

Short Note

Deciduous Tooth and Dental Caries

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THE DIFFERENCES BETWEEN DECIDUOUS AND PERMANENT TEETH

In humans, deciduous tooth development begins before birth and is complete by about the fourth postnatal year. They are lost when the patient becomes 11 years old. The permanent teeth appear by 6-7 years (and later for the wisdom teeth). Most are successional, and a few are non successional. The coronal part of the human tooth is composed of two hard tissues: enamel and dentin, and this part includes the dental pulp, located in the crown.

Enamel and dentin differ in composition in terms of type and quantity of organic and/or inorganic phases, amount of water, concentration of minor elements, and crystal size [1]. The differences that are apparent may play a role in the development of carious lesions.

In the deciduous teeth, the mean rod head has the same size than measurement carried out on the permanent enamel. Slight variations were detected between the rod head for the primary teeth (3.22 μm to 3.47 μm) and for the primary permanent teeth. Mean values for the permanent teeth varied between 3.84 $\mu\text{m} \pm 0.73$ to 4.34 $\mu\text{m} \pm 0.95$ for rod heads. In addition, a "prismless" outer surface layer, as well as prisms (or rods) displayed slight differences between the two types of teeth. Neonatal lines (NNL) in the enamel of primary teeth have a hypomineralized character. Analyzes of undecalcified sections indicate that the enamel has a more porous structure than the enamel located in permanent teeth. Change in the growth direction of the prisms was also observed in NNL. This is an optical phenomenon due to an alteration in height, and to the mineralization as analyzed in the enamel prisms [2].

Apparently, caries progression is faster in the deciduous teeth compared with the permanent dentition. Deciduous tooth enamel was softer than permanent tooth enamel. SEM showed no clear differences in the ultrastructure of enamel specimens among the different groups [3]. The clinical diagnosis of severe dental erosion in the cases studied was reliable as verified by SEM. The greater susceptibility of deciduous enamel appears to be due to greater porosity. The deciduous enamel was softer, more prone to fracture and with larger hydroxyapatite crystallites [4]. In the deciduous enamel the mean thickness measurement was 1.14 mm, and in the permanent enamel 2.58 mm. The

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numerical density of enamel was higher in the deciduous teeth than recorded in the permanent teeth, mainly near the dentino-enamel junction (DEJ) [5].

Variations are detected in the chemical composition of the deciduous and permanent enamel types. These differences may be one of the several factors contributing to faster caries progression in deciduous teeth. The carbonate ion occupies two different positions: (A) the hydroxide position, and compared to (B), the phosphate position. The deciduous enamel contains more type A carbonate than B in permanent enamel. The total carbonate content (A+B) is significantly higher in deciduous than in permanent enamel. This may contribute to differences in the speed of carious decays.

ABRASION, ATTRITION, ABFRACTION AND EROSION: NON-CARIOUS PROCESSES

Lower mineral levels are found in deciduous enamel. Non-carious destructive processes affect the teeth. This includes the occurrence of six different events: abrasion, demastication, attrition, abfraction, resorption and erosion [6]. Abrasion describes the wearing away of a substance through mechanical processes such as grinding, rubbing or scrapping. Demastication is a physiological process affecting primarily occlusal and incisal surfaces. Attrition describes the action to rub against something. Proximal surfaces are worn by attrition during mastication. Abfraction: describes a special wedge-shaped defect at the cemento-enamel junction. Microfractures propagate with time fissures perpendicular to the long axis until enamel and dentin break away. Tooth erosions are termed extrinsic, intrinsic, or idiopathic.

The "prismless" outer layer seen along the deciduous enamel is forming about 70% of the surface, whereas it is continuous in permanent enamel (100%). In thickness, the prismless average is approximately 30 μm . Parallel laminations are seen near the enamel surface (Figure 1). The crystallites within this layer are oriented with their 'c' axis almost perpendicular to the enamel surface. The crystallite orientation of the prismless layer differs from that of underlying enamel by $26.9^\circ \pm 7.6^\circ$.

