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Review Article

Enamel and Dentin Carious Lesions

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Abstract

A decrease in calcium, phosphorus and carbonate composition was established in the initial lcarious lesion, whereas magnesium was increased both in the carious enamel translucent and dark zones. Non-apatitic phases were detected in arrested carious dentin, characterized as weddellite, whewellite, calcite, brushite, whitloockite and octocalcium phosphate. Matrix metalloproteinases (MMPs) and cysteine cathepsins are endopeptidases degrading all extracellular components during caries degradation. After an initial lesion (white spot), the carious disease expands throughout the whole enamel thickness. Afterward, the carious lesion reaches the dentino-enamel junction and penetrates into dentin, crossing the mantle dentin, and then infiltrating the outer part of circumpulpal dentin. Lesions are active or arrested, and this evolution determines the speed of caries progression. Dentin-like bridges of reactionary or reparative dentins are formed along or inside the pulp. The lumens of the dentinal tubules are occluded by sclerotic re-precipitations. From the outer to the inner dentin layers, two carious zones are found: 1) the body of the lesion (25-50 % porosity) located approximately 15 to 30 m beneath the overlying intact enamel surface zone (1 to < 5% pore volume) forming the bulk of the decay, and 2) the translucent zone (with a loss of about 1-2 % mineral). More inwardly, two zones were also identified: 1) the dark zone (5-10 % porosity) situated beneath the sclerotic zone and 2) the translucent zone limitting the lesion. The carious lesions may be classified into a superficial lesion [dental plaque & acquired pellicle, in close association with the surface layer (2 - 3 m)], deep lesions (between 3 - 5 m) and very deep lesions, leading to pulp exposure, and subsequenly to pulp inflammation and necrosis.

INTRODUCTION

The structure of the enamel and dentin carious lesion implicates a series of demineralized layers associated with remineralized zones. After an initial carious lesion (white spot) located in the outer enamel, susceptible to heal spontaneously, the lesion is expanding throughout the whole enamel thickenss. At this stage, the carious lesion reaches the dentino-enamel structure and penetrate into dentin, crossing the mantle dentin, and then, diffusing into dentin due to its tubular structure. At very early stage, the enamel lesion is reversible. Afterward, enamel collapse and an open cavity is formed. The active carious lesions involve a surface zone and subsurface porosities. Inactive (or arrested) lesions have a sawtooth appearance, but in the subsurface, there is a mineral loss. A true subsurface remineralization is rarely achievable, since the surface zone is acting as a diffusion barrier **[**1**]**.

In dentin, the carious lesion reacts to the stimulus of the biofilm, accompanied in circumpulpal dentin by the subjacent sclerotic dentin formation **[**2**]**. Dental tissues (enamel, dentin, and cementum) are the relevant oral solid surfaces. The primary colonizers and secondary organisms generate a matrix of exopolymer within which bacteria grow.

From the outside ot the tooth toward the inner circumpulpal dentin, a surface layé is found, where food debris (muscle fibrils, collagne fibers, vegetal cell walls) accumulate. Bacteria colonies increase in thickness. The carious dentin is increasing in size, diffusing in the subjacent tissue throughout the dentin tubules. Gradually, the lesion invades the coronal dentin and expands in the direction of the dental pulp. Active or arrested lesions are determining the speed of caries progression. Beneath a sclerotic layer, followed by an apparently sound dentin layer, reactionary dentin is formed along the pulp or in case of a pulp exposure, a reparative dentin-like bridge is created, occluding the pulp perforation.

In the carious dentin, three stages have been identified :

1. Early microbial invasion of the carious dentin,
2. Early spreading along the DEJ, weakening gradually the sound enamel,
3. The early onset of an irreversible pulp response.

The early pulp response is limitted and still reversible. Slowly progressing lesions stimulate reparative or reactionary dentin formation, beneath a calcio-traumatic line. It may stimulate osteodentin-like formation. Later, the rate of caries progression is reflected by the quantity and quality of the tertiary dentin, creating a dentin layer resembling to normal tubular dentin. Rapidly progressing lesions lead to the production of atubular dentin or/and to the complete absence of tertiary dentin, as well as pulp necrosis involved in apical pathology **[**1**]**.

