International Journal of Science and Research Archive
eISSN: 2582-8185
Cross Ref DOI: 10.30574/ijsra
Journal homepage: https://ijsra.net/



(REVIEW ARTICLE)

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ALVEOLAR BONE IN HEALTH

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International Journal of Science and Research Archive, 2026, 21(02), 872–877

Publication history: Received on 09 October 2026; revised on 14 November 2026; accepted on 17 November 2026

Article DOI: https://doi.org/10.30574/ijsra.2026.21.2.2035

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ABSTRACT

Periodontium Unlike many other skeletal sites, its development, architecture and lifelong turnover are closely linked to tooth eruption, periodontal ligament (PDL) function and occlusal loading. In health, alveolar bone is maintained through a coordinated balance between osteoblast-mediated formation and osteoclast-mediated resorption, with osteocytes acting as mechanosensors and regulators of local cellular activity. The alveolar process comprises alveolar bone proper, supporting cortical plates and cancellous bone. Its specialised socket-facing surface provides attachment for PDL collagen fibre bundles, while its vascular, marrow and cellular compartments support continuous adaptation. Development is principally intramembranous and involves dental follicle-derived progenitor populations and tightly regulated epithelial–mesenchymal signalling. At the molecular level, RANK–RANKL–OPG, Wnt/β-catenin, BMP, TGF-β, Hedgehog and mechanosensitive pathways contribute to bone homeostasis. Physiological occlusal forces are not merely tolerated: they help preserve alveolar bone mass and regulate progenitor-cell activity. This review summarises the normal anatomy, development, cellular biology, remodelling mechanisms and clinical relevance of healthy alveolar bone, with emphasis on evidence from original experimental and observational studies.

Keywords: Alveolar Bone; Periodontal Ligament; Bone Remodelling; Osteoblast; Osteoclast; Osteocyte; Wnt Signalling; Occlusal Force; Periodontium.

1 INTRODUCTION

Alveolar bone is the specialised portion of the jaw bones that forms and supports the tooth sockets. Together with cementum, the PDL and gingiva, it constitutes the periodontium. Its primary functions are to anchor teeth, distribute and dissipate forces generated during mastication, maintain the spatial relationship of teeth within the arches and provide a mineralised interface for periodontal attachment. It also participates in calcium and phosphate storage and contains marrow and vascular spaces. Alveolar bone is distinctive because its form and metabolism are closely associated with the presence and function of teeth. Experimental work and developmental studies show that dental follicle-derived cells contribute to the formation of alveolar bone, while PDL–bone signalling helps maintain the tooth-supporting complex [1–4].

Healthy alveolar bone should therefore be understood as a dynamic tissue rather than a static socket wall. Its architecture is continuously adjusted in response to physiological loading, tooth eruption and drift, and normal tissue turnover. The central principle is coupling: bone resorption and formation occur in a coordinated sequence so that local structural adaptation does not become net tissue loss. This review focuses on normal physiology; disease-associated

inflammatory bone destruction is mentioned only where it clarifies the distinction between physiological and pathological remodelling.

2 ANATOMICAL ORGANISATION AND HISTOLOGICAL FEATURES

2.1 Components of the alveolar process

The alveolar process is commonly described as three interrelated components: (i) alveolar bone proper, which lines the tooth socket; (ii) supporting cortical bone, forming the buccal and lingual/palatal plates; and (iii) supporting cancellous or trabecular bone, located between the cortical plates and the socket walls. The relative thickness of these components varies with jaw region, tooth position, root morphology, age and functional loading. The cortical plates are often thinner in the anterior maxilla and mandibular anterior region than in posterior regions, although substantial individual variation exists [5].

2.2 Alveolar bone proper

Alveolar bone proper is the thin layer directly adjacent to the PDL. In histological sections, it may appear as bundle bone because bundles of Sharpey's fibres from the PDL become embedded within it. Radiographically, the socket wall is represented by the lamina dura, a radiopaque line whose appearance depends on projection geometry and local anatomy. The alveolar bone proper contains perforating vascular channels and is remodelled in close relation to PDL function. It is not simply a passive barrier: it is the mineralised attachment site through which tooth-generated forces are transferred to the supporting jaw [5,6].

