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Editorial
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Enamel Aging

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Editorial

The dental enamel is a structure organized as a puzzle, and the pattern of enamel formation implicates the association of mineralized structures (96%), water (1% either coating the mineral, and 2%-3.6% being unbound to the crystallites, free water being mainly located in tiny intercrystallite spaces), and extracellular matrix proteins (ECM) (0.4-0.6%). The ECM includes a mixture of enamel structural proteins (amelogenins, ameloblastin, enamelin, amelotin, and odontogenic ameloblast-associated gene), enzymes [metalloproteinases (MMP-2, -3, -9), enamelysin (MMP20), kallikrein-related peptidase 4, chymotrypsin C, cathepsin C, adaptor protein complex-2, Lamp 1, Lamp2 , CD63], phospholipids and type XVII collagen.

Crosscut section of crystallites impliquates the association of hydroxyapatite monoclinic crystallites into hexagonal crystals. Theses sub-units display the following parameters a=b= 9.432, and c= 6.881 Å [1].

The thickness and width of human enamel crystallites are 263 Å ± 21.9 Å and 683 Å ± 134Å respectively for mature enamel crystallites [2]. The average crystal size has a diameter about 40 and 5nm. Lattice defects have been recognized during the initial crystal dissolution. Measurements of the hexagonal crystallite indicate a mean value of 50nm (30nm+70nm= 100 nm / 2= 50nm). As the size of an enamel crystallite is about 30nm x70 nm, a section at right angles of a crystallite includes about 2100 monocrystals. In length the average HAP crystal is 254.nm, with a thickness diameter of 12nm to~.20nm. In longitudinal sections, the crystals form thin ribbons, they are uninterrupted between the dentino-enamel junction and enamel surface, but fragments may result from sectioning.

Implicated by carious dissolution or by acidic attack, dislocations of the so-called Burger vectors are of two types. Screw dislocations are parallel to the c-axis, and/or edge-dislocations constitute the initial alterations along the outer crystal surface. This is occurring during the early stages of crystal dissolution. They are like the initial carious lesion [3]. Enamel crystals exhibited perforations in their centers and defects associated with the lateral surfaces. The perforations were situated near the central dark line of the crystallite. Hexagonal structures in transverse sections are tightly associated and form a homogeneous prismatic structure, including rods and interrod enamel.

Group of crystallites are associated within prisms measuring about 5mm, whereas interprisms width is about 0.5mm. They merge regularly each prism being composed by millions of tightly packed small crystallites. Remnants of enamel proteins are in the sheaths located at the surface of crystallites, and in the organic part (enamel sheaths organic-rich) located between rods and interrod structures. Enamel rods constitute cylindric-like structures. Enamel sheath separate rods from the interrod substance. They are forming a continuous network of six-sided honeycomb-like structure. The bulk of enamel is formed by rods and interrod structures, with crystallites forming a 60° angle between the two enamel structures. Therefore, preferential dissolution occurs along the c-axis, and eliminate either rods or interrod, depending on the crystal orientation. Therefore, the pattern of dissolution removes from enamel either rods or interrod or is apparently flat (Types I -3 etching pattern) [4]. A thin aprimatic layer is found in the inner part near the dentino-enamel junction (inner aprismatic enamel), and near the enamel surface (outer aprismatic enamel), where all the crystallites display a parallel orientation, almost perpendicular to the enamel surface.

Two groups of incremental lines are observed in enamel: The Hunter-Schreger lines (at right angles with the dentino-enamel junction, forming diazones and parazones) and striae of Retzius (or "Wilson" Bands). These increments lines cross the whole enamel layer, and end as perikymata at the enamel surface. The outer enamel is porous at the time of eruption, and is gradually filled with mineral, (issued from saliva, food and toothpastes) along the lateral faces of the crystallites. Structural variations in the surface zone were located mostly where prism reached the surface, but also where prisms were not discernible and those formed by a complex of both types. The thickness of the aprismatic border varied between 15 and 75mm. Cristal volume expands and gradually the internal spaces were closed. Some matrix proteins are resistant to protein degradation and persists. Metalloproteinases are involved in the biological activity of remnants of enamel proteins, displaying catalytic activity. In addition to the merging of HAP crystals, a thin layer of mineral is added to the lateral surfaces of crystals where water and matrix remnants are originally located. This accounts for enamel post-eruptive mineralization. The thin crystal plates gradually develop into hexagonal rods. In the most mature enamel, measurements

indicate 500 to 600 Å and 250-300Å in thickness. Ninety to 95% of the tissue volume is occupied by hydroxyapatite crystals. The crystals grow in width and thickness and replace fluids which had supplanted the degraded protein matrix.