The crystallites in the "prismless" enamel are arranged parallel to each other with their c-axis perpendicular to the striae of Retzius as they reach the enamel periphery.

It is concluded that the “prismless” enamel is always present in non-erupted deciduous molar. It forms a well-defined layer (Figure 2). Made by the assembly of hydroxyapatite crystallites, the crystals are parallel to each other and perpendicular to the enamel surface. The thickness of this layer is 7.257 micrometers, and don't vary between the occlusal, middle and cervical regions.

DIFFERENCES IN DENTINAL STRUCTURES

In human dentine of deciduous teeth ($18,243 \pm 3845$) and permanent teeth ($18,781 \pm 5855$), no significant difference was found in the number and diameter of dentinal tubules [7]. Demineralized and ground methods reveal tubules with primary and secondary curvatures, canaliculi, interglobular dentine, predentine and intertubular dentine [8]. Careful examination shown thicker deciduous peritubular dentine compared to permanent teeth. Deciduous teeth have smaller diameter and tubular density than permanent dentine, and consequently have less permeability. This is depending on the age of the tooth. In human deciduous teeth, bone sialoprotein (BSP) and osteopontin (OPN) are present in intratubular, intertubular and peritubular dentine matrix.

Therefore, the structural differences between the carious lesions of deciduous and permanent teeth were feeble. After the collapse of the overhanging enamel, the carious cavity contains necrotic food debris. The demineralized soft carious dentine layer

displays enlarged canaliculi, without peritubular dentine. This layer constitutes the infected dentine. In the affected dentine, peritubular dentine reappears gradually. The tubule diameter was returning to the normal, and precipitations inside the lumen of the tubules contained either non-active bacteria, or mineral precipitations due to non-apatitic structures (amorphous calcium phosphate, whitlockite etc.).

DENTAL CORONAL CEMENTUM IN HUMAN DECIDUOUS MOLARS

Cementum is an avascular structure, covering the root surface. It is classified as cellular and/or acellular depending the presence or absence of cementocytes. Covering the cemento-enamel junction, afibrillar cementum is formed by collagen, glycosaminoglycans and blood osteopontin. Three types of cementum enamel junctions are identified: an overlap (in which cementum overlaps enamel, and is called coronal cementum), abutment (cementum butts with enamel) and a gap depending on the sectioning plane. The elastic modulus of the coronal cementum (11.0 ± 5.8 GPa) is significantly lower than primary cementum (15.8 ± 5.3 GPa) [10].

The following molecules have been identified: the tissue non-specific alkaline phosphatase, bone sialoprotein, dentin matrix protein-1 (DMP-1), fibronectin, osteocalcin, SPARC/osteonectin, and osteopontin. Other molecules attract and stimulate cementum cell differentiation e.g. the cementum-derived growth factor (CGF) and/or the cementum derived attachment protein (CAP).

The dental plaque accumulates in the gingival sulcus. The flora present in the sulcus adheres to the enamel emerging at the gingival margin. Adhesion of the dental plaque to enamel surface initiate the formation of an initial carious lesion. The cemento-enamel junction is scalloped in deciduous teeth, and smooth in permanent teeth. Four situations were seen occurring at the collar of deciduous or permanent teeth: 1) cement is formed over enamel, 2) a thin layer of enamel accumulates over cement, 3) vis a vis, and finally 4) a gap is seen between cementum and enamel, with exposed dentin.

At the cervical margin, the initial carious lesion develops and grows forming a class V lesion. The occlusal part of the lesion myolysis or cervical erosion is mostly related to enamel, whereas in the cervical part indentine, the lower part is horizontal and interfere with the translucent hyaline zone (the so-called “prismless” or aprismatic zone) followed by the subjacent Hopewell-Smith and the Tomes granular zones. Non-carious erosion or abfraction differed from the carious lesion, the deeper part containing either sclerotic dentine, or at the surface, comprising a softened or demineralized carious layer with enlarged tubules, filled by bacteria. This is the invaded layer, and the affected layer is located in the deeper part. Far from the non-carious or carious lesion, the pulp reacts by the formation of reactionary dentine.