2.3 Supporting cortical and cancellous bone

The outer cortical plates provide structural rigidity, whereas cancellous bone contains trabeculae separated by marrow spaces. Trabecular orientation and thickness reflect local functional demands. The cancellous compartment contributes to metabolic exchange and contains marrow-derived cellular populations. The alveolar crest forms the coronal boundary of the supporting alveolar housing and is continuous with the socket wall. The architecture is therefore regionally adapted rather than uniform.

2.4 Matrix and mineral composition

Like other mineralised connective tissues, alveolar bone consists of an organic matrix and an inorganic mineral phase. Type I collagen is the predominant organic matrix protein, providing tensile strength and a scaffold for mineral deposition. Non-collagenous proteins—including osteocalcin, osteopontin, bone sialoprotein and osteonectin—participate in matrix organisation, mineralisation and cell–matrix interactions. The mineral phase is primarily carbonated hydroxyapatite. The combination of collagen and mineral gives bone its characteristic balance of toughness and stiffness.

3 DEVELOPMENT AND FORMATION OF ALVEOLAR BONE

Alveolar bone develops in close association with tooth development and root formation. Neural crest-derived ectomesenchyme contributes to the dental follicle, which gives rise to the supporting periodontal tissues, including cementum, PDL and alveolar bone. Much of the alveolar process forms by intramembranous ossification, in which mesenchymal progenitors differentiate directly into osteoblasts without a cartilage template. Classic developmental work traced the origin of alveolar bone to the developing periodontium [1].

Development is regulated by reciprocal epithelial–mesenchymal interactions and signalling networks. In experimental models, the Msx1/BMP4 pathway was shown to regulate alveolar bone formation through downstream osteogenic transcription factors including Dlx5 and Cbfa1/Runx2 [2]. TGF-β receptor II signalling is also important for the proliferation and differentiation of osteogenic progenitors during postnatal alveolar bone formation [3]. More recent lineage-tracing work has identified PTHrP-expressing dental follicle populations and shown that Hedgehog–Foxf signalling must be appropriately timed for these cells to adopt alveolar osteoblast and ligament lineages [4].

These findings support a key developmental concept: alveolar bone is not formed independently of the tooth. Its emergence and maintenance are coordinated with root development, eruption and establishment of the PDL attachment apparatus. The developing socket is progressively shaped around the erupting tooth, and subsequent functional loading contributes to maturation and adaptation of the supporting bone.

4 CELLULAR COMPONENTS OF HEALTHY ALVEOLAR BONE

4.1 Osteoblasts

Osteoblasts are matrix-forming cells derived from mesenchymal progenitors. They synthesise osteoid, a collagen-rich organic matrix, and regulate its mineralisation through alkaline phosphatase and other matrix-associated proteins. Osteoblast differentiation is governed by transcriptional regulators such as RUNX2 and SP7/osterix and by signalling pathways including BMP and Wnt/β-catenin. Some osteoblasts become embedded in the matrix and differentiate into osteocytes; others become bone-lining cells or undergo apoptosis. Osteoblasts also regulate osteoclastogenesis by expressing RANKL and its decoy receptor osteoprotegerin (OPG), thereby coupling formation and resorption [7,8].

4.2 Osteocytes

Osteocytes are mature bone cells embedded in lacunae within the mineralised matrix. Their long cellular processes extend through canaliculi, forming a communication network that senses changes in fluid flow and mechanical strain. Osteocytes regulate bone adaptation by producing signalling molecules such as sclerostin, RANKL and OPG. In alveolar bone, osteocytes are particularly important because the tissue is exposed to repeated occlusal loading. Experimental evidence demonstrates that osteocytes can regulate periodontal progenitor-cell activity through sclerostin-mediated inhibition of Wnt signalling, and that this feedback is influenced by occlusal force [9].

4.3 Osteoclasts

Osteoclasts are multinucleated cells derived from the monocyte–macrophage lineage. They attach to mineralised surfaces, establish a sealed resorption compartment and dissolve mineral using acid while degrading organic matrix with proteolytic enzymes, including cathepsin K. Osteoclast differentiation requires macrophage colony-stimulating factor (M-CSF) and RANKL signalling through RANK. OPG acts as a soluble decoy receptor that binds RANKL and limits osteoclast formation. In health, osteoclast activity is spatially and temporally regulated and is followed by osteoblast recruitment and new matrix deposition [7,10].