Dental caries involves interactions between 1) the tooth structure, 2) the microbial biofilm formed at the tooth surface and

3) sugars. Saliva composition and genetic regulations influence the progression of the lesion. Caries are a dietary carbohydrate-modified bacterial infectious disease. Its key feature is a dietary carbohydrate-induced enrichment of the plaque microbiota with organisms such as *Streptococcus Mutans* (SM) and lactobacilli which causes an increase of plaque's pH-lowering and displays cariogenic potential. A broader concept includes a major role of saliva in the regulation of the exposure of tooth surfaces to carbohydrate, cariogenic potential of dental plaque, its acidity, microbial composition, and pH lowering **[3]**.

Protective factors promote remineralisation and lesion arrest, whereas pathological factors shift the balance in the direction of disease progression. The dental hard tissues are exposed to the oral environment. They are the targets of the carious process and all tooth surfaces are susceptible throughout an individual's lifetime to such lesions. However, caries will not occur in the absence of a cariogenic dental biofilm and frequent exposure to dietary carbohydrates, mainly free sugars. Therefore caries must be considered as a microbial disease.

THE INITIAL ENAMEL CARIOUS LESION (WHITE SPOT)

Dental caries can be best described as a complex biofilm-mediated disease that can be mostly characterized by behaviours involving frequent ingestion of fermentable carbohydrate (sugars such as glucose, fructose, sucrose, and maltose) and poor oral hygiene, in combination with inadequate fluoride exposure.

Although a wide range of organic acids can be generated by dental biofilm microorganisms, lactic acid is the predominant end-product from sugar metabolism and is considered to be the main acid involved in caries formation. Once sugars are cleared from the mouth by swallowing and salivary dilution, the biofilm acids is neutralized by the buffering action of saliva. The pH of biofilm fluid returns toward neutrality and becomes sufficiently saturated with calcium, phosphate, and fluoride ions so that demineralization stops and re-deposition of mineral (remineralization) is favoured. Due to the dynamic nature of the disease process, the early clinical stages of caries can be reversed or arrested, especially in the presence of fluoride.

When the pH drops down, the rate of mineral loss becomes greater in the subsurface than at the surface. The formation a subsurface lesion results from this early stage. When sufficient mineral is lost, the lesion appears clinically as a white spot. Dental caries is a disease involving repeated cycles of demineralization and remineralization throughout the day. Saliva plays a critical role in maintaining this beneficial microbiota by buffering the oral environment at a neutral pH (optimal for the growth and metabolism of most of the oral microbiota), while providing proteins and glycoproteins as nutrients.

The oral microbiota grows on surface as structurally organised communities of interacting species, termed dental plaque. Tooth surfaces are covered by a conditioning film of proteins and glycoproteins (the acquired *pellicle*) that are derived mainly from saliva, but also contains components from bacteria and their products. The acquired pellicle provides binding sites for adherence by early bacterial colonizers of the tooth surface leading to dental biofilm formation, and acts as physical barrier preventing acid diffusion.

Early studies of caries lesions found higher proportions and incidence of SM and S. sobrinus compared with sound enamel. Lactobacilli were also isolated from advanced lesions. Subsequent laboratory studies confirmed that other bacteria found within dental biofilms could also generate a low pH from sugars, whereas others could reduce the potentially damaging effect of lactic acid by converting them to weaker acids, or by generating alkali from the metabolism of arginine or urea in saliva. These findings provided support for the 'non-specific plaque hypothesis', in which caries is a consequence of the metabolic activity of the biofilm. Microorganisms can be present in biofilms on sound enamel, but at a level or activity that is too low to be clinically relevant **[4]**. It has been shown that caries susceptibility is high just after eruption and decreases afterwards.

The dental enamel is composed of hydroxyapatite (92-94%), water (2-3%), carbonate (2%), trace elements (1%), fluoride (0.01-0.05%), proteins and lipids (<1%) (Table 1).

The dental enamel is organized into rods-like (or prisms), with a width measuring between 4 and 6 µm. Rods extend from the dentino-enamel junction up to the enamel surface. Interrods (or interprismatic substance) are located between rods (0.5µm in width). Each crystal of hydroxyapatite has a hexagonal configuration with a basal face that is 25nm (α-axis) x 40nm (β-axis). Residual marix wrap up each HAP crystal. In sound enamel the dental hydroxyapatite has a ratio of Ca/P= 1.61 to 1.64. Fluorohydroxyapatite, fluoroapatite, calcium-deficient HaP, dicalcium phosphate dihydrate, tricalcium phosphate, octacalcium phosphate are also found (Table 2).