The youngest crystals have the shape of long plates. They appear as parallel ribbons with 200-300 Å in width and a thickness about 10Å and later there is an increase in the size of the crystals. Whitlockite, brushite and other calcium phosphate crystals are found in carious lesion. In sound enamel, crystal thickness is about 275.9 Å, and crystal width 477.6.Å. Cross-sectional dimensions of carious lesion indicate a 328.4Å in thickness and 653.4 Å in width [5].

Enamel cracks are related to the surface toughness. Cracks are either running parallel to the occlusal surface or perpendicular to the inner layers. The outer old enamel has a lower fracture toughness and higher mineral content than the young enamel. This is compatible with the reduction in the interprismatic organic matrix observed during enamel maturation. The aspartic acid in human tooth enamel shows increasing racemization with age. 8% of the total aspartic acid will be the D-enantiomer after 60 Years [6]. In dentine and enamel, enrichment in D-aspartic acid content of 0.1% per year has been reported.

A complex series of hydroxyapatite monocrystal units are connected inside larger crystallites grouped into rods and interrod. These structures contribute to the early carious lesion (white spot lesion), and/or to the demineralization/re-mineralization process. They are contributing to cross-striations (Retzius lines), and Hunter-Schreger bands. Micro porosities, intercrystallite resin infiltration and the 30 m outer surface zone of enamel involve the complex enamel organization.

Dentin and Bone Aging

In addition to enamel aging, there is also an increase of mineralization in dentin and bone, including sclerotic dentin and an age-related apposition of cementum. Bone Nano-crystals hydroxyapatite are 1- 1.5nm thick x 5-25nm wide x 8-40 nm long [7]. Measurements for HAP indicate that a= 9.418 Å and c= 6.884Å. The size of dentin apatitic crystals is roughly 5x30x100nm.

Dentinal tubules measuring about 1.21± 0.08 mm (2-4mm) in diameter are surrounded by a hyper mineralized peritubular dentin, and between tubules, a softer collagen-rich intratubular dentin constitute the bulk of dentin. Mineral analysis indicates a carbonate-substituted hydroxyapatite. Peritubular dentin crystallites are characterized by small 250 Å equal-sized microcrystals [8], or a=36.00 nm(mean length), b=25nm (width) and c= 9.76 nm (thickness) [9].

The number of dentin tubules is about 18.000 and 21.000 tubules / mm2 [10]. Plate-like crystallites, 2-5 nm in thickness and 60nm in length display a needle-like appearance. Inside the lumen of the tubules, rhombohedral whitlockite crystallites have been identified. Dissolution and re-precipitation are occurring. Initial inorganic crystalline deposits consist of whitlockite with the presence of hydroxyapatite at later stages, and aggregates of calcium phosphate crystallites within the tubular lumen (Mg-substituted b-tricalcium phosphate, bTCP), brushite (calcium hydrogen phosphate dehydrate), amorphous and crystalline calcium phosphate. They have been found in aging dentin. Amorphous calcium phosphate is the first mineral deposited during the calcification process.

It is a metastable precursor of crystalline bone apatite. Dentin caries consists of six zones

i. The softened dentin,

ii. A zone of demineralization,

iii. A zone of early infection,

iv. A zone of dead tracts,

v. A zone of translucency and

vi. A zone of vital reaction.

Whitlockite completely occlude the dentin tubule, and sclerotic dentin constitutes a limit for the progression of the carious decay. Therefore, age is beneficial, making slower enamel and dentin development of the carious lesion.

Cementum

Three cementum types are characterized in human:

i. Acellular a fibrillar cementum,

ii. Cellular intrinsic fiber cementum containing cementocytes embedded in a collagenous matrix. (They are deposited by cementoblasts in the space between the Hertwig'root sheath and the dentinal surface),

iii. Acellular extrinsic fiber cementum [mainly found in the cervical and middle root portions, with faster growth rates on distal (4.3 mm/year) than on mesial (1.4mm/year) root].

Less mineralized than root dentin, the mineral phase is a hydroxyapatite with small amount of amorphous calcium phosphate (0.5-0.9% magnesium). Type I collagen constitute about 90% of the collagens. Type III coats type I collagen fibrils and account for only 5%. Bone sialoprotein (BSP) and osteopontin (OPN) bind to the collagenous matrices. OPN promotes adhesion and differentiation. It regulates bone cell differentiation and mineralization. Growth factors include BMP-2, -3 and -4, PDGF, a and b-FGFs, TGFb, PTH and IGF-I [11]. Two growth factors are also implicated in cementum formation: the cementum-derived attachment protein (CAP) and the purified cementum growth factor (CGF). With age the cementum layer increases in thickness and may compensate tooth abrasion. Enamel, dentin, bone and cementum are subjected to variations reflecting aging and maturing occurrences.

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