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Dental caries: a dynamic disease process

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ABSTRACT

Dental caries is a transmissible bacterial disease process caused by acids from bacterial metabolism diffusing into enamel and dentine and dissolving the mineral. The bacteria responsible produce organic acids as a by-product of their metabolism of fermentable carbohydrates. The caries process is a continuum resulting from many cycles of demineralization and remineralization. Demineralization begins at the atomic level at the crystal surface inside the enamel or dentine and can continue unless halted with the end-point being cavitation. There are many possibilities to intervene in this continuing process to arrest or reverse the progress of the lesion. Remineralization is the natural repair process for non-cavitated lesions, and relies on calcium and phosphate ions assisted by fluoride to rebuild a new surface on existing crystal remnants in subsurface lesions remaining after demineralization. These remineralized crystals are acid resistant, being much less soluble than the original mineral.

Key words: Dental caries, remineralization, demineralization.

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INTRODUCTION

What is dental caries and what do we know about it?

Dental caries is commonly known as tooth decay. In the minds of the lay person, and surprisingly even within dentistry, dental caries is often thought of as holes in the teeth rather than an entire disease process. However, it has been known for over 100 years that dental decay is caused by bacteria fermenting foods, producing acids and dissolving tooth mineral. In recent decades the process has been much better defined from several aspects including microbiology, saliva, tooth mineral composition, tooth ultrastructure, diffusion processes, kinetics of demineralization, the reversal of demineralization that is known as remineralization, and factors that contribute to the reversal of the process.[1–12] Now, we have a rather deep understanding of what goes on in the mouth, but this knowledge is far from being effectively utilized in dental practice.

The so-called cariogenic bacteria are essential to the disease process. At least two major groups of bacteria, namely the mutans streptococci and the lactobacilli species, are able to produce organic acids during metabolism of fermentable carbohydrates by these bacteria.[5,12–14] The acids produced include lactic, acetic, formic and propionic, all of which have been shown to readily dissolve the mineral of the enamel and dentine.[2,15,16] Bacteria which produce acid as a by-product of their metabolism are known as acidogenic, and some, such as the groups named above, are aciduric and can live in an acid environment.

When the organic acids are produced by the bacteria in dental plaque on the tooth surface they readily diffuse in all directions and of course diffuse through the pores of enamel or dentine and into the underlying tissue. As the acid diffuses into the tooth it finds acid soluble mineral and begins to dissolve it.[2,17] If this process progresses long enough, the end result is a cavity. This process in the mouth usually takes many months or years to progress to cavitation, the end-point of the disease process known as dental caries.

Dental caries is a transmissible bacterial disease caused primarily by the bacteria listed above feeding on the carbohydrates taken into the mouths of humans.[11,12] The so-called cavity or hole in the tooth is the end-point. Bacteria are transferred to babies from mothers or caregivers very early in the child's life, with colonization of soft tissues possible even before the teeth erupt.[8,10,18] As the teeth erupt the cariogenic bacteria colonize them, establish as dental plaque, and the cycle of destruction begins.

The process of dental caries is now very well understood, as outlined above. There is a good

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understanding of how demineralization and remineralization occur, and the role of fluoride in these processes.[7] The mineral of our teeth and bones is a defect-containing carbonated hydroxyapatite with a complex crystal structure that is readily dissolved in acid.[19,20] Many of the functions of the salivary proteins have been established together with the very important protective role of saliva in the reversal or arrestment of the caries process.[3,21] Saliva buffers the acids, provides a saturated solution of calcium and phosphate to inhibit demineralization and produce remineralization, has several antibacterial components, and carries calcium in a soluble complexed state.[3,21] However, much is still to be learned about the complex microbiology involved and the nature of the biofilm commonly known as dental plaque.

What is known about the process of dental caries in the mouth?

