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Review

Cytoskeleton, intercellular junctions, planar cell polarity, and cell movement in amelogenesis

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ABSTRACT

Background: Tooth enamel is composed of highly mineralized rods surrounded by interrod crystals that are formed by ameloblasts derived from dental epithelium. Secretory ameloblasts migrate during hard tissue formation, both away from the dentin and in groups that slide past each other, resulting in rod decussation. Enamel rod decussation is commonly observed in many animal teeth including humans.

Highlight: Cytoskeleton fibers, such as microtubules, intermediate filaments, and actin filaments, are associated with ameloblast movement. Rat incisor enamel is composed of initial, inner, outer, and final layers. Secretory ameloblasts forming the inner enamel layer move laterally and have proximal and distal junctional complexes attached to actomyosin filaments. Conversely, secretory ameloblasts forming the outer enamel layer cease lateral movement. Secretory ameloblasts forming the inner layer are characterized by anisotropic distribution of adherens junctions, desmosomes, and actomyosin filaments in transverse distal junctional complexes. Isotropic distribution is observed in distal junctional complexes in secretory ameloblasts forming the outer layer. Actin cytoskeleton and junctions may act as a motor apparatus to control the sideways movement of ameloblasts. However, the mechanism that determines whether secretory ameloblasts forming the inner layer move medially or laterally is unclear. One potential group of proteins that may be involved in this process is the core planar cell polarity (PCP) proteins.

Conclusion: One core PCP protein, VANGL2, is proposed to be a key molecule determining the direction of ameloblast movement.

Contents

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1. Introduction

Inductive interactions between dental epithelium and underlying mesenchyme form ameloblasts and odontoblasts. Ameloblasts form enamel, and odontoblasts form dentin. Epithelial-mesenchymal interactions results in ameloblast differentiation (Fig. 1). The secretory and maturation stages have been previously characterized: secretory ameloblasts secrete enamel matrix proteins such as amelogenin, ameloblastin and enamelin, and maturation ameloblasts remove water and degrade matrix proteins to make space for further mineralization [1]. During enamel formation, the entire enamel surface area of a tooth increases. Ameloblasts migrate during this process and their coordinated directional movement coupled with extracellular matrix secretion is necessary for correct tooth enamel architecture formation.

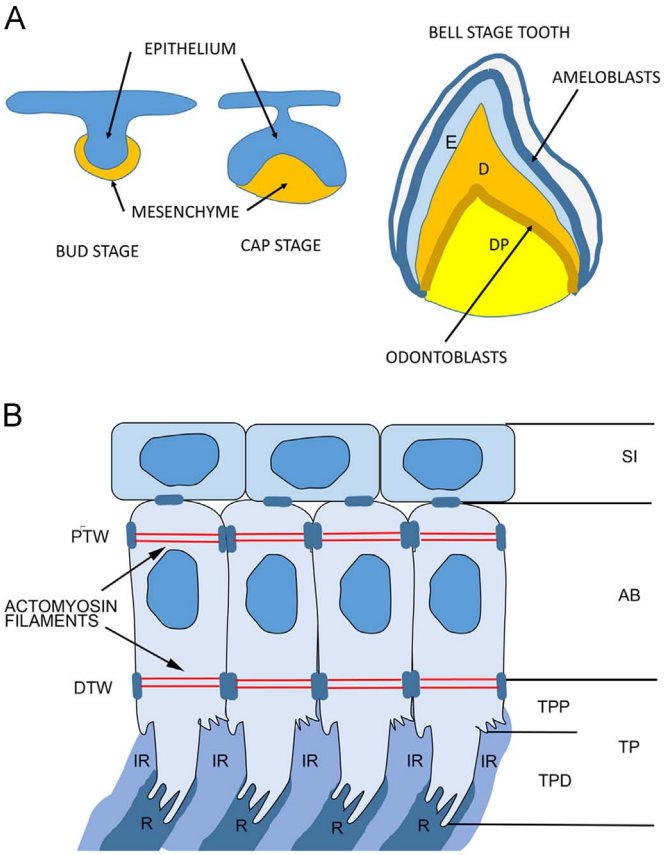

Fig. 1. (A) Schematic representation of tooth development. Inductive interactions between epithelium and mesenchyme form dental lamina at the bud stage, followed by the cap and bell stages. At the bell stage of tooth development, ameloblasts form enamel (E) and odontoblasts in the dental pulp (DP) form dentin (D). (B) Schematic representation of secretory ameloblasts. Secretory ameloblasts are characterized by their ameloblast body (AB), Tomes' processes (TP), and movement. Tomes' processes are further subdivided into the proximal portion (TPP), which is responsible for interrod enamel (IR) formation, and the distal portion (TPD), which is responsible for rod enamel (R) formation. Distal junctional complexes and actomyosin-based filaments (DTW) are evident between AB and TP. Proximal junctions and actomyosin filaments (PTW) are also present close to the stratum intermedium (SI) region that overlays the ameloblast layer.

Ameloblasts contain well-developed cytoskeletal components, such as actin filaments, microtubules and intermediate filaments. These cytoskeletal components are linked to desmosomes, adherens junctions, or other areas of the plasma membrane. Elucidation of the cytoskeletal components linked to cellular junctions involved in ameloblast movement during matrix secretion is important for understanding enamel formation. Dynamics of their cytoskeletal components during maturation is also important and de novo synthesis of tight junction proteins in the Golgi body have been proposed, however this topic is beyond the scope of this review [1,2].

In this review, we describe the cytoskeletal components of ameloblasts in rat incisors and junctional complexes, such as adherens junctions with actomyosin-based contractile machinery, followed by ameloblast-enamel attachment. Finally we describe ameloblast movement, its contribution to enamel rod alignment, and discuss the potential involvement of core planar cell polarity proteins in the directional movement of ameloblasts.

2. Cytoskeleton

2.1. Intermediate filaments

Intermediate filament proteins are variable among cells, such as vimentin in fibroblasts, desmin in muscle cells, and cytokeratin in epithelial cells [3]. Lesot et al. [4] demonstrated the presence of keratin in ameloblasts using immunofluorescence microscopy. Cytokeratin 14 is expressed in dental epithelial cells and has been used to identify their distribution patterns [5]. Mutation of hair keratin, KRT75, has been reported to increase dental decay risk in enamel, suggesting the involvement of KRT75 in amelogenesis [6].

Cytokeratin filaments bind to both desmosomes and other filaments. Tonofilaments, which are cytokeratin intermediate filaments, are known to run longitudinally in ameloblasts [7]. Desmosomes play a role in preventing cell disruption and maintaining cell shape. They are abundant between ameloblasts and stratum intermedium cells or papillary layer cells [8,9]. Analysis of the desmosome component, desmoplakin, by immunocytochemistry revealed that demosomes are also abundant between ameloblasts at the level of the proximal junctional complex and distal junctional complex [10].

2.2. Microtubules

Microtubules are abundant in preameloblasts and ameloblasts in the secretory stage. Microtubule numbers increase in the apical direction close to the dental pulp. These ameloblast microtubules are composed of 13 protofilaments, the number typically found in microtubules, though they often form unusual bundles [11]. Most microtubules in columnar preameloblasts run lengthwise down the cell body. This pattern remains unchanged in subsequent secretory ameloblasts [11]. Additional microtubules are localized at the more apical part of the cell, also along the long axis of ameloblasts. Some unidentified motor proteins have been shown to transport secretory granules containing enamel matrix proteins along microtubules. Anti-microtubule drugs, that inhibit its

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