through dense connective tissue fibers known as the periodontal ligament. Physiologically, the alveolar bone supports most of the tooth root, with the level of bone attachment terminating approximately 1–2 mm apical to the clinical crown, defined as the portion of the tooth located above the gingival margin. Bone defects in the oral and maxillofacial region represent frequent clinical challenges that substantially impair masticatory function, facial aesthetics, and overall quality of life. A wide range of biomaterials has therefore been employed to support the repair of these defects. Bone tissue engineering combines biomaterial scaffolds, cellular components, and bioactive molecules to create biomimetic constructs aimed at promoting bone regeneration (Koons et al., 2020). Advances in additive manufacturing technologies, along with topographical, chemical, and biochemical surface modifications, have further improved the osteogenic potential of tissue-engineered constructs (Zhang et al., 2019; 2020).

Biologically, the development of alveolar bone defects is not merely a mechanical consequence of hard tissue loss but also involves complex inflammatory and immune responses. Inflammation represents an essential initial component of bone remodeling and healing; however, excessive or prolonged inflammation may exacerbate bone destruction. Among proinflammatory mediators, one of the is interleukin-1β (IL-1β) plays a central role in periodontal inflammation and alveolar bone resorption by promoting osteoclastogenesis and disrupting bone homeostasis. Despite extensive experimental and clinical evidence highlighting the involvement of IL-1β in periodontal bone loss, the available data remain scattered and have not been systematically integrated within the context of orofacial bone defects.

Therefore, this scoping review aims to map the available evidence regarding the role of interleukin-1β (IL-1β) in the pathogenesis of orofacial bone defects, particularly alveolar bone loss associated with periodontal disease, and to discuss its relevance to oral and dental health. This review is intended to provide an integrated overview of IL-1β-mediated molecular mechanisms involved in bone destruction and to highlight potential implications for future therapeutic and regenerative strategies in dental research and practice.

MATERIALS AND METHODS (OR METHODOLOGY)

This study was conducted as a scoping review to map the available evidence regarding the role of interleukin-1β (IL-1β) in orofacial bone defects, particularly alveolar bone loss associated with periodontal disease. A literature search was performed using the PubMed and Science Direct database to identify relevant peer-reviewed articles. The search strategy employed predefined keywords, including “alveolar bone loss,” “IL-1β,” “IL-6,” “TNF-α,” and “periodontal bone defect.” Articles published in English between 2016 and 2026 were considered eligible. After removal of duplicate records, titles and abstracts were screened based on predefined inclusion and exclusion criteria. Original research articles investigating inflammatory mechanisms related to periodontal or orofacial bone loss were included, while review articles, book chapters, and conference proceedings were excluded to avoid duplication of primary data. Relevant information extracted from the included studies comprised author names, publication year, study design, and key findings related to IL-1β–mediated mechanisms. The study selection process is summarized in Figure 1

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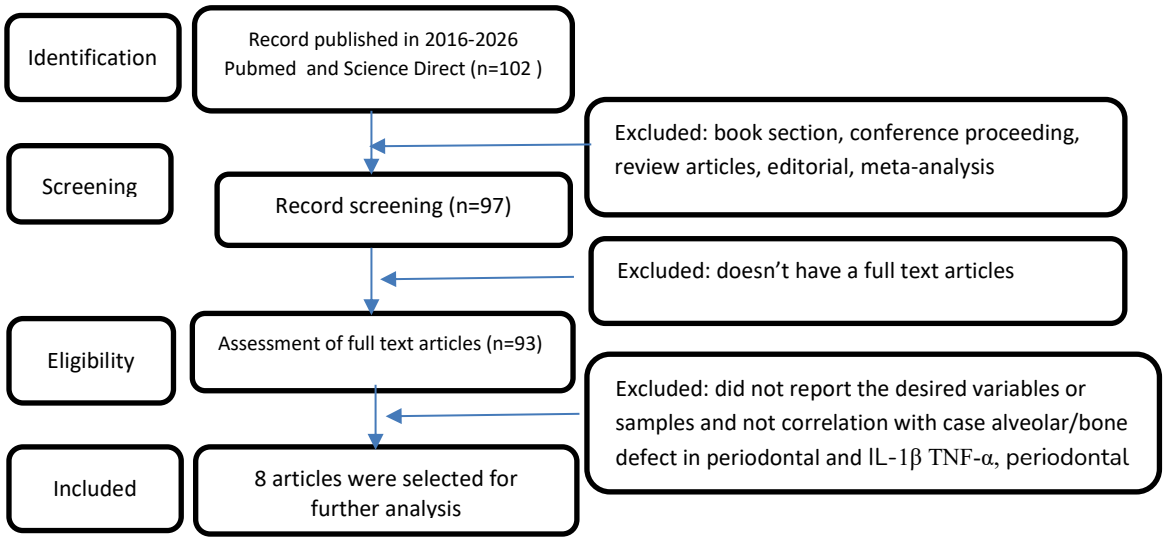

Figure 1. The article selection process flow diagram

RESULTS

A total of eight studies met the eligibility criteria and were included in this review. The main characteristics of the included studies are summarized in Table 1.

