Dictionary of Normal Cells

The Ameloblast

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Introduction. The ameloblasts are cells of ectodermal origin responsible for the enamel formation. This type of cells was noticed on histological slides only during the formation of the tooth, when it is arranged as a continuous layer on the enamel surface. After the eruption of the tooth in the oral cavity, the ameloblasts disappear. These cells loss, makes the enamel a non-vital and insensitive matrix which, when destroyed by any means (usually wear or caries), it cannot be replaced or regenerated. To compensate for this inherent limitation, the enamel acquired a complex structural organization and a high degree of mineralization due to the almost complete absence of the organic matrix in its mature state. These characteristics reflect the unusual life cycle of ameloblasts and the unique physicochemical characteristics of matrix proteins that regulate the formation of extremely long hydroxyapatite crystals in enamel.

Differentiation of ameloblasts. The ameloblasts differentiation begins in the internal epithelial layer of the enamel organ. It becomes polarized under the influence of inductive factors. Before the onset of dentinogenesis, the cells of the internal epithelium of the enamel organ become columnar, dividing rapidly to accommodate the growth of the dental germ, being arranged on a basement membrane that separates the enamel organ from the dental papilla. In the bell stage, the cells in the inner layer of the enamel organ no longer grow, and the divisions stop. These changes prevent a reversal of polarity in these cells and thus they very quickly induce different dental papillary cells adjacent to the internal epithelial layer in odontoblasts. Even before the formation of the first layer of dentin by odontoblasts - the dentin mantle, in the cells of the internal epithelial layer the nucleus migrates to the opposite pole of the dental papilla, reversing the polarity of the cell. Cells turn into precursors of ameloblasts called preameloblasts.

Morphology and ultrastructure. The morphology of ameloblasts changes at each stage of amelogenesis, such as: pre-secretory, secretory, transitional, maturation and postmaturation. In the presecretory stage of amelogenesis, the cells which were noticed are preameloblasts. They are columnar cells, with a length of approximately 25-30 μm, width 2–4 μm and functional polarity. Thus, preameloblasts have two poles: a secretory one towards the odontoblasts and a nuclear one that comes in contact with the intermediate epithelial layer of the enamel organ, where the nucleus is located. In the cytoplasm of preameloblasts are found many non-specific cytoplasmic organelles such as: mitochondria, rough endoplasmic reticulum, Golgi complex, vesicles. At the nuclear pole they have numerous desmosomes, which connect the preameloblasts. Preameloblasts release enzymes through exocytosis that degrade the basal lamina and then followed by a resorption process of degradation products by endocytosis. Preameloblasts secrete proteins above the dentin matrix, some of which diffuse through the matrix and are used by odontoblasts.

At the beginning of the secretory phase of amelogenesis, secretory ameloblasts are tall cells, over 60 μm in height and 2–4 μm wide, with their nucleus at the basal pole (far from the enamel formed). After the deposition of the initial enamel, thin, aprismatic, a cone-shaped process is formed, the Tomes process, at the distal or secretory pole of the ameloblast. The shape of the Tomes processes is responsible for the prismatic structure of the enamel. At the secretory pole (apical pole) are numerous free ribosomes, mitochondria, microfilaments of keratin, microtubules and secretory granules. Between the cells, there are tight junctions at both poles. The enamel proteins (enamelin, amelogenin, ameloblastin, amelotin, tuftelin) are synthesized in the endoplasmic reticulum, then pass into the Golgi complex, where they are condensed and stored into secretory granules.

After the formation of the partial mineralized enamel, the ameloblasts go through the transition stage – the so-called transition ameloblast, characterized by the decrease in cell height and volume, but also the decrease in the number of intracytoplasmic organelles. Excess organelles are included in vacuoles of autophagy and digested by lysosomal enzymes. The remaining ones accumulate in the distal cytoplasm of the cell, giving rise to a striated border.

When the enamel maturation is complete, the ameloblasts lose the striated border and secrete a material arranged between the enamel surface and the distal pole of the cells (postsecretory ameloblasts). These are flattened and structurally, the material deposited on the enamel surface is identical to that of the basement membrane.

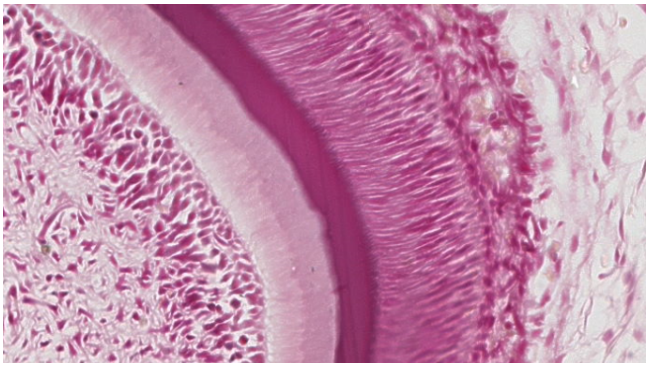

Figure1. Developing tooth, secretory ameloblasts with functional polarity, nuclear basal pole, and distal secretory pole with Tomes's process, hematoxylin eosin staining

Functions. The ameloblasts are cells that secrete enamel proteins (enamelin and amelogenin), which subsequently mineralize to form the enamel, the hardest structure in the human body. Ameloblasts control the ionic and organic compositions of the enamel. Ameloblasts adjust their secretion and resorption activities to maintain favorable conditions for biomineralization.

Clinical correlations. There are several factors that can affect the differentiation and development of ameloblasts, causing the formation of abnormalities in the structure of the tooth. An example is BMP (bone morphogenetic protein), which has an important role in the ameloblasts differentiation. When some BMP inhibitors are expressed in the developing tooth epithelium, ameloblasts do not differentiate or form enamel. High childhood fever is also a factor that causes disruptions in enamel production. Fluoride ions interfere with ameloblast function also, resulting areas of reduced mineralization in the enamel.

REFERENCES

[1] Bronckers ALJJ. Ion transport by ameloblasts during amelogenesis. J Dent Res. 2017; 96:243–253.

[2] Bronckers ALJJ, Lyaruu D, Jalali R, Medina JF, Zandieh-Doulabi B and DenBesten PK. Ameloblast Modulation and Transport of Cl– , Na+ , and K+ during Amelogenesis. Journal of Dental Research 2015; 94(12):1740–1747

[3] Miyazono K., Kamiya Y, Morikawa M. Bone morphogenetic protein receptors and signal transduction. Journal of biochemistry. 2010; 147:35–51.

[4] Thesleff I, Mikkola M. The role of growth factors in tooth development. International review of cytology. 2002; 217: 93–135.

[5] Honda, MJ, Shinmura Y, Shinohara Y. Enamel tissue engineering using subcultured enamel organ epithelial cells in combination with dental pulp cells. Cells, tissues, organs. 2009; 189:261–267,

[6] Gallon V, Chen L, Yang X, Moradian-Oldak J. Localization and quantitative co-localization of enamelin with amelogenin. Journal of structural biology. 2013; 183:239–249.