THE CARIOUS PROCESSES

Enamel carious lesion

The initial carious lesion includes 4 layers: 1) a surface layer, well mineralized, (20-50 μm , with an average of 9.9% reduction

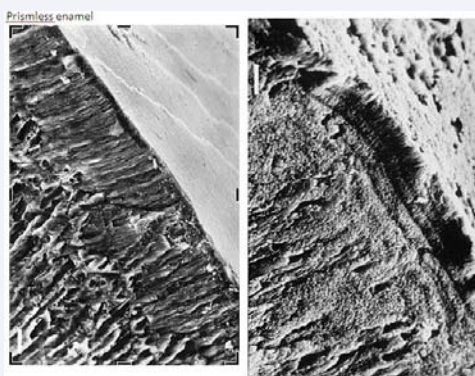


Figure 1 At the surface of the tooth. Parallel lines are visible near the enamel surface.

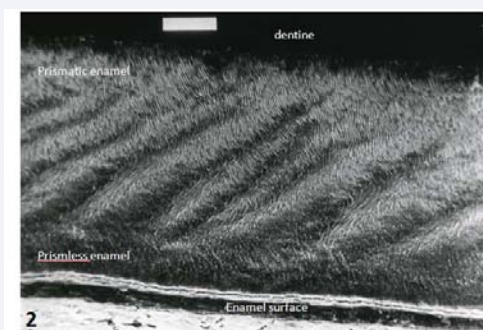


Figure 2 Prismless enamel is visible beneath the enamel surface. The main part of enamel is formed by prismatic enamel. Dentine is located more internally.

in mineral content), 2) the sub-surface layer or body of the lesion (the most demineralized part, showing 10- 24% reduction of the mineral content). In this part the Retzius lines are more apparent, together with prisms that display a 4mm periodicity), 3-4) the two condensation zones comprising a dark (6% loss – associated with a hypomineralized layer) and a translucent zone (with a 1.2 % loss), located at the limits of the lesion separated from the sound enamel (Figure 3). The chalky surface layer is due to mineral reprecipitation. Mineralization is reduced in the subjacent zone. Differences between deciduous and permanent enamel initial caries are not obvious.

The initial white spot lesion cross the whole thickness of enamel, and expand at the amelo-dental junction where the carious lesion penetrate into the dentine.

DENTINE CARIOUS LESION

Beneath enamel, the carious lesion displays a surface zone where canaliculi have lost intratubular precipitations as well as peritubular dentine. Intertubular dentin reaches rapidly its normal mineral content. Tubule diameter reach soon the usual size and number of canaliculi. Sclerotic dentin is formed in the deeper part of the lesion, above a layer of dentin isolating the carious lesion from the dental pulp (Figure 4).

Along the walls of the pulp chamber, a layer of reactionary dentin is located beneath the calcio-traumatic line (Figure 5). When the lesion is close to the dental pulp, penetrate and reveal a thin opening of a pulp horn, reparative dentine forms, occluding rapidly the pulp perforation. Within the pulp, some stem cells proliferate. They perform asymmetric divisions. Pulp cell differentiate and may acquire different phenotypes. Self-renewal is occurring. Finally, recolonization of pulp remnants occurs. This is leading to pulp healing and regeneration.