It is well established that demineralization and remineralization occur in the mouth, that the process can lead to cavitation together with all of the possible clinical complications that may follow. Laboratory models have elucidated several aspects of what goes on in the mouth.[7,22–24] Many *in situ* studies have been published that confirm aspects of the microbiology, biochemistry and physical chemistry involved.[7,22–27] However, in the dental profession we still talk of diagnosing dental caries as being the detection of demineralized regions and especially cavities. The practising dentist "fixes caries" by drilling and filling rather than intervening therapeutically before cavitation occurs, and while the process of mineral loss is still reversible, or at least can be arrested.

The earliest clinical sign that dental caries is in progress in the mouth is the so-called "white spot lesion", an example of which is shown in Fig 1. This is the first sign that can be seen by the human eye, and yet by this time the process has been going on for months.

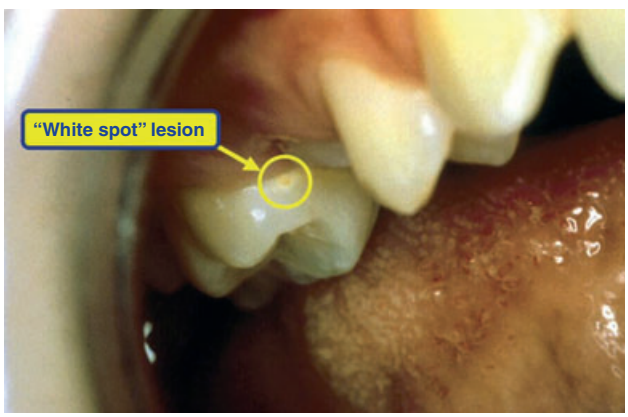

Fig 1. A "white spot lesion" in the proximal surface of a tooth.

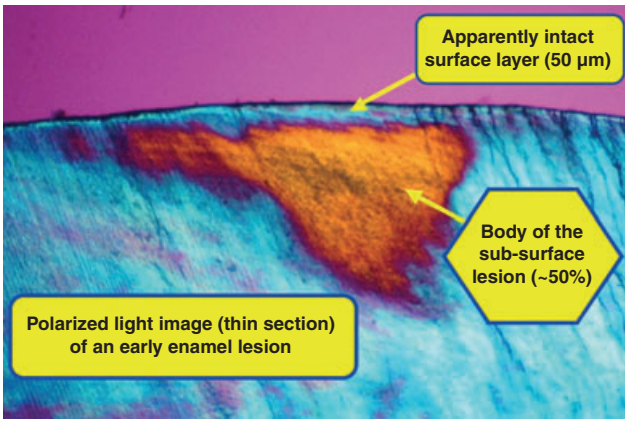

Fig 2. A cross-section of an early non-cavitated natural early carious lesion in enamel viewed under polarized light showing the body of the lesion and the apparently intact surface layer overlying it.

Cross-sections through these early lesions can be visualized under an optical microscope as illustrated in Fig 2. As these lesions progress into enamel they can then be detected clinically by radiographs.[28] At this stage in the process, prior to cavitation, therapeutic intervention can arrest or reverse the process by remineralization.[2,29] Demineralization when it first starts can only be seen at the electron microscopic level on extracted teeth in the laboratory.[30–33] As demineralization progresses the mineral loss becomes deeper into the enamel or exposed dentine until it can be detected radiographically, visually, or by the more recent optical methods, such as laser-induced fluorescence.[34–36] The major point is that if a carious lesion is non-cavitated, and especially if it is in the enamel, it can be reversed or arrested chemically as described below.

Demineralization at the crystal level

Sound enamel and dentine crystals are very small, on the order of 40 nm and 10 nm in diameter, respectively. They are comprised of a hydroxyapatite-like mineral that contains many impurities and inclusions of other ions that cause the mineral of enamel and dentine to be much more soluble than pure hydroxyapatite or fluorapatite. The major inclusion that makes dental mineral much more acid soluble than hydroxyapatite or fluorapatite is the carbonate ion that substitutes for the phosphate ion in the crystal lattice, producing defects and calcium deficient regions. Approximately 1 out of 10 of the phosphate ions in enamel are replaced by carbonate and 1 out of 5 in dentine.[37–40]

