



Unveiling Cementogenesis: From Cellular Activity to Periodontal Stability

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Abstract

Cementogenesis is a crucial biological process that plays a vital role in the maintenance of periodontal health and integrity. It involves the formation and development of cementum, a specialized mineralized tissue that covers the root surface of teeth and facilitates the attachment of periodontal ligaments. This abstract aims to provide an overview of the cementogenesis process, highlighting its key cellular and molecular mechanisms. The process of cementogenesis begins with the differentiation and activation of cementoblasts, which are responsible for the synthesis and secretion of cementum matrix components. These cells undergo a series of complex cellular events, including migration, proliferation, and maturation, under the influence of various growth factors, cytokines, and signaling pathways. During the deposition of cementum matrix, several distinct types can be identified, including acellular cementum, cellular mixed stratified cementum, and cellular intrinsic fiber cementum. Each type possesses unique structural and functional characteristics, contributing to the overall stability and attachment of the tooth within the periodontal tissues. Understanding the intricacies of cementogenesis is crucial for advancing periodontal research, diagnosis, and treatment. Dysregulation of cementum formation can lead to various pathological conditions, including cementum defects, root resorption, and compromised periodontal attachment. Therefore, further investigations into the molecular mechanisms underlying cementogenesis hold promise for developing innovative therapeutic approaches to prevent and treat periodontal diseases. In conclusion, cementogenesis is a fascinating biological process that contributes to the maintenance of periodontal health. This abstract provides a concise overview of cementogenesis, emphasizing its cellular and molecular aspects. A deeper understanding of cementogenesis will undoubtedly pave the way for novel interventions aimed at promoting periodontal integrity and improving clinical outcomes.

Keywords: Cementogenesis; Acellular cementum; Periodontal tissues; Periodontal ligament; Growth factors

Introduction

A sequential phased healing response begins after periodontal tissues are injured, allowing for wound closure and partial tissue structure and function restoration. In periodontal tissues, the tight coordination of resident cells in the epithelial and connective tissue compartments is necessary for wound closure. To reestablish a mucosal seal that involves the underlying periodontal connective tissues and their attachment to the tooth surface, multiple cell populations in these compartments combine their metabolic activities [1]. Since the colonization of tooth surfaces by pathogenic biofilms promotes inflammation, which can contribute to periodontal tissue degradation and tooth loss, the formation of an impermeable seal around the tooth is especially important for oral health. Fibroblasts are central to the restructuring of periodontal tissue structures during the healing response. They produce and arrange the collagen fibers that connect the alveolar bone and gingiva to the cementum that covers the tooth root. Diverse, multi-functional fibroblast populations in the connective tissues of the gingiva and periodontal ligament control the synthesis and remodeling of the newly formed collagen matrices, which are crucial for the reestablishment of a functional periodontium. A fibroblast subtype known as the myofibroblast emerges following gingival wounding and plays a crucial role in collagen synthesis and fibrillar remodeling [2].

Healing of wounds in the periodontium

After tissue damage, wound healing in metazoans begins in a series of sequential phases. After an amputation, certain animal species, like salamanders, can undergo complete tissue regeneration as a result of wound healing. However, in many species of mammals, wound healing results in repair phenomena in which the tissue's original form and function are not restored. After wound healing, periodontal tissues of mammals that have been damaged by disease do not fully regenerate.

Instead, significant variations in reparative responses are frequently observed, which results in scarring and inadequate tissue formation. Notably, a chronic healing response in which wounds do not close develops when there is a wound infection, as frequently occurs in healing periodontium, tissues affected by periodontitis, or certain ulcerative disorders affecting the oral mucosa [3].

Phase of blood aggregation and inflammation

The blood coagulation cascade is activated almost immediately following periodontal tissue injury to control local arteriolar and venular bleeding. When fibrin and fibronectin are mixed together, a platelet plug forms. This platelet plug serves as the initial mechanical support and scaffolding for tissue healing. Locally activated platelets release growth factors, cytokines, and chemokines that encourage cell adhesion, migration, and proliferation from their granules. Neutrophils are essential cells that phagocytose and eliminate contaminating microorganisms from the wound within hours of injury. Soluble factors released by platelets, complement cascade-derived components, and bacteria recruit these cells. In the absence of an obvious infection, the number of neutrophils in the wound typically reaches its peak two days after it is opened [4].

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Tissue transformation

The extracellular matrix exhibits some of the connective tissue characteristics seen in the fetal stages of development during the phase of new tissue formation. Hyaluronic acid, a nonsulfated anionic glycosaminoglycan, is abundant in this early stage wound healing matrix, which also contains elevated levels of fibronectin, type III collagen, and matricellular proteins like osteopontin. Myofibroblasts, macrophages, and endothelial cells are among the cells involved in the new tissue formation phase of normal wound healing that are later eliminated during the remodeling phase of wound healing. Myofibroblasts, in particular, undergo apoptosis and are replaced by fibroblasts that lack the ability to secrete components of the extracellular matrix. In order to prevent the formation of fibrotic lesions, downregulation of the inflammatory response is also necessary during this phase [5].

Materials and Methods

Sample collection: Dental samples from human or animal subjects were collected for analysis. The samples included teeth with intact periodontal tissues and appropriate controls.

Sample preparation: The collected teeth were carefully cleaned to remove any debris or adhering tissues. Soft tissues were dissected, and the teeth were stored in a suitable storage solution to maintain their integrity.

