

## CARIES Factoid

Microbiological studies by Loesche & others have been able to provide a great deal of information on the different types of **bacteria** that are responsible for the caries process. Caries is an invasion of normal indigenous **oral bacteria**, which simply take advantage of a patient's poor oral hygiene & an increased oral intake of sugars & carbohydrates. These **Strep & Lactobacillus (LBA) bacteria** easily enter into enamel lamella, cracks & any miniscule defect & after they eat the abundant sugars. In their isolated & hidden niches, these **bacteria** empty their enzymes & organic acids—mostly lactic—into the surroundings, which easily demineralizes the calcium hydroxyapatite (CHA) nano-crystals of enamel & eventually those of dentine.

If the enamel lesion is only a “white-spot” lesion, it means the demineralization is ONLY sub-surface without any cavitation (break) of the outer enamel surface. If the clinician leaves this white spot lesion alone (**AS THEY SHOULD**) & they encourage the patient to increase their brushing & flossing hygiene habits & the clinician to then treat the area with fluoride, the **bacteria** will rapidly die out & the demineralized area will quickly remineralize—generally harder (sclerotic) than the original substrate.

But when these **bacteria** proceed deeper through the enamel to the EDJ interface, these **bacteria** easily spread peripherally along the EDJ interface & the dentine becomes subjected to the same **Strep & LBA** along with their lactic acids. The organic acids immediately attack the most-dense CHA of the dentine—peritubular dentine first—then the CHA of the intertubular dentine & leaves the organic proteins (collagen, GAG's, proteoglycans, etc.) unaltered.

Histological studies of the carious lesion in dentine have been carried out since the mid 1800's (Clapp-Williams). For the sake of histo-differentiation, the lesions in dentine have been differentiated into 4-zones of degeneration by Bernick—more specifically than the 2-layers as described of Fusayama & Terashima.

Anyone who reads this CARIES FACTOID should learn that there is **NOT** just one EXACT line of discrimination between each of the four zones. The change from one zone to another zone should be thought of--in a manner of degrees. On the other hand, there **IS** a great deal of biological discrimination of the outer two-layers. The outer zone, known as the **infected carious zone** is completely dead. The next deeper zone contains

vital collagen & I personally prefer to call it the affected demineralized zone instead of using the word caries—as that term of caries tends to make most clinicians & readers think it is dead & so the clinician then thinks that they should simply remove the entire lesion with a diamond bur with ultra-high speed to remove the demineralized zone—however it SHOULD be left in place to become hypermineralized-sclerosed.

1) The outermost zone towards the oral cavity is called the **superficial infected carious dentine zone**. Interestingly enough, this zone is the most pathologically infected zone with many-millions of **bacteria**, being a non-vital dead zone characterized by complete CHA demineralization & decomposition of all organic cell components within the tubule e.g. odontoblast cell processes, collagen in the tubule space as well as the proteins of the intertubular dentine—due to the **proteolytic bacteria** secreting collagenase, which depolymerizes the carbohydrate proteins that in turn causes swelling & complete dissolution of the ground substance (see figures 1-3). By TEM, this outer zone is seen as a soft crumbly mass with NO resemblance to normal dentine architecture, composed of both **cocci & rod shaped microorganisms**. The **bacteria** are polymorphic (many shaped) & appear to be secreting their organic acids in lacuna of the peritubular wall. Some of the **proteolytic bacteria** (*Bacteriodes m*) are digesting (eating) the organic components of the odontoblast processes.

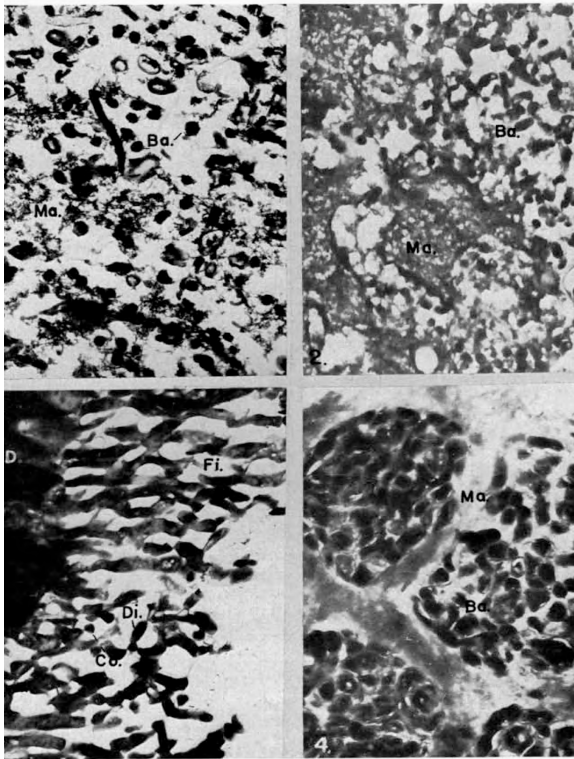
2) Below the outermost infected carious zone is the **affected demineralized zone** that is characterized by incipient—just beginning—demineralization in which the peritubular dentine wall is completely destroyed. There is often a complete disruption of the tubule morphology. The **bacteria** that reside in this 2<sup>nd</sup>ary zone are those same pioneer **Strep & LBA bacteria** that continue their invasion into this deeper zone from the initial lesion. The important point in differentiation of this 2<sup>nd</sup> **affected zone** is—the vital proteins of collagen remain unaltered leaving the remaining collagen fibers of 640-Å striations along with normal intertubular matrix of ground substance & CHA (Bernick). This remaining substrate serves as a structural-framework for the remineralization of the peritubular CHA into a future zone of dense sclerotic dentine. As mentioned before in several of my CC articles that I have written for the MI-EIKO Journal, caries is a cyclic disease with a rapid progression, which may rapidly stop---with improved oral hygiene to deprive the **bacteria** from sugar & carbohydrates—in part due to an improved use of

