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Review Article

Maturation of Dental Tissues

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Abstract

In addition to initial maturation, dental tissues maturation physiological development involves ions and molecules adsorption at the surface of tooth enamel. Demineralization/remineralization, absorbance and exchanges are associated to tooth function. Crystals lengthening and thickening provide significant enamel maturation. Dentin maturation implicates the closure of tubules, and tooth eruption in the oral cavity. Pulp chamber is gradually reduced, the teeth becoming more resistant to the progression of dental caries. Pulp stones or/and diffuse mineralization significantly reflects tooth maturation.

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INTRODUCTION

Enamel early maturation

The eruption of deciduous teeth starts at 0.629 years, and ends at 2.333 years. For permanent teeth, eruption begins at 6.24 years and ends at 20.50 years. During this period of time, enamel matures. Maturation changes are also taking place in dentin.

Exposition of enamel to saliva, foods and toothpastes leads to absorption of mineral at the enamel surface and on the acquired pellicle. Therefore enamel becomes more resistant to the carious decay. Saliva and bacteria enrich the outer part of enamel. Enamel maturation is related to the period of time during which the tooth is exposed to oral fluids in the oral cavity. The changes at the enamel surface are associated to the stimuli and tooth function.

However, "aged" tooth is influenced by the endogenous and exogeneous pellicle, the composition of foods and toothpastes, and the ions that are released. The pellicle is enriched by the microbial plaque and exchanges occur between the oral cavity and enamel surface. The successive demineralization / remineralization effects on enamel orchestrate the changes occurring both at the surface and in the subsurface.

The initial carious lesions (white spots) mature and may disappear. Restorative procedures and trauma are implicated in the changes occurring along the enamel surface, including the various exchanges of enamel composition, ions absorbance and exchanges with saliva and exogeneous pellicle [1].

After the initial secretion phase, ruffled and smooth ended ameloblasts are implicated in early maturation, before teeth erupted in the oral cavity. Interactions between the enamel surface and secondary maturation are due to exchanges with ions interactions [2]. It is also obvious that the pH displays oscillations that contribute to modify the surface of enamel. These variations are required to keep open intercrystalline spaces.

Degraded enamel matrix proteins are removed while hydroxyapatite crystals are growing in width and length.

Two steps occur during enamel secondary maturation:

1. During step 1, at the crystal surface, enamel proteins are hydrolyzed. Fragments of amelogenins and enamelin are retained, and in addition ions are added [2].
2. During step 2: the crystal volume expands and the internal spaces are reduced. The concept of thixotropic gel [3] for enamel proteins, is still valid.

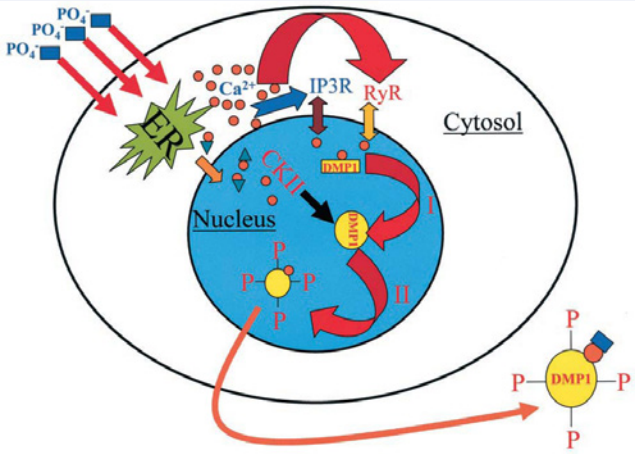
Amelogenins, ameloblastin (also named amelin and/or sheathlin), enamelin, enamelysin (MMP-20), amelotin (AMTN) and enamel matrix serine proteinase-1 (EMPS1) are secreted proteins involved in enamel mineralization. MMPs include matrilysin (MMP-7), gelatinases A and B, enamelysin, serine proteinases, cathepsin B, proteinase inhibitors. Altogether, MMPs are also implicated in enamel maturation [4].

Many of the genes encoding extracellular components are specifically associated with dental enamel formation. They are grouped in a family of molecules, named SCPP (secretory calcium-binding phosphoprotein), and enamel proteins [amelogenin (AMEL), enamelin (ENAM), ameloblastin (AMBN), amelotin (AMTN), as well as the secretory stage enamel protease (MMP20].