Demineralization occurs in two steps. First, the bacteria metabolize fermentable carbohydrates producing organic acids that diffuse into the tooth through the water amongst the crystals. When the acid reaches a susceptible site on a crystal surface calcium and phosphate are dissolved into the surrounding aqueous

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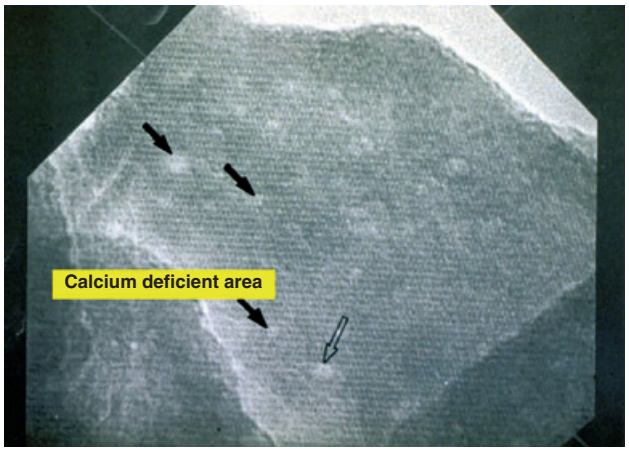

Fig 3. High resolution electron micrograph at 3 000 000× of a sound enamel crystal showing rows of calcium atoms (black dots) visualized by this technique. White patches (arrows) are calcium deficient/carbonate rich regions in the crystal.

phase between the crystals. Figure 3 is a high resolution electron micrograph of a single crystal from a region of sound enamel. This technique only visualizes the electron dense calcium ions that show up as straight rows of dots that each are derived from calcium ions.[31,32] Phosphate, hydroxyl and fluoride ions cannot be seen with this technique. The white patches are regions of calcium deficiency that are most susceptible to acid attack because of the substitution of phosphate ions by carbonate ions during tooth and bone development. This is illustrated in Fig 4 that shows two crystals from the body of a natural carious lesion. The small calcium deficient, defect areas, visualized in Fig 3, have expanded into hexagonal shaped regions where mineral has dissolved during acid attack. This is the first stage of demineralization which is occurring at the atomic level far before it can be seen visually as gross demineralization. This is the first step in the continuum of the dental caries process, that can eventually lead to cavitation.

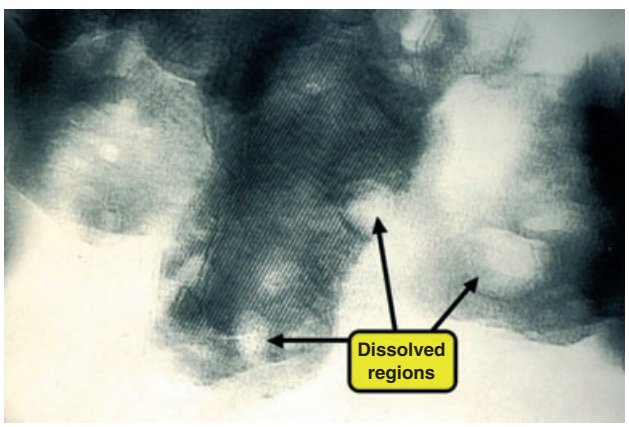

Fig 4. High resolution electron micrograph at 3 000 000× of crystals from a carious lesion showing rows of calcium atoms (black dots/lines). The hexagonal white patches (arrows) are where acid has dissolved mineral from calcium deficient/carbonate rich regions of the crystal.

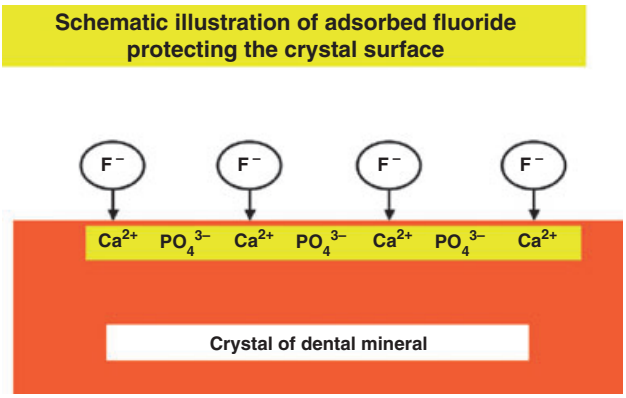

Fig 5. Schematic illustration of adsorbed fluoride ions on the surface of dental mineral crystals.

