The Development and Structure of The Tooth Enamel and The Nature of Deterioration

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Abstract

The tooth enamel is developed through a careful process to give its characteristic hardness, but bacteria colonization in the form of plaque and certain dietary habits can compromise this. The development of the tooth involves an intricate process that incorporates proteins and minerals to produce the toughest tissue in the human body. The enamel possesses a distinct crystal lattice with many minerals involved. Under acidic conditions, the integrity of the crystals that make up the tooth enamel can become compromised. Harmful bacteria, such as *Streptococcus mutans* of the mouth combine with carbohydrates that are consumed to produce acids that are harmful to the basic crystals making up the tooth enamel. The acidity causes dissolution of the enamel crystals, producing small concavities on the surface of the teeth that can be particularly difficult to clean and thus leads to further deterioration. This demonstrates the importance of maintaining proper dental hygiene to prevent deterioration.

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Stages of Tooth Development

Bud Stage, Cap Stage, and Bell Stage

In most children, the formation of primary, or deciduous teeth begins in the first few months of gestation (Anatomy and Development, n.d.). During this period, the materials making up each tooth are developed, and the hardening tissues that provide endurance are formed. Once formed, the deciduous, or baby teeth are forced out of their place, and by the age of thirteen, the child has a full set of permanent teeth, excluding the third molars which erupt later (Teeth Eruption Timeline, n.d.). Under ideal conditions, the permanent teeth remain with individuals for the rest of their life, and thus must have a highly durable structure. The tooth enamel has a distinct histological and chemical structure that deems it the toughest human tissue in the body, but with improper hygiene and dieting, the durability of the enamel can be compromised (Bocskay & Waldhofer, 2005). Therefore, it is critical that proper dental hygiene be practiced, to keep the mouth, teeth, and gums clean and healthy to ensure that they do not deteriorate rapidly or become susceptible to disease (Dental Hygiene, n.d.). A thorough analysis and summary of the development and chemical composition of the tooth enamel and the effect that plaque can have on a tooth's durability can inform individuals of how to effectively care for their teeth throughout their lifetime.

Tooth formation occurs through a series of three stages, including the bud stage, the cap stage, and the bell stage, which encompasses the development of the tooth germ from the membranes of the mouth, the formation of tooth layers, and the mineralization of cells to produce durable teeth as are seen in one's smile. The bud stage is characterized

by the formation of enamel organs, which are said to be swellings of the dental epithelium with the assistance of ectomesenchyme cells present beneath the oral epithelium in what is known as the dental papilla, forming the dental lamina (Rathee & Jain, 2021). Mesenchymal cells help to induce the swelling of the basal layer of dental lamina and develop the enamel organ structures. The swellings of the dental lamina can appear in many images as the formation of a kind of invagination deeper into the mesenchyme, but this behavior occurs because of the increased replication of dental epithelium that invades further into the mesenchyme (Havorakova et al., 2018). This stage occurs after approximately eight weeks *in utero*, and through this stage, the dental lamina begins to swell and eventually forms rounded, seed-like structures, which correspond to separate tooth formations as depicted in Figure 1. Ultimately, the swelling of the dental epithelium causes it to become greater in size compared to the interdental tissues. The cells of the inner enamel epithelium adjacent to the dental mesenchyme have a columnar shape, while the interior cells are less ordered polygonal cells. In the early formations of the jaw, twenty buds can be seen that will form into the upper (maxillary) and lower (mandibular) deciduous teeth. The timing of this process corresponds to the deciduous teeth, but the same process occurs for permanent teeth beginning in the fourth prenatal month except for the second and third molars, which begin developing after birth. The development of the permanent teeth arises from epithelial tissues lingual to the enamel organs of the deciduous teeth.

Figure 1. Bud stage of tooth development. The oral epithelium, also known as the dental epithelium, extends deeper into the dental mesenchyme. The deeper “bulb” of the dental

laminar extension is surrounded by the dental papilla, or condensed ectomesenchyme (DentoMedia, 2020).