Calcium may be replaced with magnesium, sodium, zinc, selenium and strontium. Phosphate may be substituted by carbonate and acid phosphate. While fluoride stabilizes mineral crystals, some of these other substitutions (magnesium, sodium, selenium, carbonate, acid phosphate) destabilize the crystals, and there is an increase in caries susceptibility. Tunnels inside the apatitic crystallite may contribute to ion exchanges or diffusion, and fluoride penetration.

After a zone of degradation, a zone of bacterial invasion is found (infected caries), followed by a zone of demineralization, a zone of dentin sclerosis and fatty degeneration (affected caries and sclerotic dentin – transparent dentin). In addition to apatitic crystals, a second group of crystals have been identified as Mg-substituted β-TCP crystals formed by whitlockite, brushite and calcium phosphate.

Post-eruption maturation of enamel

The enamel surface undergoes maturation by exchanging

Table 1: Composition of sound enamel.

S.no		In Weight	In Volume
1	Mineral Phase	96%	87-91%
2	Organic Phase	0%	2%
3	Water	Bound: 3.4%	
		free: 1%	7-11%

more soluble mineral components for less soluble mineral. Plaque is supersaturated with respect to calcium and phosphate and contains increased fluoride levels compared with saliva. During episodes of acidogenic challenge, more soluble carbonate-rich HAP is replaced by more acid-resistant FHAP. In the presence of fluoride ion availability, it is possible for fluoride to be acquired by 1) adsorption of fluoride onto HAP crystals; 2) exchange of fluoride with hydroxyl groups in HAP; 3) dissolution and reprecipitation of HAP to form FHAP; 4) and precipitation of mineral phases with FHAP crystal growth.

The organic material and water content allow soluble fluids from the plaque to penetrate enamel up to a depth of 200 μm. With post-eruption maturation, access to the underlying enamel is restricted to the outer most 20 nm of enamel. This may reflect preferential dissolution of the more soluble mineral phases and replacement with less soluble mineral phases of larger cross-sectional crystal diameter than the original crystals, effectively reducing the organic matrix space and decreasing permeability.

Partial dissolution of dental hydroxyapatite occurs with exposure to organic acids derived from acidogenic bacteria in the dental plaque or ingestion of acidic beverages and foods. The caries process is a gradual one that requires repeated episodes of prolonged exposure to acidic conditions consistently below the critical pH for enamel dissolution (pH 5.5, demineralization) with intervening periods of return to the resting pH of plaque (pH 7.0, remineralization period).

Subsurface white spot lesion formation

Two zones of demineralization are present: 1) the translucent zone (with a loss of about 1% -2 % mineral or 1 % pore volume) along the advancing front of the lesion; and 2) the body of the lesion (25-50 % porosity, >5 to 25 % pore volume) representing the majority of the lesion and situated approximately 15 to 30 nm beneath the overlying intact enamel surface.

Two zones of remineralization are also present: 1) the dark zone (5-10 % porosity, or 2-4 % pore volume) situated near the advancing front just superficial to the translucent zone; and 2) the surface zone (1 to < 5% pore volume) forming the intact surface overlying the lesion. It is possible to create artificial or caries-like lesions in enamel that mimic naturally occurring white spot lesions. The presence of increased levels of fluoride in plaque favors reprecipitation of dissolved mineral. Increased fluoride content of native enamel in the form of FHAP would lessen the extent of demineralization and favor mineral reprecipitation **[**5**]**.

Edge dislocation indicates the loss of one or more unit cell(s) within the Burger's circuit. Screw dislocation splits the crystal in two. Small angle boundaries, atomic vacancies and atom rotations are also seen near the white spots. Demineralization of enamel crystals is synonimous with dissolution of HAP crystals. Dissolution occurs either at the periphery or in the center of the crystals. The central perforations form first, spread and fuse with adjacent crystals. The central perforation varies in shape, but can be triangular, rhombohedric, trapezoidal or hexagonal.

Different forms of remineralization can be observed after partial dissolution. Addition of apatite unit cells can cause smoothing of formerly saw-tooth dissolution edges of the peripheral regions of dissolved crystals Small crystals with elongated hexagonal configurations similar to normal ones can be observed to repair defects on crystal surfaces.