4.4 Progenitor and periodontal ligament cells

Mesenchymal progenitor populations contribute to routine turnover and repair. PDL-derived stem/progenitor cells can generate multiple periodontal cell types, including cells contributing to alveolar bone and cementum. Lineage-tracing studies identified Gli1-positive PDL cells as multipotent cells involved in periodontal tissue turnover and injury repair [9]. Other work has demonstrated osteogenic potential in periodontal ligament cell populations and the contribution of PDL cells to alveolar bone repair [11,12]. The PDL therefore functions as more than a fibrous suspensory ligament; it is an active cellular partner in alveolar bone homeostasis.

5 PHYSIOLOGICAL BONE REMODELLING AND HOMEOSTASIS

Bone remodelling is the coupled replacement of old or microdamaged bone by newly formed bone. A typical remodelling sequence includes activation, resorption, reversal, formation and mineralisation. Osteoclasts first remove a small volume of bone; mononuclear reversal cells prepare the surface; osteoblast-lineage cells then deposit osteoid, which subsequently mineralises. In a healthy adult, the amount of bone formed is broadly balanced with the amount resorbed over time. Modelling, in contrast, changes bone size or shape through formation and resorption occurring on different surfaces and need not be coupled at the same site.

Alveolar bone turnover is influenced by local periodontal signals and systemic regulators, including parathyroid hormone, vitamin D, calcitonin and sex steroids. However, the local tooth–PDL–bone unit provides a distinctive regulatory environment. Genetic manipulation of Wnt signalling in the periodontal complex alters alveolar bone turnover and PDL width, demonstrating that Wnt activity is required for normal homeostatic relationships [8,13]. The balance between RANKL and OPG is another central control point: increased RANKL availability favours osteoclastogenesis, whereas OPG limits this process.

6 MOLECULAR PATHWAYS REGULATING HEALTHY ALVEOLAR BONE

6.1 RANK–RANKL–OPG axis

RANK is expressed on osteoclast precursors, while RANKL is produced by osteoblast-lineage cells, osteocytes and other local cells. Binding of RANKL to RANK activates intracellular signalling required for osteoclast differentiation, activation and survival. OPG competes for RANKL and prevents its interaction with RANK. The RANKL/OPG balance therefore acts

as a molecular rheostat controlling physiological resorption. Healthy bone requires sufficient osteoclast activity for turnover, but not unrestrained activation.

6.2 Wnt/β-catenin signalling

Canonical Wnt signalling promotes osteoblast differentiation and activity. Wnt ligands bind Frizzled receptors and LRP5/6 co-receptors, stabilising β-catenin and allowing it to regulate target-gene transcription. Sclerostin and DKK proteins inhibit this pathway. In periodontal tissues, experimental Wnt activation and inhibition have shown that Wnt signalling controls alveolar bone turnover and helps maintain PDL width [8,13]. Osteocyte-derived sclerostin provides a feedback mechanism linking mechanical loading to progenitor-cell activity [9].

6.3 BMP and TGF-β signalling

BMP signalling supports osteogenic commitment and maturation, in part through RUNX2 and SP7. Developmental experiments have implicated Msx1/BMP4 signalling in alveolar bone formation [2]. TGF-β signalling regulates progenitor proliferation, matrix production and differentiation; disruption of TGF-β receptor II in osteogenic progenitors impairs postnatal alveolar bone formation [3]. These pathways interact with Wnt and other signals rather than functioning as isolated switches.

6.4 Mechanosensitive pathways

Mechanical forces generated during mastication are transmitted through the PDL to alveolar bone. Cells detect these forces through integrins, cytoskeletal structures, ion channels and primary cilia. Piezo1 is a mechanically activated ion channel implicated in force sensing. In mice, occlusal force was shown to support alveolar bone homeostasis through Piezo1-dependent osteogenic activity and regulation of osteoclast survival [14]. Piezo1 in PDL cells has also been implicated in force-associated alveolar remodelling [15]. These findings provide a cellular explanation for the importance of maintaining normal occlusal function.

7 RELATIONSHIP BETWEEN PERIODONTAL LIGAMENT AND ALVEOLAR BONE

The PDL occupies the space between cementum and alveolar bone proper and contains collagen fibre bundles, fibroblasts, vessels, nerves and progenitor cells. Its principal fibre groups resist intrusive, extrusive, tipping and rotational components of tooth movement. The ends of principal collagen bundles become embedded in cementum and bundle bone as Sharpey's fibres. This attachment system distributes functional forces over the socket wall and helps prevent direct tooth–bone contact.