Following the bud stage is the cap stage, which is demarked by the development of a concavity of the dental lamina. Through an uneven division of epithelial cells, the bud begins to invaginate and engulf a portion of the dental papilla, which will eventually form the tooth pulp (Bei, 2009). In the late stages of the cap stage, the inner cuboidal epithelial cells of the enamel organ that border the dental papilla form the inner enamel epithelium with columnar cell shape to define the tooth crown, while the outer layer of enamel organ cells retain their shape and will form the outer enamel (Rathee & Jain, 2021). Within the layer of columnar epithelial cells is the stratum intermedium found along the inner enamel epithelium, which is important for nutrient transport and for the formation of the ameloblasts responsible for enamel formation. The stellate reticulum forms between the stratum intermedium and the outer enamel epithelium, and this structure serves to protect underlying structures and maintains tooth shape. These structures form at about 14 weeks *in utero*. In addition to the cell shape changes that occur in this stage, lateral extensions or branches form deeper into the mesenchyme to create a shape resembling a cap as shown in Figure 2, which gives this stage its name.

Figure 2. Cap stage of tooth development. The inner enamel epithelium extends outward around the dental papilla during the cap stage. The stellate reticulum makes up the inner portion of the structure between the inner and outer enamel epithelia (DentoMedia, 2020).

The final stage of tooth formation includes the bell stage, which is characterized by the disintegration of the dental lamina and incorporates the mineralization of the enamel. During the bell stage, inner enamel epithelium is differentiated into cells called

ameloblasts, and signals from these cells to the dental papilla initiates the conversion of the dental papilla into different cells called odontoblasts shown in Figure 3 (Kawashima & Okija, 2016).

Figure 3. Bell stage of tooth development. During the bud stage, the dental lamina disintegrates, and the forming tooth is isolated within the tissues deep to the oral epithelium. The dental papilla cells along the inner enamel epithelium are converted into odontoblasts. The stratum intermedium neighbors the inner enamel epithelium, and the stellate reticulum is between the stratum intermedium and outer enamel epithelium (DentoMedia, 2020).

The structural changes that occur through the bud, cap, and bell stages of tooth development are controlled by a structure called the enamel knot. The enamel knot is known to be a cell cluster in the central portion of the dental epithelium bordering the dental mesenchyme (Vaahtokari et al., 1996). The enamel knot itself is formed in the bud stages of tooth developments, and their roles are carried out mainly through the cap and bell stages. The enamel knot structure arises from a thickening of epithelial cell to form what is called the initiation knot which is a signaling center for progression of tooth development and is the predecessor to the enamel knot. This structure is approximately at the deepest portion of the enamel epithelia and remains in the center of the epithelial extensions in the cap stage as shown in Figure 2 (Mogollón et al., 2021). It has been observed that the juxtaposition of cells with active “wingless-related integration site” (WNT) signaling pathways to Sonic hedgehog (SHH)-expressing cells leads to the maturation of the initiation knot into the enamel knot. WNT signaling has been found to play critical roles in tooth development regulation and adult tooth maturation, and the

expression of the SHH gene is known to play critical roles in cell growth and specialization (Tamura & Nemoto, 2016; U.S. National Library of Medicine, 2020). The enamel knot is different in the cap and bell stages in its structure, and therefore the different forms of the knots in these stages are termed the primary and secondary enamel knots respectively. The enamel knot serves in the regulation of tooth morphogenesis by primarily determining the shape of the developing tooth with which it is associated through the cap and bell stages.

Enamel Mineralization and Structure

Mineralization in the bell stage includes the process of dentinogenesis involving the odontoblasts previously formed from the dental papilla along with the differentiation of the inner enamel epithelium into ameloblasts. The odontoblasts and ameloblasts will be mineralized and form the dentin and enamel structures, respectively. In the past, it was unclear how the mineralization process occurred, either through mineralization of matrix vesicles in odontoblasts or through the secretion of a gelatinous matrix that is later solidified through the infusion of minerals (Goldberg el al., 1995). Dentin structures are characterized by a network of tubules from the dental pulp to the enamel to facilitate the transport of needed minerals from the bloodstream, and recent studies have shown that these structures are responsible for the deposition of unmineralized matrix for later mineralization into the enamel structure. These tubule structures arise from the secretion of a collagenic matrix that is arranged to form what is called the pre-dentin, which is the precursor to dentin. Analysis of dentin structures has shown that this layer contains high collagen type I as well as lower amounts of collagen type III, type V, and several non-collagenous proteins in the dentin extracellular matrix (Kawashima & Okiji, 2016). The

odontoblasts are responsible for making the dentin link together with junctions that allow the passage of ions and proteins from the pulp vasculature necessary for the mineralization of the dentin, but the majority of the materials needed to produce the dentin is provided by the odontoblasts themselves. The collagenous structure observed in dentin is like that observed in bone, but the non-collagenous proteins secreted are specific to dentin formation. Immediately following the formation of the dentin, the enamel is allowed to form.