In the case of a central perforation, initially a few small crystals may appear in the hole site. New crystal formations are observed at all areas of carious enamel lesions. They appear in lesions on the outermost surface layers as well as in large intercrystalline spaces in subsurface lesions of the demineralized layer. Usually smaller than the original enamel crystals, on their c-axes, newly formed crystals are either regular hexagonal or elongated hexagonal. Lattice-striation intervals are different: 8.12 Å in regular-hexagonal crystals and 8.17 Å in elongated-hexagonal crystals. In addition, newly formed whitlockite, brushite and other calcium phosphate crystals can be found in carious lesions. Demineralization and remineralization occur constantly, either simultaneously or alternately, on enamel surfaces exposed in the oral cavity. When the remineralization rate is greater than the demineralization rate, post-eruptive maturation occurs. When the demineralization rate is greater than the remineralization rate, caries develops **[**6**].**

After one week of undisturbed biofilm formation, no changes in the enamel were seen clinically, even after samples had been careful air-dried. However, at the ultrastructural level, there were signs of direct dissolution of the outer enamel surface. This was seen as an enlargement of the intercrystalline spaces due to partial dissolution of the individual crystal peripheries.

After two weeks the enamel changes are visible clinically when samples were air-dried. After three to four weeks, these changes could be seen, the lesion being opaque with a matte surface. Ultrastructurally, there was a complete dissolution of the thin perikymata overlappings; marked dissolution corresponding to developmental irregularities such as Tomes' processes, pits, and focal holes; and a continued enlargement of the intercrystalline spaces. These zones are the surface zone and the body of the lesion. These changes were best seen after imbibition of sections in water (Table 3).

A protective role of salivary proline-rich proteins and other salivary inhibitors, such as statherin, has also been emphasized. They inhibit demineralization and prevent crystal growth. These macromolecules cannot penetrate the deeper parts of the enamel; therefore, their stabilizing role is limited to the surface enamel alone. The outer enamel is special in terms of its ultrastructure and chemical composition, but is unlikely to play a significant role in caries lesion initiation.

Caries on an occlusal surface are also a localized phenomenon in the deepest part of the groove-fossa system, where the bacterial accumulations receive the best protection against functional wear. The lesion forms in three dimensions, again guided by prism direction. The lesion assumes the shape of a cone, with its base toward the enamel-dentin junction. It appears that the active biofilm is above the entrance of the narrow fissures and grooves.

Clinical caries diagnosis

The assumption that lesion activity, defined as progression or regression, will be reflected at the surface of the lesion: and

Table 2 : Components of the sound enamel organic matrix.		
1	Amelogenins	
2	Non-amelogenins	
a	Enamelin	
b	Ameloblastine (ameline,sheathline)	
c	Tufteline	
d	Amelotin	
e	Enzymes	MMPs:MMP=2,MMp-3,MMp-20,MT1-MMP
		Serine protease 17:kallikreine 4KLK4
		Phosphatases acide et alcaline
f	Proteins of the serum	
g	Glycoproteines et proteoglycans (GAGs)	GAGs
		DCN, BGN
		Glycoproteins sulfated
		BGN : interaction with amelogenins
h	Lipides et phospholipides	Phospholipides membranaires
i	Proteines with transitory expression (DSPP, DSP, DMP-1, BSP)	
j	Proteines binding calcium	Annexines
		Calbindines, calmoduline (EF-hands)

Table 3: Mineral lost from each zone of caries lesions in human (Reported from [7]).					
S.no		**Sound Enamel**	**Translucent zone**	**Dark zone**	**Body of the lesion**
1	Calcium	37%	≈ 30%	≈35%	37%
2	Phosphorus	18.50%	≈13%	≈ 16.6%	18.50%
3	Carbonate	2-4%	≈ 28%	≈3%	≈1%
4	Magnesium	0.2-0.4%	≈ 2%	≈3%	≈0.16%

Table 4: Composition of sound dentin.		
1	Mineral phase	70% (carbonated hydroxyapatite and magnesium)
2	Organic phase	20%
3	Water	10-12%

defined as matt, or 'chalky'. Rough enamel lesions are 'active', and shiny, whereas smooth enamel lesions are 'inactive' or 'arrested'. However, it is difficult to differentiate between an active diseased enamel and an inactive « sound » surface, due to insufficient plaque removal prior to the examination. These effects are most pronounced for 'active' non-cavitated lesions supporting the notion that fluoride exerts its predominant effect on the active caries process. 'Active' non-cavitated lesions had a considerably greater risk of progressing to a cavity than 'inactive' noncavitated lesions. 'Inactive' lesions do not need professional treatment whereas 'active' lesions because of their progressive nature demand professional treatment **[7]**.