fluoride, the demineralization process may rapidly turn into a phase of remineralization showing the presence of **WHITLOCKITE** caries crystals of remineralization.

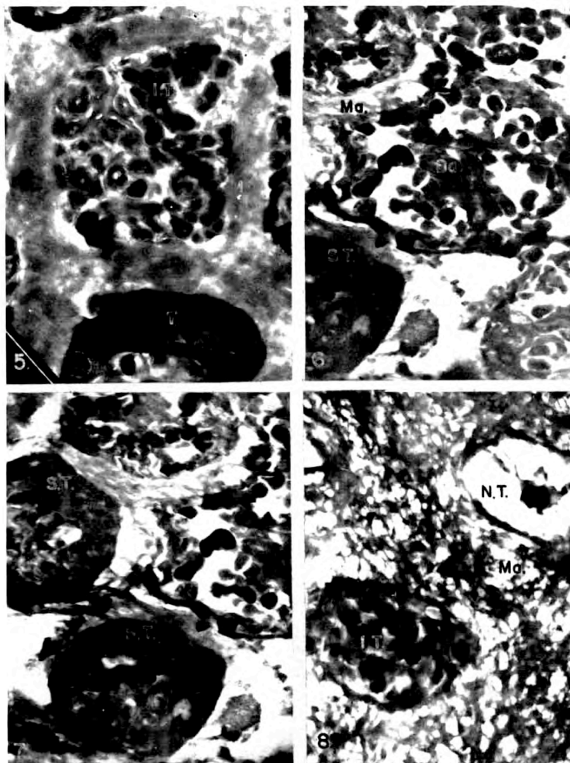
3) The next (3<sup>rd</sup>) deeper zone has been called the **transparent zone** of dentine. This may be thought of as a thin or self-healing zone of remineralization the tooth may effect on its own. The organic acids from the 2<sup>nd</sup>ary-affected zone seem to seep-down along some of the tubule walls, which begin to demineralize the outer CHA-dense-peritubular walls. With fluoride & pH intervention of a calcium-phosphate rich paste, they may undergo the process of remineralization of the peritubular CHA into new nano-crystals. Under transmitted light with ground sections, this 3<sup>rd</sup> zone shows the tubules filled with small transparent (refractory) nano-crystals. To allow the tubules to fill in with crystals, after the cell processes of the odontoblast processes had collapsed—some colleagues think as due to having become retracted from the EDJ & other researchers suggest they have become deflated of their contents to leave a remaining space between the vital process & tubule wall becomes filled with the remineralizing nano-crystals. This transparent dentine zone is easily seen in tooth sections that have been prepared by ground sectioning, in which the mineral tooth substrate of the tooth was not demineralized in an acid for thin sectioning in paraffin or plastic.

4) This is the deepest & innermost zone & is seen as a very thin zone that is situated quite far ahead of any actual demineralization of the peritubular dentine complex. For the most part, there are only a few of the dentinal tubules that may be invaded by any sort of **microorganisms**. G.V. Black reported in his histological sections in 1864 the observation of a **few bacteria** in the tubules well below the outermost caries, but could not understand how they could reach so deep. With today's understanding, we now realize that the **Strep & LBA bacteria** simply demineralize their way down the tubules by breaking apart the peritubular dentine & then reproduce to fill-in the enlarged space. In their passage from the outermost zones down the tubule, the **bacteria** also produce various immunological factors that initiate a fatty degeneration of some of the odontoblast processes—however most of the dentinal tubules & their contents generally appear normal—as the invasion front was **NOT VERY SEVERE**. The deepest zone (figures 7-8) seems to show the initial process of cell wall breakdown while showing some degree of remineralization (sclerosis) of the CHA of the peritubular dentine.