The process of enamel formation, or amelogenesis, begins via ameloblasts, formed with the help of mesenchymal cells near the dental pulp (Papagerakis & Mitsiadis, 2013). The process of amelogenesis is divided into four stages: the presecretory, secretory, transitional, and maturation stages. Inner enamel epithelial cells differentiate into ameloblasts, which are highly important to the mineralization, or crystallization of the enamel. The presecretory stage encompasses odontogenesis and the laying of the dentin extracellular matrices, and this stage finalizes with the secretion of enamel matrix proteins from ameloblasts. These proteins play a large role in mineral formation as they rapidly initiate the mineralization process (Bartlett, 2013). During the secretory stage, the ameloblasts elongate into columnar cells possessing a structure called the Tomes' process, which looks like a pointed extension or chamfer of the cell on the side facing the secreted enamel matrix. This structure forms while the enamel matrix is being secreted as the ameloblasts move away from the enamel matrix and drags the Tomes' process "tail" structure surrounded by matrix. Additionally, the secretions of the ameloblasts come from the same face of the Tomes' process, and the ameloblasts have the same paralleled orientation, giving order to this layer as the matrix is excreted. During

the transition stage of amelogenesis, the last of the enamel matrix is excreted from the ameloblasts to achieve the desired thickness. At the same time, the ameloblasts begin to retract their Tomes' processes and compress into a shape more like cuboidal cells to prepare for amelogenesis maturation. The changes in ameloblast structure from elongated columnar cells to shorter and fatter cell also result in a change in the cell's functions, such that they operate less as secretory cells and more as a barrier against the enamel matrix to retain its shape.

Precursors to the enamel crystals, or hydroxyapatite crystallites, begin to form from the crystalized collagen fibers within the dentin. The dentin possesses a high concentration of collagen fibers, and these fibers provide a foundation for inorganic minerals or ions to undergo heterogenous nucleation and form crystallites extending towards the dentin-enamel junction (Lacruz et al., 2017). Nucleation occurs as ions in solution form solid hydroxyapatite crystallites in the presence of a solid nucleating agent, which in this case is the solid collagen present in the dentin. The organization of the enamel crystallites assumed during the mineral nucleation is primarily influenced by the matrix proteins present in the enamel matrix deposited by ameloblasts. Though the nucleation process is still a point of active study, it has been observed that the protein amelogenin secreted from ameloblasts is a primary promoter of the precipitation of calcium phosphate in crystal formation (Tarasevich et al., 2007). These proteins are moved into the enamel matrix and ameloblast membranes to assume the appropriate enamel thickness despite its gelatinous composition as it is infused with the enamel matrix proteins and water (Lacruz et al., 2017). This occurs during the maturation stage, and along with the insertion of enamel crystallites, the condensed ameloblast layer

secretes the protein kallikrein-related peptidase 4 (KLK4) to aid in the partial removal of the gelatinous matrix that was previously excreted (Bartlett, 2013). The organic enamel matrix is degraded as many of the enamel matrix proteins are removed from the matrix followed by the uptake of various minerals, increasing the enamel crystal growth, and thus, exchanging the previously soft, gelatinous structure with the characteristic hardness and durability of mineralized enamel (Robinson, 2014). This removal of enamel matrix creates space for the crystallites that are being inserted to be able to expand and assume the appropriate lattice structure. Minerals involved in this process include calcium, phosphate, magnesium, fluoride, carbonate, and others, and the uptake of these minerals continues in humans until it reaches the appropriate ion concentration, which is approximately 95% by volume with the outer surface enamel more mineralized than the inner enamel (Robinson et al., 2003). Several of the ions involved in this process do not directly contribute to the makeup of the hydroxyapatite crystallites but are present in large concentrations at the center of the crystals to assist in providing support to the crystal. The additional incorporation of the minerals making up the tooth enamel with the previously synthesized precursor crystals allows for crystal enlargement to form an intricate network of minerals that gives the enamel its characteristic strength.