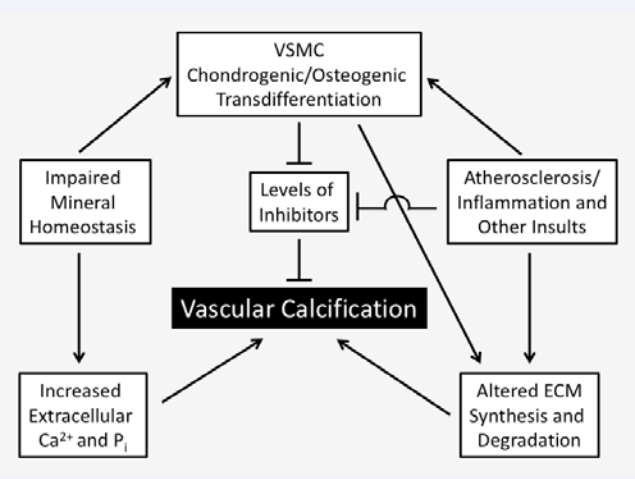
Ameloblasts orchestrate crystal growth via multiple cellular and acellular activities, proteolysis, and endocytosis. The bulk of the enamel is firstly formed and subsequently mineralized by the same cells (post-secretoy maturation ameloblasts). Cell death by apoptosis and regression are following enamel maturation [5].

Ruffle-ended ameloblasts appear to function primarily as a transport epithelium. They are controlling the movement of calcium and other ions such as bicarbonate into enamel and maintain buffering capacity. The reasons ruffle-ended ameloblasts become smooth-ended periodically is not understood, although these events seem to be crucial for sustaining long-term crystal growth.

Amelogenin, ameloblastin and enamelin are retained in enamel at the maturation stage. KLK4 was decreased, but no change was noted for MMP-20. Enamel maturation involves the degradation and removal of growth-inhibiting residual enamel



Shematic representation 1 DMP-1 expression was reduced in aged human dental pulp. The osteocalcin mRNA was expressed in young adult dental pulp but decreased in aged human dental pulp.



Shematic representation 2 Shematic drawing showing the key determinants of the mechanisms of vascular calcification. [20].

matrix proteins and secondary mineralization at the sides of the expanding apatite crystallites.

Enamel surface is covered by a monolayer of bacteria, and exchanges occur with the multispecies biofilms present in the oral cavity. This leads to the formation of the endogenous acquired pellicle.

During the stage of maturation, which transforms the immature enamel into a highly mineralized tissue, ≈ 86% of the mineral content of mature enamel is deposited. During enamel matrix secretion, binding of enamel proteins at the side of the enamel crystallites ensures mineral deposition. They are mostly accessible at the tip of the growing crystallites, allowing lengthening, although displaying limitted expansion in width.

Reference should be made also to the prismless outer layer of the primary enamel [6]. This layer ranged in thickness from 15 to 75 µm. Radiolucent lines at approximately 6 µm intervals were observed running through the body of the early carious lesion. The striae of Retzius were visible mainly in the subsurface zone. The surface outer layer was prismless, and negatively birefringent. This layer seems to be due to mineral re-precipitations.

Dentin maturation

Owen was the first to report laminations in dentin. Measurements of the intervals between two laminations indicate a variation about 3,5 to 6 µm. They are formed daily and known as incremental von Ebner and/or Andresen's lines.

A part of the extracellular dentin matrix is incorporated during dentin formation by the distal end of odontoblasts and into odontoblast processes via large coated vesicles. Then, they are digested intracellularly via a lysosomal system located in the distal odontoblast's cell bodies. In addition, roots were short with enlarged root canals. As an accentuation of Owen's contour line, the calcified dentin layers were separated by a dentin layer forming a daily pattern of dentin deposition that progress at about 6 µm per day in the crown, and 3.5 µm per day in the root. The secondary dentin seemed less tubular and showed entrapped cells, probably odontoblasts implicated in the formation of osteodentin. The secondary dentin in the region of pulp horns seemed to bear a greater density of cells than in younger specimens.

The cell population and the number of collagenous fibers bundles increase. The loss in the volume of the pulp chamber is apparently related to a reduction of ground substance. Reticular fibers are abundant, whereas collagenous fiber bundles are observed at all ages. Dentin sialophosphoprotein (DSPP) is cleaved into dentin sialoprotein (DSP) and dentin phosphoprotein (DPP). DSP, the amino-terminal part of DSPP, is a sialic acid-rich, glycosylated protein. The members of the SIBLING (Small Integrin-Binding Ligand N-linked Glycoproteins) family, include DSP, DPP, DMP-1, OPN, and MEPE. DMP-1 influences bone mineralization and controls serum phosphate levels by regulating serum FGF-23 levels. DMP-1 activates integrin signaling and is endocytosed into the cytoplasm. It is further translocated to the nucleus. DSP plays a significant role in the initiation of dentin mineralization, whereas DPP is involved in the maturation of mineralized dentin [7].