If fluoride ions are present at the crystal surface in sufficient concentration before or during demineralization these ions can adsorb onto the surface of the crystals and markedly inhibit demineralization by acid.[41] This is shown schematically in Fig 5. The strongly adsorbed, highly electronegative fluoride ion can protect, at least partially, against demineralization by acid, as illustrated in dissolution studies in the laboratory. This phenomenon is one of the mechanisms of action of fluoride when it is available topically on the teeth and in the plaque.[7]

Remineralization/tooth repair

Remineralization is the body's natural repair process for subsurface non-cavitated carious lesions.[7] Remineralization is simple in concept (Fig 6). Calcium and phosphate, primarily from saliva, but possibly from other topical sources, diffuses into the tooth and, with the help of fluoride, builds on existing crystal remnants (Fig 7), rather than the formation of new crystals.[2] The new crystal surface however, is composed of a veneer of well-formed mineral most likely similar to fluorapatite, depending on the amount of fluoride present (Fig 8).

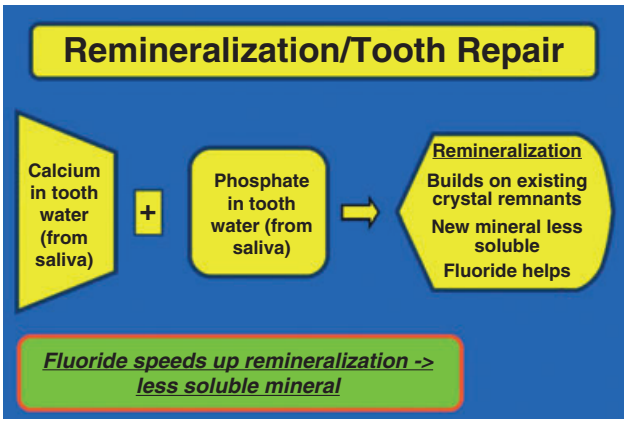

Fig 6. Outline of the remineralization process.

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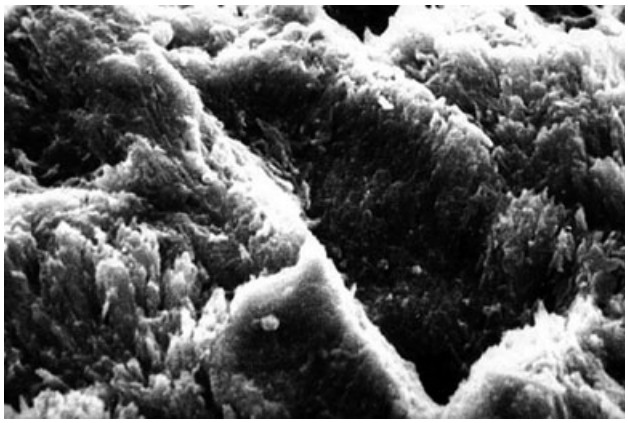

Fig 7. Scanning electron micrograph at approximately 30 000× in the body of a subsurface carious lesion showing the remaining crystal remnants that can be remineralized.