Structure of Enamel Crystals

Following the maturation stage of tooth development, a complex network of inorganic substances can be observed in the tooth enamel making up what is called hydroxyapatite, thus awarding the enamel its title as the hardest tissue in the human body. The precipitation of hydroxyapatite into a lattice shown in Figure 4 can be visualized by the equilibrium reaction as follows:

$10\ Ca^{2+} + 6HPO_4^{2-} + 2H_2O \rightleftharpoons Ca_{10}(PO_4)_6(OH)_2 + 8H^+$ (Simmer & Fincham, 1995).

Hydroxyapatite can also be found in human bone but is different in the enamel because it does not contain collagen, and the crystals are somewhat larger because they contain additional minerals, such as magnesium intertwined in their structure. In the human enamel, the role of collagen is replaced by proteins called amelogenins and enamelins that provide a framework for mineralization (Habibah et al., 2021). These proteins are also present in the laying of the enamel matrix, but as the matrix is replaced with crystallites and the enamel matures, these proteins are resorbed out of the matrix. Their role is highlighted by the observation that organisms not expressing these genes also lack a tooth enamel. Though small amounts of these proteins are present in the mature enamel, their function is essential to the organization and support of the tooth enamel in the absence of collagen.

Figure 4. Lattice structure of calcium apatite crystals. The structure of apatite crystal with the solidification of oxygen (red), calcium (light blue), fluorine (green), and phosphate (blue) possesses an internal hexagonal structure within each unit (Simmer & Fincham, 1995).

The crystal structure of hydroxyapatite is relatively large in relation to other crystal forms, and they are composed of inorganic materials. Thus, through examination of the lattice structure, it is known that hydroxyapatite that occurs in nature as a mineral has a hexagonal lattice structure with paralleled c-axes (Elliot, 1997; Pajor et al., 2019). The hexagonal structures form elongated and compact prism arrangements that number

from five million to 12 million prisms in the enamel of a single tooth crown as shown in Figure 5a (Pajor et al., 2019). In addition to the calcium and phosphate ions forming the lattice structure of the tooth enamel, various other minerals, such as magnesium (Mg^{2+}), fluoride (F^{-}), sodium (Na^{+}), chloride (Cl^{-}), and others have been found to make up the composition of the tooth enamel, but the strength of the enamel is the result of the intricate lattice of the hydroxyapatite crystals. The ions that are not directly correlated with hydroxyapatite crystals take the form of magnesium phosphate and calcium fluoride to contribute to additional support to the structure (Klimuszko et al., 2018). Additional hardness and stability of the tissues can also be attributed to the substitution of hydroxyl groups (OH^{-}) with fluoride ions in a portion of the crystals making up the enamel. Exchanging the hydroxyl groups for fluoride ion gives this crystal the name fluorapatite crystals, and these crystals are slightly smaller than hydroxyapatite crystals. The prisms crystals have a rod shape and sit parallel to one another. These crystals are composed of thousands of the smaller hydroxyapatite and fluorapatite crystallites in addition to other ions intertwined in the lattice structure. Imaging at the atomic level reveals the structure and arrangement of minerals and crystals down the longitudinal axis of a crystal. Images produced by Northwestern University in Figure 5b show the individual crystallites arranged in a tightly packed formation. The dark spots observed at the center of the crystals are believed to be deposits of minerals such as sodium, magnesium, and fluoride. This agrees with earlier findings by Pilar, Pajchel, and Kolmas (2019) that at the center of each crystal are channels occupied by these ions.

Figure 5. Scanning electron microscopy of crystallites of the tooth enamel. (a) Scanning electron microscopy produces imaging of the surface structure of the apatite crystal prisms

made from the precipitation of hydroxyapatite and fluorapatite crystals. (b) Atomic-level 3D imaging along the long axis of a hydroxyapatite crystal produced by Northwestern University (DeRocher et al., 2020).