The following TEM's are from published studies of Dr. Sol Bernick



- 1) Zone 1 shows the complete demineralization & decomposition with **bacteria (Ba)** & no organized matrix (Ma) substrate. All tissue is dead & must be removed using **DiscovRED** before restoration.
- 2) Zone 1 with some demineralization with many **bacteria Ba** & disorganized dentine matrix Ma.
- 3) **Ba** of many profiles are seen lying on the disorganized dentine (D) surface of the affected zone.
- 4) The affected 2<sup>nd</sup> ary zone of incipient demineralization shows many **Ba** in the tubules & adjacent matrix (Ma). The peritubular dentine is gone due to lactic acid. This zone will remineralize.



- 5) Deeper part of 2<sup>nd</sup> affected zone with many **bacteria** above & sclerosed nano crystals in the lower tubule.
- 6) Deeper part of 2<sup>nd</sup> affected zone with breakdown of middle tubular wall & with sclerosed nano-crystals below left.
- 7) Deeper part of 2<sup>nd</sup> affected zone with sclerosed nano crystals in the left two tubules.
- 8) Deeper zone 4- with normal tubule (NT) above many **bacteria** in the lower left infected (IT) tubule & stable intertubular zone between the tubules.

The eight TEM pictures from the research study of Dr. Sol Bernick & colleagues demonstrates—beyond a doubt—there is NOT any one clear zone of ideal demarcation between the two deeper regions of 3 & 4 of bacterial infection into dentine. Please remember that in biological systems, nothing is ever perfectly defined in black or white!

The morphology of the carious bacteria have been known since Miller published his data showing 3-types of bacteria as cocci, bacilli & filamentous. Each type of bacteria has the capacity to grow separately & yet all bacteria are simultaneously present in the Infected carious biofilm-mass of dead tissue.

The histochemical studies of Engel in 1950 clearly demonstrated that the ground substance glycoproteins of the outermost zone of infected carious dentine becomes depolymerized (non-vital) due to the enzymatic action of the *Bacteriodes melanogenicus* & other proteolytic organisms, which produce substances that alter the organic components. Bernick & others described this effect of the proteolytic enzymes—especially collagenase—that specifically depolymerize (completely destroys) the carbohydrate protein complex, which results in the swelling & the dissolution of the ground substance.

It should be noted that in normal human dentine, the intertubular matrix is composed of a ground substance, collagen fibrils of 640-Å with patterned striations with CHA crystal apatite that is situated throughout. During incipient carious invasion with *Strep m* & *LBA bacteria* begin their demineralization--first along the peritubular CHA of the tubule wall. As the carious lesion progresses deeper into the dentinal tubule complex, a few pioneer bacteria secrete acids to begin the demineralization, however the vital proteins remain untouched.

The issue of sclerosed tubules has been studied by many & the sclerosed nano-crystals have been demonstrated by many in the affected demineralized zone, in which there was—as earlier mentioned—previous demineralization that simply stopped & reversed to form remineralized Whitlockite CHA peritubular dentine crystals that reform in the absence of the odontoblast process. The crystals block the tubules & stop fluid flow as well as to prevent the further penetration of bacteria deeper into the dentinal tubule complex.

It is well known in the hundreds of biological research publications on soft connective tissue healing, that degenerating & necrotic tissues have an affinity for the binding of calcium. Consequently, it is no wonder that many of the dentinal tubules of the 3<sup>rd</sup> & 4<sup>th</sup> deeper zones of early **bacterial** invasion, in which the odontoblast processes have become altered into a sort of homogenous mass—similar to a hyaline mass as seen in the 4<sup>th</sup> zone. Dr. Bernick noted this was no different than degeneration exhibited by connective tissue found in other parts of the body. Thus, these deepest tubules of zone 2, 3 & 4 become remineralized with caries crystals that are known as sclerotic dentine. In addition, a significant point Dr. Bernick reported was that the number of **bacteria** within the sclerosed dentinal tubules was always less than in the surrounding tubules of the more superficial carious dentine—whereas, in the deepest zone of demineralization, only a few tubules contain **microorganisms**.

So now we are left with the removal of all of the dead infected carious dentine & leaving the 2<sup>nd</sup>ary deeper affected demineralized dentine as well as the deeper transparent & deepest 4<sup>th</sup> zone to become sclerosed.

### **SO-----CAN WE LEAVE SOME BACTERIA IN THE DEEPER LESION ? YES**

Questions are always asked by some clinicians, if we only remove the infected carious dentine, then what will happen to any of the deeper **bacteria** that have found their way into the deeper parts of the affected demineralized 2<sup>nd</sup>ary zone & the deeper zones? The **ANSWER IS VERY STRAIGHTFORWARD, THEY SIMPLY DIE OUT** & the remaining vital collagen substrate provides a framework for the accumulation of calcium from any extracellular fluids & more importantly from the CHA of the peritubular dentine to re-form the new nano-crystals of the Whitlockite caries crystals. This death of carious **bacteria** has been demonstrated in longitudinal clinical studies (Mertz-Fairhurst, Kidd, Foley). Thus, studies show it is biologically permissible to leave the affected demineralized & deeper zones underneath the outermost **bacterially laden-dead zone of caries**.

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