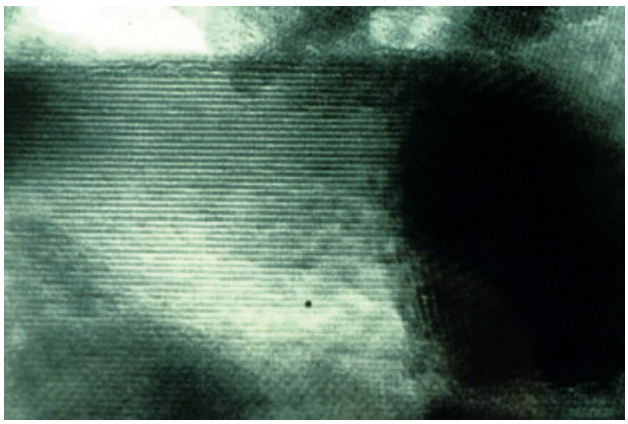

Fig 8. High resolution electron micrograph at 3 000 000× after remineralization in the laboratory with calcium, phosphate and fluoride, showing a well-formed, low solubility, fluorapatite-like veneer overlying the original defective crystal.

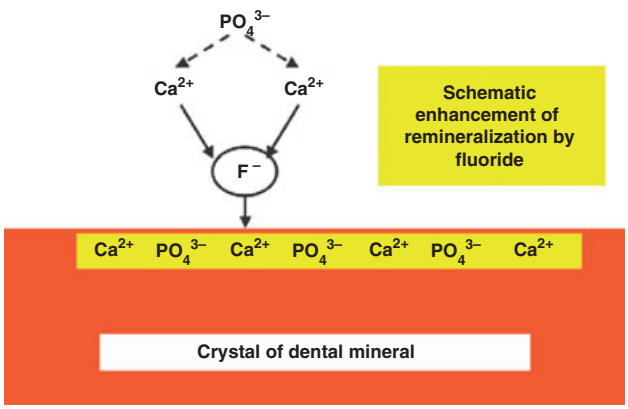

Fig 9. Schematic representation of fluoride ions adsorbed to the apatitic crystal surface of dental mineral attracting calcium ions, which then attract phosphate ions, starting to build a fluorapatite-like remineralized veneer on the crystal surface.

The manner in which this new veneer is thought to form at the atomic level is illustrated in Fig 9. Fluoride ions adsorb to the apatitic crystal surface of dental mineral attracting calcium ions, which then attract phosphate ions, starting to build a fluorapatite-like remineralized veneer on the crystal surface. The crystal surface is now much less soluble than the original carbonated hydroxyapatite mineral and is more difficult for the acid to dissolve next time there is an acid challenge from the plaque.

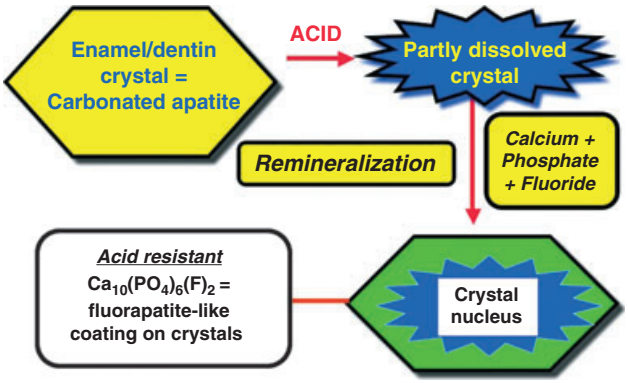

Fig 10. Summary of the caries process including demineralization by acids produced by the cariogenic bacteria and subsequent remineralization to form a low solubility surface on the crystals.

The overall process of demineralization and remineralization is shown in Fig 10. Calcium and phosphate in saliva are held in a supersaturated state by small salivary proteins such as statherin that complex the calcium and hold it in solution in a readily available reversible fashion.[21] In the case of impaired salivary flow and/or function (hyposalivation) for reasons such as medications or radiation of the salivary glands, insufficient calcium and phosphate is available for remineralization and rampant caries is the result.[3,42] Supplementation of the salivary components by-products that contain calcium and/or phosphate is obviously beneficial in these cases.

Fluoride speeds up the remineralization process, is itself consumed during remineralization and forms an integral part of the new veneer on the crystal surface.[7] This is one of the major mechanisms of action of fluoride in the inhibition and reversal of the caries process.