The phosphorylated DMP1 is exported out into the extracellular matrix, where it regulates hydroxyapatite nucleation. DMP1 is highly anionic and rich in aspartic acid, glutamic acid, and serine residues. Fifty two per cent of the serine is potentially phosphorylated by casein kinase II. DMP1 play an important role in mineralized tissue formation, implicated in the initiation of nucleation. Imported into the nucleus, DMP1 requires the presence of nuclear localization signals (NLS) and is associated with the transport machinery. Inspection of the primary sequence of DMP1 led to the identification of three potential sequences.

a) DMP1 migrate from the nucleus rather than maintained in a nuclear pool.

b) Phosphate groups confer a very high capacity to DMP1 for binding calcium ions, which is important for its potential function in mineralization.

c) To examine whether DMP1 is differentially

phosphorylated *in vivo*, DMP1 labeling was seen inside the nucleus, cytosol, and extracellular matrix.

Deletion of DMP1 leads to a partial failure of maturation of predentin into dentin, hypomineralization and expanded pulp and root canal during postnatal tooth development [8-10].

Osteonectin is concentrated around dentinal tubules. Its binds to hydroxyapatite and collagen. MMP-2 (gelatinase A) increases significantly during dentin maturation, as well as purified DPP. Quantitative reverse-transcription polymerase chain reaction and immunohistochemical staining confirmed the microarray results. They are strongly expressed in the coronal pulp, whereas *SMOC2, SHH, BARX1, CX3CR1, SPP1, COL XII*, and *LAMC2* are mainly expressed in the apical pulp [8].

Odontoblasts maturation [11].

Many large and small coated vesicles, and dense multivesicular vesicles are distributed throughout the odontoblast cell bodies. Concerning the ultrastructural localization of acid-phosphatase (AcPase) activity in old odontoblasts, the reaction products are commonly observed in the Golgi complex and in lysosomes. Therefore, a part of the extracellular dentin matrix is absorbed by old odontoblasts via large coated vesicles and then digested intracellularly by an extensive lysosomal system.

Extracellular Matrix maturation: in the Root: Using a paste containing Augmentin as an intracanal medicament, the formation of root apex was expanding, but without increase in toot length. However, the formation of the root apex is possible without pulp regeneration. Apexogenesis and apexification constitute two alternatives possibilities to endodontic therapies. The relatively thin dentin walls of the large canals place the tooth at greater risk for root fracture over time [12,13].

Pulp chamber maturation

During pulp maturation there is a restriction of space due to continuous dentin apposition. It is the fastest on the floor of the pulp chamber. The comparison between young and old pulp chamber indicates three characteristics events that are age-dependent [14] (Figure 1).

Young pulp chamber:

a) Thickening of the inter-root dentin (thickening of the floor)

b) Lateral walls formation

c) Roof dentin formation.

Aging pulp chamber:

a) Dentin formation is faster in the lateral walls

b) The roof dentin formation (occlusal) is accelerated

c) In the furcation - inter-root dentin formation comes in third position.

During aging: the pulp displays an elevated density of nerves and an increased blood supply. The dental pulp becomes more fibrous and less cellular.

Dentin aging and maturation include:

a) Age changes reduction in size and volume of the dental pulp. (Figure 1) The pulp is reduced in the occlusal direction. The reduction in height is greater than in the mesio-distal diameter.

b) There is a decrease of cellular components,

c) There is an increase in the number of bundles of collagen fibers,

d) Together with the presence of dystrophic calcified masses (either calcospherites or diffuse mineralization),

e) There is a decrease in the number of blood and lymphatic vessels and their associated nerves. Some dentin is formed on the occlusal wall (roof of the pulp chamber), and less is formed on the side walls.

f) These processes are fastest on the floor of the pulp chamber. Fibroblasts (or pulpoblasts) are spindle shaped cells with ovoid nuclei.

Pulpstone

Pulpstones are classified as true denticles, false denticles and diffuse calcifications. They are occurring at the roof (1) and floor (2) of the pulp chamber, and less in the lateral walls (3) (Figure 3). Pulp calcification and pulp stone differences were established between false, free, adherent and embedded pulp stone (calcospherites), forming continuous rings around vessels. The different types and formation of pulp stones, leads to use a terminology of denticle, fibrodentin and dystrophic calcification. Osteonectin and osteocalcin were not detected by immunohistochemistry whereas osteopontin was expressed both in atherosclerotic plaques and urinary stones [18].