DENTIN CARIOUS LESION

The dimensions of HAP crystallites in dentin are the following: α- and β-axis =3nm, whereas HAP has 60nm in length.

The superficial carious dentin lesion includes two layers : a superficial infected layer (necrotic zone), crossing the dentino-enamel junction (DEJ) and including the mantle dentin. The carious lesion occurrs along the DEJ and enlarge the interface between enamel and dentin. Degradation of this superficial layer leads to accumulation of bacteria. The superficial dentin is altered (soft carious dentin, or decalcified layer), and collagen fibers are partially destroyed by endogenous and bacterial metalloproteinases (MMP-1, MMP-2, MMP-8, MMP-13, MMP-14, and MT1-MMP). These enzymes cleaved the collagen fibrils into ¼ and ¾ segments. Cathepsin K cut both tooth helical C- and N-terminals, and remove the telopeptides from the collagen fibers. Cysteine cathepsin degrade type I collagen, laminin, fibronectin and proteoglycans. MMPs and cysteine cathepsins are co-distributed in dentin, showing proteolytic activities. CT-K and MMP-2 are both active, synergically.

Active lesions differs from arrested dentinal caries by its degree of pigmentation. Active carious lesions are characterized by viable bacteria within the tubules, impermeability to dyes and isotopes, and a lower calcium content and hardness than the arrested decay [8**]**.

The lumens of the sclerotic **layé** in an arrested carious lesion are almost filled with dense calcified material. The intertubular zone is hypermineralized, and in continuity with the peritubular dentin. Reactionary and reparative dentin are formed within the pulp, in front of the carious lesion **[**9**]**.

Table 5: Composition of the sound extra cellulaire matrix of dentins.

Collagènes 90%		**Collagen : type I (89%) + I trimer (11%)**	**+ 1-3% type III et V collagens**
Protéines non-collagéniques 10%	Phosphorylées	SIBLINGs	**DSPP** (entre 155 et 95kDa) cleavage in:
			>**DSP** (N-terminal- proteoglycan forming dimères) : 100- 280kDa
			DGP ✓
			DPP ✓
			DMP-1 ✓
			BSP : ✓
			OPN ✓
			MEPE ✓
	=	Amélogenins	
	=	Other matrix molecules	Ameloblastine
	Non-phosphorylées	Ostéocalcine DPG : Dentin gla-protéine (acide g carboxyglutamique)	
		Matrix Gla Protein (MGP)	
	=	Osteonectine- SPARC	
	=	Proteines of the sérum	Albumin
			α2-HS glycoproteine
	=	SLRPS	CS/DS PGs: decorine, biglycan
			KS PGs: lumican, fibromodulin, osteoadherine
	=	Facteurs de croissance	FGF2, TGFβ1, BMPs, ILGF I & II, PDGF
	=	Enzymes	Phosphatase alcaline, acid phosphatase, serine proteases
			Collagenases: MMP-1, 8, 13
			Gelatinases A: MMP-2,
			B: MMP-9
			Stromelysins 1: MMP-3
			MT1-MMP, enamelysin: MMP-20
			ADAMs et ADAMTS
		Proteolipides	Phospholipides membranous
			and phospholipids present in the extracellular matrix

Called 'tubular sclerosis' or 'translucent dentin', the carious dentin appears translucent when examined in transmitted light. It has been shown that it never extend beyond the limits of the enamel lesion contact area with the dentino-enamel junction. The other important defense reaction is the presence of dead tracts, and the formation of reactionary dentin. This may begin before the bacterial invasion of dentin.

Once the cavity is directly exposed to bacteria, superficial tubular invasion occurs. The most superficial part of the dentin becomes the zone of destruction.