STEM CELLS FROM HUMAN EXFOLIATED DECIDUOUS TEETH

Stem cells isolated from human exfoliated deciduous teeth (SHED) may be cultured and showed differential expression of CD29, CD44, CD71, CD117, CD166. Cells did not show any signs

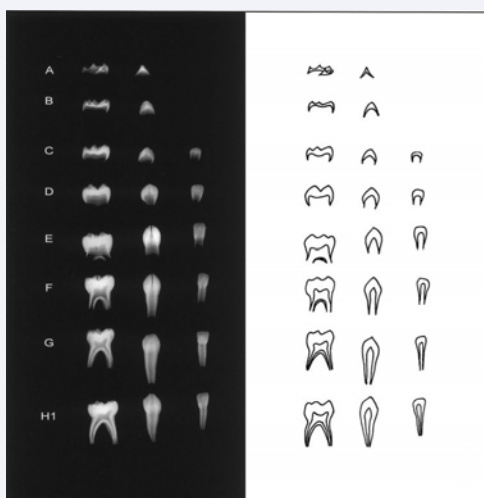


Figure 3 Evolution of teeth, from the crown to root formation [9].

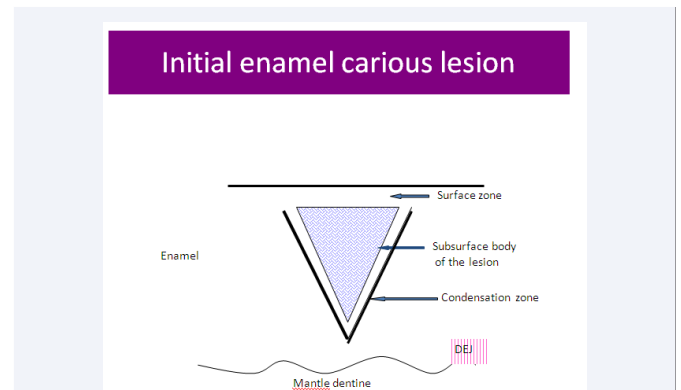


Figure 4 Schematic representation of the initial enamel carious lesion (white spot). Beneath the surface zone, the subsurface body of the lesion is found. Condensation zones are limiting the lesion. The dentino-enamel junction (DEJ) separate enamel from the superficial mantle dentine.

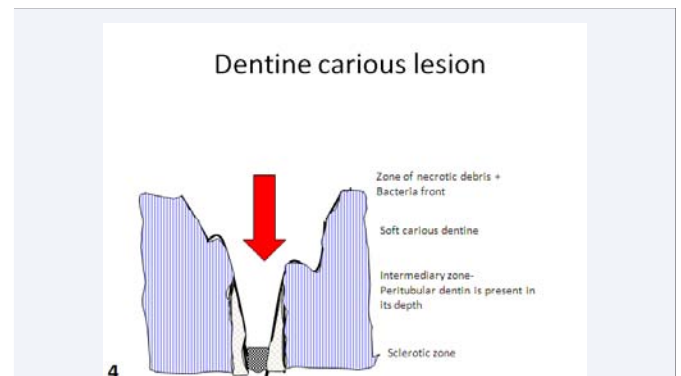


Figure 5 Schematic representation of a dentin carious lesion. In the infected zone, necrotic debris and bacteria are found. This is the soft carious dentine, demineralized, where tubules are enlarged. Beneath this layer, an intermediary zone is found, where peritubular dentine appears gradually. This is the affected zone. The sclerotic zone is present in the deepest part of the carious lesion.

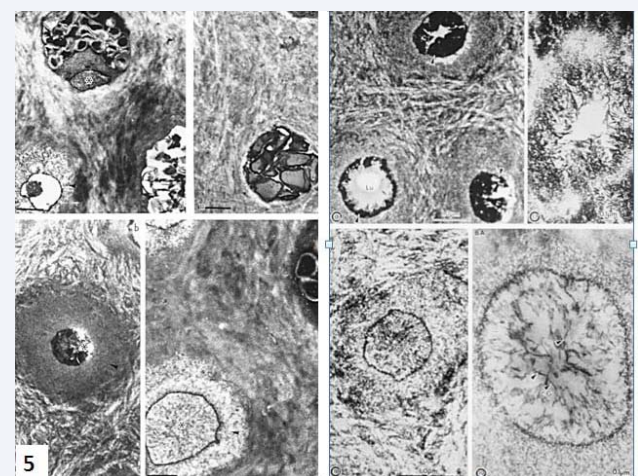


Figure 6 Non-active bacteria fill some tubules. Needle-like structures are visible inside the lumen of tubules. Non-apatitic structures fill the lumen of tubules located in the sclerotic zone.