The caries continuum, the caries balance and clinical relevance

Dental caries is a disease that is manifested as a dynamic process in the mouth. Cycles of demineralization and remineralization continue in the mouth as long as there are cariogenic bacteria, fermentable carbohydrates and saliva present. Whether demineralization or remineralization is proceeding at any one time is determined by the balance between pathological factors and protective factors (Fig 11).[1,2,43] The key factors are illustrated as part of the so-called "caries balance" in

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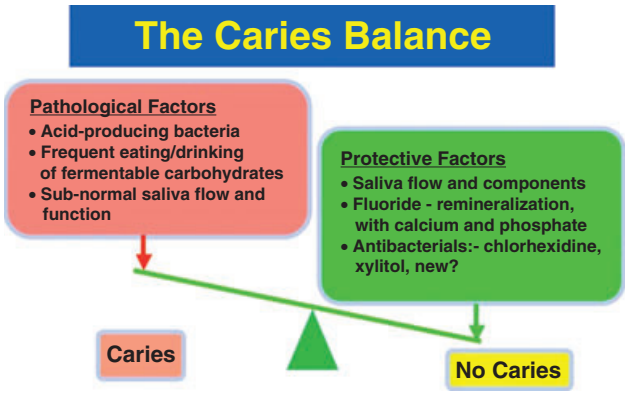

Fig 11. The caries balance concept. The key pathological and protective factors determine which side the balance swings and whether the caries process progresses, reverses, or is in balance.

Fig 11. The concept of a balance is supported by clinical observations that the caries process can be arrested for a long time and then progress if one of the components is changed. An obvious extreme example is when a person becomes xerostomic as a result of radiation to the head and neck as cancer therapy. The salivary gland function can then be severely impaired leading to rampant caries in months if aggressive preventive measures are not taken. For some people, brushing twice a day with a fluoride-containing toothpaste can tip the balance and eliminate future carious lesions. When the bacterial challenge is high it is difficult for fluoride therapy to overcome the challenge and caries can progress. This can be the case in high caries risk individuals when antibacterial therapy as well as fluoride therapy is needed.

One of the keys to understanding how the caries process can be arrested or even reversed is the realization that caries is a continuum from the very first stages of loss of calcium and phosphate from the crystal surface through to cavitation. Clinical dentistry, even today, generally considers that placing a restoration "fixes" dental caries. Unfortunately, placing the restoration only removes the offending bacteria from that cavity in that tooth. It does not deal with the disease in the rest of the mouth.

Remineralization therapy, as described above, produces mineral crystals that are much more resistant to acid challenge than the original mineral of the enamel and dentine. Therefore remineralization alone is often sufficient to stop the progression of caries and to repair the subsurface lesions so that operative dentistry is not needed. Numerous *in situ* and *in vivo* studies have confirmed this.[22–25,44–47] The many clinical trials with fluoride toothpastes have shown dramatic reductions in overall decay levels as measured by cavity formation. One can conclude from those clinical trials that remineralization was most likely a major contributor to the success of the products. *In vivo* and *in situ* studies that have directly measured remineralization or inhibition of demineralization have given further proof of the concepts.[24] Examples of this are provided by studies that investigated demineralization or remineralization around orthodontic brackets in vital teeth.[45,48] These studies showed that the caries process can be altered or reversed by the use of fluoride products that inhibit demineralization and enhance remineralization.

CONCLUSIONS

Dental caries is a bacterial disease process caused by acids from bacterial metabolism diffusing into enamel and dentine and dissolving the mineral. The bacteria responsible produce organic acids as a by-product of their metabolism of fermentable carbohydrates. The caries process is a continuum resulting from many cycles of demineralization and remineralization. Demineralization begins at the atomic level at the crystal surface inside the enamel or dentine and can continue unless halted with the end-point being cavitation. There are many possibilities to intervene in this continuing process to arrest or reverse the progress of the lesion. Remineralization is the natural repair process for non-cavitated lesions, and relies on calcium and phosphate ions assisted by fluoride to rebuild a new surface on existing crystal remnants in subsurface lesions remaining after demineralization. These remineralized crystals are acid resistant, being much less soluble than the original mineral.

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