Beneath this zone, tubular invasion by bacteria is seen. With rapid lesion progression, the odontoblastic processes are destroyed without having produced tubular sclerosis. They are called dentin dead tracts. These empty tubules are invaded by bacteria, and groups of tubules may coalesce to form liquefaction foci. This area is called the zone of bacterial penetration. In the sclerotic dentin, the translucent zone is a zone of demineralization resulting from acid demineralization.

With respect to inorganic composition, a decrease in calcium, phosphorus and carbonate composition was established, whereas magnesium was increased both in the carious enamel translucent and dark zones **[**10**]**.

In the sound enamel and dentin, inorganic components are mostly formed by hydroxyapatite mineral (96% and 80%, respectively). Free and bound water are forming 3.6-4% of enamel and 12% of sound dentin. In sound enamel, 0.4-0.6% is composed by extracellular matrix organic components (amelogenin and other molecules, and lipids). Non-apatitic phases were detected in arrested carious dentin, characterized as weddellite, whewellite, calcite, brushite, whitloockite and octocalcium phosphate.

Type I and III collagen and non-collagenous molecules formed the bulk of dentin matrix composition (SIBLINGs, non

phosphorylated proteins, serum derived molecules, enzymes proteases). Matrix metalloproteinases (MMPs) and cysteine cathepsins are endopeptidases degrading all extracellular components during caries degradation (Table 4,5).

Carious dentin has a pH lower than that in sound dentin. Dentin from active lesions showed a mean pH of 4.9, and the dominant acid was lactate. Arrested lesion showed a higher pH 5.7 with acetate and propionate as the dominant acids. The pH in the dentin beneath a restoration was similar to those of an arrested lesion **[**11**]**. Pigmentation differs between active and arrested caries lesions, the arrested lesion being darkly pigmented, in contrast with the active lesion that is white/yellowish. Distinction between active and inactive caries lesions can be made on the basis of a combination of visual and tactile criteria. The transition of an active lesion into an arrested/inactive lesion is accompanied by characteristic changes of the surface features of the lesion. Thus, the typical initial active caries lesion exhibits a whitish opaque appearance with a rough surface whereas the active lesion of root/dentin is soft or leathery and discoloured (Table 6) [12-15].

Root caries is similar to enamel caries in being a subsurface demineralization, but, unlike enamel caries, the surface may appear softened at an early stage of lesion development. Bacteria penetrate at an earlier stage than in coronal caries. Secondary or recurrent caries is primary caries at the margin of a restoration **[**2,16**]**.

Treatment of deep dentin caries lesions is using the excavation of deep caries lesions. Indirect pulp capping and the stepwise excavation approach are supposed to make a change within the cariogenic environment. The lesion is clinically arrested, resembling a slowly progression lesion **[**17**]**.

In the two layers of carious dentin the superficial first layer is characterized by extensive decalcification, degenerated collage fibers and it is physiologiquement unrecalcifiable. In the underlying second layer, the decalcification is not total. The cross-linkage of collagen showed a change in the cross-linkage. The first later showed virtual disappearance of cross-links indicating irreversible denaturation of collagen. The second may be recalcifiable **[**18**]**.

The techniques available to excavate demineralised dentine can be classified as mechanical, rotary (handpieces & burrs) and -mechanical, non-rotary (hand excavators, air-abrasion, air-polishing, ultrasonic, sono-abrasion, ultrasonic, sono-abrasion), chemo-mechanical methods (Caridex, Carisolv, Enzymes), and photo-ablation (lasers) **[**13**]**. Chemochemical caries removal involves the chemical softening of carious dentin, followed by it eviction by gentle excavation. Mixing aminoacids with sodium hypochlorite. N-monochloroamine acids degrade demineralized collagen in carious dentin. The dentin surfacee is irregular, and suitable for the bonding of a composite resin or a glass ionomer. Originally marketed in solution (Caridex), it is now a gel requiring volumes of 0.2-1ml accompanied by specially designed instruments **[**19**]**.

Secondary caries is the lesion appearing at the margin of an existing restoration. These are called “wall lesions,” and they are the result of microleakage. They are different from inactive residual caries. The diagnostic of secondary caries constitute the main reason for replacing fillings **[**20, 22**]** (Figure 1,2).

Clinical diagnosis of recurrent caries [21, 22].