Table 1. Data extracted from included studies

No	Authors/ year	Title	Methods	Result
1	Hong Yu, Chao Sun, Kelley M. Argraves	Periodontal inflammation and alveolar bone loss induced by Aggregatibacter actinomycetemcomitans is attenuated in sphingosine kinase 1-deficient mice	Animal model, RNA extraction and real time PCR, ELISA, Histological tissue, and other molecular methods	IL-1β–associated inflammatory responses were not significantly altered in SK1-deficient conditions, yet reduced IL-1β–related osteoclast activity was accompanied by attenuated alveolar bone loss.
2	Luciana S. Branco-de-Almeida; Gilson C. N. Franco; Myrella L. Castro; Mayana S. Vieira; Leonardo V. Galvão-Moreira; Sheila C. Cortelli; Ana L. Anbinder; Toshihisa	Protective effects of desipramine on alveolar bone in experimental periodontitis	Animal model, PCR, zymography	Ligature-induced periodontitis caused significant alveolar bone loss accompanied by increased IL-1β expression, whereas desipramine treatment reduced bone loss and significantly downregulated IL-1β mRNA levels compared with the ligature group

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	Kawai; Pedro L. Rosalen			
3	Alexandra Cloitre, Boris Halgand, Sophie Sourice, JocelyneCaillon, Olivier Huck, Isaac Maximiliano Bugueno, Fareeha Batool, Jérôme Guicheux, Valérie Geoffroy, & Philippe Lesclous	IL-36γ is a pivotal inflammatory player in periodontitis-associated bone loss	Cell line: human OKF6/ TERT2 OEC, P. gingivalis, RNA extraction and RT-qPCR analyses, immunohistochemistry, western blot	In periodontitis, IL-1β–mediated inflammatory responses are associated with increased alveolar bone loss, as elevated IL-36γ expression correlates with a higher RANKL/OPG ratio and promotes IL-1β expression, thereby contributing to enhanced bone resorption
4	Yuhua Wang, Xiaoqian Yu, Jiang Lin, Yang Hu, Qian Zhao, Toshihisa Kawai, Martin A. Taubman, Xiaozhe Han	B10 Cells Alleviate Periodontal Bone Loss in Experimental Periodontitis	Animal model, real time PCR, Flow cytometry analysis, Measurements of bone resorption	In experimental periodontitis, adoptive transfer of TLR-activated B10 cells reduced periodontal bone loss, which was accompanied by significant suppression of gingival IL-1β expression and a decreased RANKL/OPG ratio.
5	Karen V Swanson* , Mustafa Girnary†, Tomaz Alves, et al	Interferon activated gene 204 (Ifi204) protects against bone loss in experimental periodontitis	Animal model, Real-Time PCR analysis for gene expression, Immunohistochemical analysis, Flow cytometry analysis	Although enhanced periodontal bone loss is often linked to an exaggerated IL-1β response from immune cells, bone marrow–chimera experiments showed that the increased alveolar bone loss observed in Ifi204-deficient mice was independent of bone marrow–derived IL-1β–producing immune cells
6	Qin Fan, Xiao-Hong Zhou, Teng-Fei Wan, et al.	Effects of epigallocatechin-3-gallate on oxidative stress, inflammation, and bone loss in a rat periodontitis model	Animal model, enzyme-linked immunosorbent assay (ELISA) method	EGCG at an optimal dose of 200 mg/kg significantly reduced alveolar bone loss in experimental periodontitis, which was accompanied by a marked decrease in IL-1β expression compared with untreated periodontitis.
7	Aram Mohammed Sha, Balkees Taha Garib, Shokhan	Effects of curcumin gel on osteoclastogenic bone markers in experimental periodontitis and	Animal model, ELISA, H&E staining	Curcumin treatment in experimental periodontitis significantly reduced serum IL-1β levels, indicating its anti-inflammatory effect in

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	Hamaali Azeez, Sarhang Sarwat Gul	alveolar bone loss in wistar rats		suppressing periodontal inflammation.
8	Lorena Vasconcelos Vieira, Luane Macedo de Sousa, Thays Allane Cordeiro Maia, et al	Milk Kefir therapy reduces inflammation and alveolar bone loss on periodontitis in rats	Animal model, Immunohistochemistry	Four-day fermented milk kefir significantly reduced alveolar bone loss in experimental periodontitis, accompanied by a marked decrease in IL-1β expression in periodontal tissues.