In the aging process, there was a gradual narrowing of the surface of the pulp due to the continuous parietal apposition of

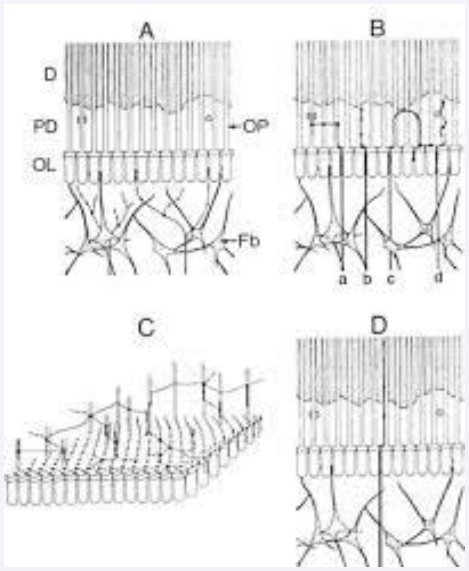


Figure 1 Shows the innervation pattern in predentin and dentin. The Raschkow plexus, establish contacts between the proximal part of odontoblasts (A), crossing predentin(B) and penetrating dentin tubules (150μm in length) (C and D). Figure reprinted from [17]).

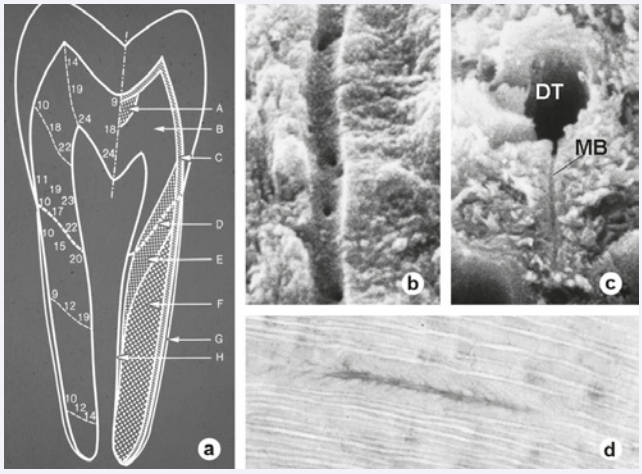


Figure 2 Dental permeability; [21].

dentin. The blood vessels and nerves appeared to become more prominent in the core of the pulp. One of the earliest changes observed in the arterioles was the deposition of a PAS-positive material that directly underneath the endothelial layer. This deposition formed a membrane that obliterated or masked the internal elastic membrane. Other arterioles in the older teeth showed intimal hyperplasia characterized by a thickened intima and a narrowed lumen.

Three types of arteriolar alteration were demonstrated. They consist of hyalinization of the arteriolar walls, endothelial proliferation, and elastic hyperplasia. The arterial vessels exhibited arterio-sclerotic changes. The majority of the vessels examined showed a deposition of PAS-positive material in the intima that extended into the media [19].

Vascular calcification implies an increased level of Ca^{2+} and P_i in the presence of a scaffold formed by the components of the ECM. Matrix Gla-Protein (MGP) initiate arterial calcification without any requirement of vascular smooth muscle cells (VSMCs). Impaired mineral homeostasis and inflammatory responses may directly affect the major determinants of vascular calcification. These pathologic conditions may also induce chondrogenic or osteogenic transdifferentiation of VSMCs which may promote the progression of vascular calcification.

CONCLUSION

Structural and compositional maturation processes of dental structures (enamel and dentin) play important roles in inflammatory processes. Transcription and growth factors allow the primary deciduous dentition to become secondary permanent dentition. The maturation processes are implicated in the transformation of dental tissues becoming tertiary dentin beneath a calciotraumatic line (Figure 3). They become more resistant to the carious decay, as well as to dental therapies.

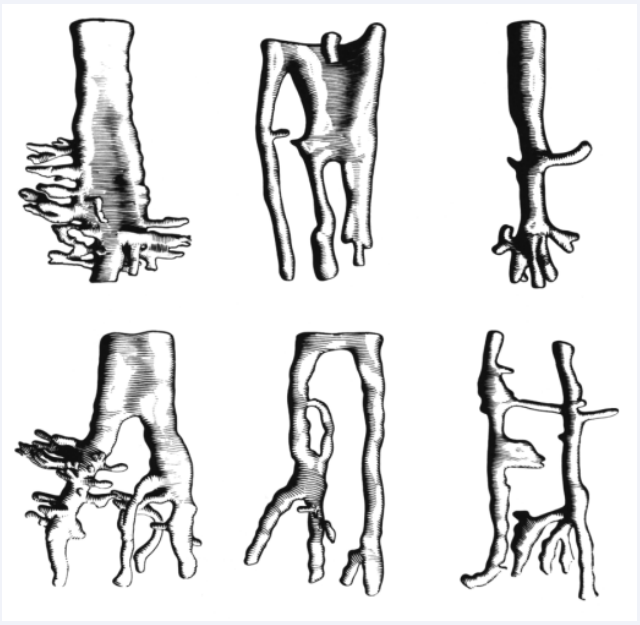


Figure 3a Complexity of the root canals. Reprint from ramifications of the apical pulp space anatomy. [1].

Figure 3b Three-dimensional reconstruction of successive sections showing the apical part of a molar [1,21,22].

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