The term “recurrent caries” denotes caries at the margin of restorations. Recurrent carious lesions are most often located on the gingival margins of Class II through V restorations. Recurrent caries is rarely diagnosed on Class I restorations. It is important to differentiate recurrent carious lesions from stained margins on resin - based composite restorations. The term “secondary caries” is used more commonly than “recurrent caries” for caries that has developed adjacent to margins of restorations. The percentage of restorations in adults that were replaced because

Table 6: Description of the caries diagnostic criteria.

Score	Category	Criteria
1	Sound	Normal enamel translucency and texture (slight staining allowed in otherwise sound fissure).
2	Active caries	Surface of enamel is whitish/yellowish opaque with loss of intact surface feels rough when the tip of the probe is moved gently across the surface; generally covered with plaque. No clinically detectable loss of substance. Smooth surface: Caries lesion typically located close to gingival margin. Fissure/pit: Intact fissure morphology; lesion extending along the walls of the fissure.
3	Active caries surface discontinuity	Localized surface defect (micro cavity only in enamel) No undermined enamel or softened floor detectable with the explorer
4	Active caries	Enamel/dentin cavity easily visible with the naked eye; surface of cavity feels soft or leathery on gentle probing. There may or may not be pulpal involvement
5	Inactive caries	Surface of enamel is whitish, brownish or black. Enamel may be shiny and feels hard and smooth when the tip of the probe is moved gently across the surface. No clinically detectable loss of substance. Smooth surface: Caries lesion typically located at some distance from gingival margin.
6	Inactive caries	Fissure/pit: Intact fissure morphology; lesion extending along the walls of the fissure. No undermined enamel or softened floor detectable with the explorer
7	Inactive caries	Enamel/dentin cavity easily visible with the naked eye; the surface of cavity may be shiny and feels hard on probing with gentle pressure. No pulpal involvement.
8	Filling (sound surface)	Caries lesion may be cavitated or non-cavitated
9	Filling + active caries	Caries lesion may be cavitated or non-cavitated
10	Filling + inactive caries	Caries lesion may be cavitated or non-cavitated

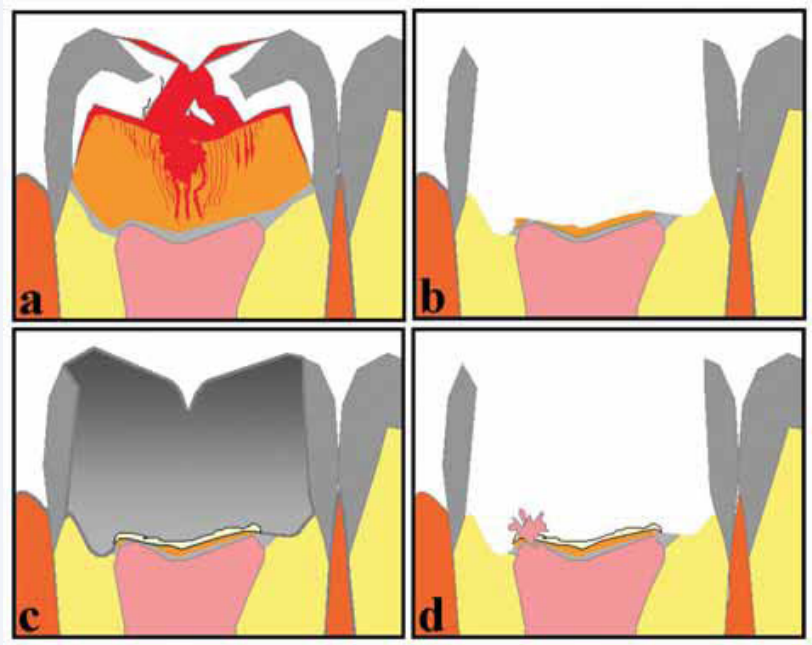

Figure 1 Indirect pulp-capping procedure.