DISCUSSION

The data summarized in Table 1 indicate that alveolar bone loss associated with periodontal disease constitutes a complex pathological process in which inflammatory responses and bone remodelling are closely interrelated. Elucidating the molecular mediators that bridge inflammatory signalling and osteoclastic bone resorption is therefore essential for understanding the mechanisms underlying periodontal bone destruction. The findings synthesized in this review consistently demonstrate that interleukin-1β (IL-1β) plays a pivotal role in this process, acting as a key mediator linking periodontal inflammation to alveolar bone loss. Interleukin-1β, a pro inflammatory cytokine, is markedly elevated in periodontitis and actively contributes to disease progression. Clinical evidence has not only established a strong association between IL-1β and periodontitis, but has also shown that increased IL-1β levels initiate a cascade of inflammatory events that promote bone resorption (Cheng et al., 2020).

Evidence from multiple experimental models further supports the involvement of IL-1β in periodontal bone destruction. In ligature-induced periodontitis models, which closely mimic plaque-induced periodontal inflammation, significant alveolar bone loss is consistently accompanied by upregulated IL-1β expression. These observations support the concept that IL-1β functions not merely as a secondary inflammatory marker but as an active driver of periodontal tissue breakdown. Notably, therapeutic interventions shown to attenuate alveolar bone loss such as desipramine, epigallocatechin gallate (EGCG), curcumin, and fermented milk kefir—have also been associated with concomitant reductions in IL-1β expression, thereby reinforcing the causal relationship between IL-1β signalling and periodontal bone resorption. With respect to its effects on bone metabolism, IL-1β belongs to a group of potent osteotropic cytokines, including IL-6, Tumor Necrosis Factor-α (TNF-α), and Transforming Growth Factor-β (TGF-β), which exert diverse and context-dependent regulatory effects on bone cells. Cytokine production by osteoblasts, as well as their induction by bacterial components such as lipopolysaccharides, is regulated by intricate hormonal and cytokine networks (Bord et al., 1996). Within the periodontal microenvironment, IL-1β promotes osteoclastic bone resorption by upregulating matrix metalloproteinase expression while simultaneously suppressing the production of tissue inhibitors of metalloproteinases. Substantial experimental evidence has therefore established IL-1β as a major contributor to inflammation-induced bone resorption (Deshpande et al., 2013). Importantly, several studies included in this review indicate that the relationship between IL-1β and alveolar bone loss is not solely determined by cytokine expression levels. Under sphingosine kinase 1 (SK1) deficient conditions, IL-1β associated inflammatory responses were not markedly altered; however, reduced IL-1β related osteoclast activity was accompanied by a significant attenuation of alveolar bone loss. Similarly, bone marrow chimera experiments demonstrated that the increased alveolar bone loss observed in Ifi204-deficient mice occurred independently of bone marrow derived IL-1β producing immune cells. These findings suggest that IL-1β–mediated bone resorption is influenced not only by cytokine abundance but also by downstream signalling pathways and the local cellular responsiveness within periodontal tissues.

The mechanistic link between IL-1β driven inflammation and osteoclastogenesis is further supported by evidence implicating the receptor activator of nuclear factor κ-B ligand (RANKL) and osteoprotegerin (OPG) axis. Host cells recognize pathogen-associated molecular patterns, including lipopolysaccharides, through toll-like receptors, leading to the release of pro-inflammatory mediators such as IL-1, IL-6, TNF-α, IL-17A, and RANKL. These mediators sustain periodontal inflammation and contribute to alveolar bone resorption through both direct and indirect mechanisms. Elevated expression of IL-36γ has been shown to correlate with increased IL-1β levels and an elevated RANKL/OPG ratio, thereby promoting osteoclast differentiation and bone resorption. Conversely,

adoptive transfer of toll-like receptor activated B10 cells has been demonstrated to suppress gingival IL-1β expression, reduce the RANKL/OPG ratio, and significantly attenuate periodontal bone loss.

Collectively, the findings reviewed here highlight IL-1β as a central regulator of inflammation-induced alveolar bone loss in experimental models of periodontitis. IL-1β functions not only as a key inflammatory mediator but also as an upstream regulator of osteoclastogenesis through modulation of the RANKL/OPG axis. Importantly, experimental evidence suggests that targeting IL-1β signaling either by suppressing its expression or by modulating downstream pathways can effectively attenuate alveolar bone destruction. However, further well designed clinical and translational studies are required to validate these mechanisms in human periodontal disease and to determine the therapeutic relevance of IL-1β targeted interventions in clinical practice.

CONCLUSION

Collectively, the evidence summarized in this review demonstrates that interleukin-1β (IL-1β) plays a pivotal role in the pathogenesis of periodontal disease–associated alveolar bone loss. IL-1β acts as a key mediator linking periodontal inflammation to osteoclastic bone resorption by promoting osteoclast activity and modulating critical pathways such as the RANKL/OPG axis. Experimental models consistently show that increased IL-1β expression is associated with enhanced alveolar bone destruction, whereas therapeutic or biological interventions that suppress IL-1β signaling effectively attenuate bone loss. Importantly, IL-1β mediated bone resorption is influenced not only by immune cell–derived cytokine production but also by local tissue responses and downstream signaling mechanisms within periodontal tissues.

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