PDL cells communicate with bone cells through soluble mediators, extracellular vesicles and direct mechanical interactions. PDL-derived progenitors can contribute to new alveolar bone, while osteocytes in adjacent bone regulate PDL progenitor activity. Experimental studies have shown that Wnt signalling is necessary for periodontal homeostasis [13], that Gli1-positive PDL cells contribute to periodontal tissue turnover [9], and that Axin2-positive PDL cells can contribute to new alveolar bone under orthodontic tension [16]. These data illustrate the reciprocal nature of PDL–bone interactions.

8 VASCULAR AND NEURAL SUPPLY

Alveolar bone is vascularised by branches of the maxillary and inferior alveolar arterial systems and by vessels entering through nutrient canals and marrow spaces. The PDL has a rich vascular network that communicates with vessels in adjacent bone. This vascular supply supports osteoblast and osteoclast activity, nutrient delivery, waste removal and tissue repair. Sensory nerve fibres in the PDL and surrounding bone contribute to proprioception and the perception of loading and injury. Neurovascular bundles are also associated with progenitor-cell niches; Gli1-positive PDL stem cells have been reported around neurovascular structures [9].

9 FUNCTIONAL ADAPTATION TO OCCLUSAL LOADING

Physiological occlusal loading is a major determinant of alveolar bone maintenance. During mastication, the PDL converts intermittent tooth loading into cellular and fluid-flow signals. Osteocytes and PDL cells respond by altering the expression of osteogenic and osteoclast-regulatory mediators. Studies of occlusal hypofunction show that reduced loading can lead to alveolar bone changes and increased sclerostin signalling [17]. Conversely, normal occlusal force can activate Piezo1-dependent mechanisms that help maintain bone mass [14].

Orthodontic tooth movement is a useful experimental model for studying the response of the PDL–bone complex to altered force. On the tension side, bone formation may occur; on the compression side, osteoclast-mediated resorption

permits movement. Mechanosensitive Piezo1, Wnt signalling and PDL progenitor-cell populations have all been implicated in these responses [15,16,18]. These experimental observations should not be interpreted as meaning that orthodontic movement is identical to ordinary mastication; rather, they reveal mechanisms by which the alveolar housing adapts to mechanical environments.

10 CLINICAL SIGNIFICANCE OF HEALTHY ALVEOLAR BONE

An understanding of normal alveolar bone biology is essential to periodontal diagnosis, orthodontics, implant dentistry and oral surgery. The height, thickness and morphology of the alveolar housing influence periodontal phenotype, recession risk, orthodontic movement limits and implant positioning. The thin buccal plate in some anterior regions is particularly relevant when planning tooth movement or surgical procedures. The lamina dura and trabecular pattern provide radiographic information, but radiographic appearance should always be interpreted alongside clinical findings and anatomical variation.

Alveolar bone is also tooth-dependent. Following tooth extraction, the socket undergoes remodelling and the ridge commonly changes in width and height. This post-extraction response is distinct from the steady-state physiology of an intact, loaded periodontium, but it highlights the influence of tooth presence and PDL-derived signals on the alveolar process. Knowledge of normal development and homeostasis provides the biological basis for understanding ridge preservation, periodontal regeneration and implant-site development.

11 CONCLUSION

Alveolar bone is a specialised, highly adaptable component of the periodontium whose structure and metabolism are inseparable from the tooth and PDL. Its socket-facing bundle bone anchors Sharpey's fibres, while cortical and cancellous compartments provide mechanical support and metabolic capacity. Development depends on dental follicle-derived progenitors and coordinated signalling pathways. In health, osteoblasts, osteocytes, osteoclasts and periodontal progenitor cells maintain the tissue through coupled remodelling. RANK–RANKL–OPG and Wnt/β-catenin signalling regulate the balance between resorption and formation, while BMP, TGF-β and mechanosensitive pathways contribute to development and adaptation. Physiological occlusal loading is a key homeostatic stimulus. A sound understanding of these processes is fundamental to interpreting periodontal anatomy and planning predictable periodontal, orthodontic and implant treatment.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Disclosure of conflict of interest

No conflicts of interest regarding this manuscript.

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