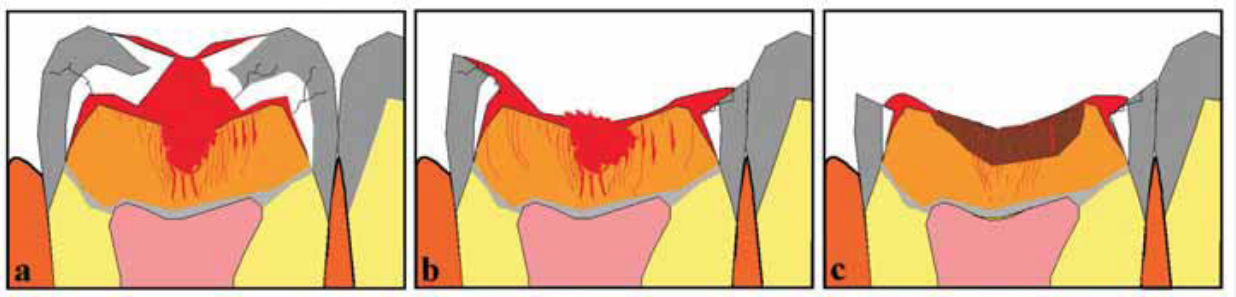

Figure 1 Indirect pulp-capping procedure.

of the clinical diagnosis of recurrent caries was about 50 percent, with a range of 45 to 55 percent. The percentage was somewhat more for amalgam than for resin-based composite restorations, and it was somewhat less for restorations in primary teeth because of the relatively high percentage of bulk fractures of restorations in these teeth and their short life spans. Recurrent caries and discoloration of resin-based composite restorations combined represent a higher percentage of replacements than do recurrent caries for amalgam restorations alone. The restorations replaced as a result of the diagnosis of recurrent caries is much higher in general dental practice than in controlled clinical trials in which recurrent caries represents 2 to 3 percent of the failures. Therefore, it is important to analyze the available knowledge on the nature of recurrent caries and explore possible preventive and alternative treatments to replace restorations that have received this diagnosis. It must be recognized that discoloration is one sign of carious lesions; another is the softening of the tissues, including disintegration and eventually cavity formation. As the lesion reaches dentin, the wetness of the lesion also is a relevant clinical criterion. These characteristics of carious lesions 1) softening of the tissues, 2) discoloration and 3) wetness of the lesions, are essential for differentiating active from arrested carious lesions.

Pulpal response to caries

There is no pulpal response to early dental caries under enamel caries **[**22**]**. Changes in the pulp have been observed beneath deep active caries.

For example, mannose- and scavenger-receptors are classic receptors for phagocytosis expressed on neutrophils and macrophages. These phagocytes also exhibit certain opsonin receptors for C-reactive protein, fibronectin, and complement 3b (C3b) to facilitate internalization. Another group of receptors are G protein-coupled receptors (GPCRs) and toll-like receptors (TLRs). They do not participate in the ingestion of microbes but activate phagocytic functions. GPCRs bind to chemokines, lipid mediators [platelet activating factor (PAF), prostaglandin E2, leukotriene B4] or bacterial proteins, which results in the extravasation of leukocytes and the production of bactericidal

Table 7: Components of pulpal innate immunity.	
1	Dentinal fluid and immunoglobulins
2	Odontoblasts
3	Neuropeptides and neurogenic inflammation
4	Innate immune cells (not Ag specific)
5	Lymphocytes: NK cells, T cells
6	Immature DCs, pulpal DCs
7	Monocytes and macrophages
8	Innate cytokines
9	Chemokines

substances. Binding of lipopolysaccharide (LPS) to TLR-4 or lipoteichoic acid (LTA) to TLR-2 leads to the induction of chemokines, cytokines, and up-regulation of T cell co-stimulatory molecules (CD86, CD80, CD40), which are important molecules in adaptive immunity (Table 7).

The dental pulp is equipped to provide an innate response to invading caries bacteria, which can theoretically slow down bacterial invasion. Persistent infection leads to the activation of adaptive immunity and overwhelming inflammation **[**23-25**]**.

CONCLUSION

The carious enamel and dentin lesions constitute the basis of pediatric treatments, leading to indirect or direct pulp capping, followed by restorative procedures. In the early carious enamel, fluorohydroxyapatite, fluoroapatite, calcium-deficient HaP, dicalcium phosphate dihydrate, tricalcium phosphate, and octacalcium phosphate are found. Dentin MMPs and cysteine cathepsins are implicated in the development of the carious decay and in the extracellular matrix degradation. Differences between the infected and affected carious dentin leads to different type of treatment. Superficial and deep carious lesions conduce to the formation of reactionary dentin (tubular or atubular dentin, orthodentin or osteodentin), whereas deep lesions are leading to a pulp exposure. The different evolution implicates the closure and sealing of the pulp orifice closed by reparative osteodentin formation and/or diffuse pulp mineralization.

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