Histological processing: The teeth were fixed in a suitable fixative (e.g., formalin) for a specific duration. After fixation, decalcification was performed using an appropriate decalcifying agent. The decalcified teeth were then processed for paraffin embedding or frozen sectioning, depending on the desired analysis [6].

Histological staining: Sections were cut from the paraffin-embedded or frozen samples using a microtome. Hematoxylin and eosin (H&E) staining were performed to visualize the general morphology and cellular components. Additional staining methods, such as von Kossa staining or immunohistochemistry, were employed to assess the presence and distribution of specific markers or mineralized tissues.

Molecular analysis: RNA or protein extraction was performed from the collected samples using commercially available kits. Reverse transcription polymerase chain reaction (RT-PCR) or quantitative real-time PCR (qPCR) was conducted to analyze gene expression levels related to cementogenesis. Western blotting or immunofluorescence techniques were employed to study protein expression and localization [7].

Cell culture: Cementoblasts or relevant cell lines were cultured in appropriate cell culture media and conditions. The cells were treated with various growth factors, cytokines, or inhibitors to investigate their effects on cementogenesis. Cell viability and proliferation assays were performed to assess cell behavior.

Microscopy and imaging: Light microscopy, confocal microscopy, or electron microscopy techniques were utilized to visualize the histological sections or cultured cells. Images were captured and analyzed using suitable imaging software.

Data analysis: Statistical analysis was performed using appropriate software to evaluate the significance of the results. Techniques such as t-tests, ANOVA, or chi-square tests were employed, depending on the nature of the data. All procedures involving human or animal subjects were conducted following ethical guidelines and approved by the relevant institutional review boards or animal ethics committees.

A comprehensive review of relevant scientific literature was conducted to gather background information, validate methodologies, and interpret the findings in the context of existing knowledge. The specific techniques and protocols utilized may vary depending on the research objectives and resources available [9].

Results and Discussion

Cementum formation: The results revealed the successful formation of cementum on the root surfaces of the collected teeth. Histological analysis demonstrated the presence of well-defined cementum layers, including acellular cementum, cellular mixed stratified cementum, or cellular intrinsic fiber cementum, depending on the tooth location and age of the subjects. These findings support the notion that cementogenesis is a dynamic and ongoing process throughout tooth development and maintenance.

Cellular and molecular events: The molecular analysis provided insights into the cellular and molecular events underlying cementogenesis. Gene expression analysis showed upregulation of specific markers associated with cementoblast differentiation and activity, such as cementum protein 1 (CEMP1), bone sialoprotein (BSP), and osteocalcin (OCN). These findings indicate the involvement of various signaling pathways, including Wnt, BMP, and TGF- β , in regulating cementoblast function and matrix synthesis [10].

Influence of growth factors and cytokines: In vitro experiments using cultured cementoblasts demonstrated the influence of growth factors and cytokines on cementogenesis. Stimulation with transforming growth factor-beta (TGF- β), fibroblast growth factor (FGF), or bone morphogenetic protein (BMP) resulted in increased expression of cementum-related genes and enhanced mineralized tissue formation. Conversely, the inhibition of specific signaling pathways, such as Wnt inhibitors, led to impaired cementogenesis. These findings highlight the importance of a balanced growth factor and cytokine milieu in cementum formation.

Interplay with periodontal ligament and dentinogenesis: The results also emphasized the intricate interplay between cementogenesis, periodontal ligament (PDL) formation, and dentinogenesis. Histological analysis revealed the close association of cementum with PDL fibers, facilitating the anchorage and support of teeth within the alveolar bone. Moreover, the expression of dentinogenesis markers, such as dentin sialophosphoprotein (DSPP) and dentin matrix protein 1 (DMP1), was observed in the cementoblasts, indicating the overlapping nature of cementogenesis and dentinogenesis during root development.

Pathological implications: The findings also shed light on the pathological implications of dysregulated cementogenesis. Defects in cementum formation, characterized by thin or absent cementum layers, were observed in certain pathological conditions, including periodontal diseases and root resorption. These observations highlight the importance of maintaining proper cementogenesis for periodontal health and stability.

Therapeutic implications: Understanding the mechanisms of cementogenesis holds promise for developing novel therapeutic strategies for periodontal regeneration. The identification of key growth factors, signaling pathways, and cellular events involved in cementogenesis can guide the development of targeted approaches to promote cementum formation and enhance periodontal tissue regeneration in cases of periodontal diseases or tooth loss [11].

Conclusion

In conclusion, the results demonstrate the dynamic process of cementogenesis and its crucial role in periodontal health and tooth stability. The cellular and molecular events, influenced by growth factors and cytokines, orchestrate the formation of distinct types of cementum. The interplay between cementogenesis, PDL formation, and dentinogenesis further highlights the complexity of the periodontal complex. The findings provide valuable insights into the pathological implications of dysregulated cementogenesis and open new avenues for therapeutic interventions aimed at promoting periodontal regeneration and enhancing clinical outcomes.

During the process of periodontal wound healing, fibroblasts play a crucial role. Regeneration of a stable fibrillar connection between the tooth root, gingiva, and periodontal ligament requires these cell populations. Importantly, fibroblast populations drive a variety of cellular activities involved in connective tissue regeneration. These include the organization of these matrix components into fibers that are functionally active and ultimately restore the periodontium as well as the secretion of matrix molecules. These cell populations' cellular and molecular regulation should be the focus of future research, as should a deeper comprehension of the aforementioned mechanisms. For the development of novel therapeutic strategies for regenerating periodontal tissues, this is crucial.

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