Table 1:

Terminology	Cause of loss of hard tissue
Abrasion	Mechanical process involving foreign objects or substances
Demastication	Mechanical interaction between food and teeth
Attrition	Mechanical process involving tooth-to-tooth contact
Abfraction	Mechanical process involving tooth flexure-by eccentric occlusal forces
Erosion	Chemical etching and dissolution forces
Resorption	Biological degradation

Table 2: Descriptive criteria of crown and root stages.

A. Canine: Beginning of mineralization is seen as a cusp tip, which has not yet reached maximum mesiodistal dimension of the crown.
B. Incisors and canine: Mineralized incisal edge/cusp tip has reached maximum mesiodistal width of tooth.
Molars: Coalescence of cusp tips to form a regularly outlined occlusal surface.
C. Incisors and canine:
a) Enamel of incisal surface is complete. Approximal edges of forming crown have reached future contact areas.
b) Dentine is visible below incisal enamel.
Molars:
a) Enamel of occlusal surface is complete. Approximal edges of forming crown has reached future contact areas.
b) Dentine is visible below occlusal enamel and beginning along sides (however, dentine is not full-thickness).
D. Incisors and canine:
a) Enamel is complete down to approximal enamel-cementum margins, with full-thickness occlusal dentine present, and roof of pulp chamber is mature.
b) Beginning of root formation is seen as a dentine spicule approximally (both sides).
Molars:
a) Enamel is complete down to approximal enamel-cementum margins (not visible mesially if cusp of Zuckerkandl is present/pronounced), with full-thickness occlusal dentine present, and roof of the pulp chamber is mature.
b) Beginning of root formation is seen as a dentine spicule approximally (both sides).
E. Incisors, canine, and molars: Root formation is more than a spicule, but root length is less than crown height (measured approximally).
Molars:
a) Initial formation of root bifurcation is seen in the form of a mineralized point or semilunar shape.
b) Root length is less than crown height (measured approximally).
F. Incisors, canine, and molars:
a) Root walls are very thin, and root length is equal to or greater than crown height (approximal).
b) Root length is incomplete, with diverging apical edges.
Molars: Midway down root, root wall is thinner than root canal.
G. Incisors and canine: Root length is almost complete, but apical edges are parallel or slightly converging.
Molars:
a) Mesial root length is almost complete, but apical edges are parallel or slightly converging.
b) Midway down root, root wall is thicker than root canal.
H1. Root length complete, with apical walls converging, but apex is still open (width = 1 mm). Mesial root of mandibular molars, mesiobuccal root of maxillary molars.
H2. Apical dentine edge is sharp. Apex is only just visible/closed (width < 1 mm).
Table reproduced from Liversidge & Molleson [9]

of degeneration nor spontaneous differentiation. Compared with DPSC, SHED proliferation rate was about 50% slower, with slightly different phenotypes [11].

Regeneration occurs mostly within the pulp and the number of pulp stem cells is relatively small. The pulp includes fibroblasts, capillaries, nerves, and inflammatory cells. The number of stem cells is quite limited. However, stem cells have the potential for differentiation in various phenotypes (odonto/osteoblasts, chondrocytes, adipocytes, nerve and capillaries together with pericytes). In addition to pulp cells phenotypic differentiation, self-renewal and stemness are characteristics of pulp healing and regeneration.

This potential is now under focus, and considered to be used to regenerate the dental pulp or to cure pulp pathology. Doing so, it is expected to pave the way for regenerative therapy, and reduce or suppress conventional treatment of the carious disease.

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