Risk for Deterioration

Hydroxyapatite crystals are weak bases, making the tooth enamel particularly susceptible to deterioration and decay by acidic substances, but saliva helps maintain the enamel strength by providing calcium and phosphate ions (Saliva, 2012). Saliva is produced by three salivary glands in the mouth, and the produced saliva is rich in organic and inorganic matter, such as bicarbonate. Bicarbonate serves as a pH buffer in the mouth to lessen the effects of changes to the pH level in the mouth and to prevent the enamel's exposure to increased acidity in the mouth (Buzalaf et al., 2012). The saliva buffer serves to keep the pH of the saliva between 6.2 and 7.6. In addition to bicarbonate, saliva is rich in calcium and phosphate ions, which are often found mineralized as calcium phosphate. The constant supply of these ions to the enamel provides additional support as these particular ions are major components of the hydroxyapatite crystals making up the enamel that join the structure by the same heterogenous nucleation as from the crystal formation (Tarasevich et al., 2007). Though the nucleation process is still a point of active study, it was observed by Tarasevich et al. that the protein amelogenin from ameloblasts is a primary promoter of the precipitation of calcium phosphate in crystal formation. It could be said that the supply of these ions to the enamel provides an extra layer of protection to the tooth enamel against decay in its various methods. In addition, the simple act of swallowing provides clearance of acidic substance taken in through the diet, saving the teeth from excessive exposure. However, despite these precautionary

measures to protect against decay, without proper care of tooth structures, salivary pH buffering and structural support is not enough to prevent the deterioration of tooth structures.

Oral Microbiota

Deterioration of the tooth enamel is the result of acidic materials, and the acidic condition of the mouth can arise from conditions causing vomiting or acid reflux, but these can be conditional to certain physiological conditions. However, a major contributor to enamel deterioration, as well as many other diseases related to the mouth are harmful bacteria. The mouth is home to a highly diverse community of bacteria, many of which serve purposes that are beneficial to the functionality of the mouth, but there are many that act as competitors to the beneficial species or produce byproducts that are harmful to the various structures of the mouth. The oral microbiota contains many thousands of different species of bacteria (Arweiler & Netuschil, 2016). Unlike the risk factors for deterioration that can be cleared or counteracted, the activity of the oral microbiota is a more permanent factor to oral health. Significant compositional changes to the oral microbiota can pose a risk to various oral diseases.

Many of the bacteria within the mouth contribute to saliva composition and other factors important to the entire oral cavity, not just the enamel. For example, various studies have observed the relationships between the mineral composition of saliva and the health of gingival tissue (Ferrer et al., 2020; Yamashita & Takeshita, 2017). Different species reside in different crevices or pockets in the mouth where they carry out their functions. The spread of the microbiota is associated with the different adhesins that each one possesses, and naturally a given bacteria will colonize with the tissues that are most

capable of providing stability for colonization. For instance, a study conducted by Aas et al. (2005) sought to identify the dominant microbiota in different sites around the mouth, including the dorsum and sides of the tongue, the hard and soft palates, the buccal fold (the gap between the lips and the alveolar bone), supragingival and subgingival plaques on teeth surfaces, tonsil tissues, and labial gingiva for the purpose of mapping the colonization of some of the various microbiota in the mouth. In addition, this study used the mapping of the normal microbiota in individuals with good oral health to individuals having oral disease. To represent the diversity of the oral microbiome, it was found in this study that a total of 141 different bacterial taxa from six phyla were observed (Aas, et al., 2005). However, it was also observed that in individuals with oral disease, several species of bacteria were observed in higher levels compared to those of healthy individuals, which include *Streptococcus mutans, Lactobacillus* spp, and others.; *S. mutans* and *Lactobacilli* have been associated with dental caries (cavities) and deep dentin cavities with *S. mutans* being the primary cause of cariogenesis (Aas et al., 2005; Grigalauskienė et al., 2015). Understanding the activity of each of these types of bacteria and each of their individual and combined contributions to tooth decay can potentially provide insight to effective preventative measures to maintain the integrity of tooth structures.

Dental Plaque

Dental plaque is a type of adhesive biofilm of a large number and variety of bacteria. The biofilm of dental plaque has been estimated to have an 80-90% water composition, and about 70% of the remaining mass is occupied by the bacteria present and 30% is composed of polysaccharides, proteins, and glycoproteins (Marsh &

Bradshaw, 1995). Plaque forms on the surfaces of the teeth, and very commonly starts forming along the gingival border of the teeth and expands closer to the occlusal surface if left unattended. As shown in Figure 6, the enamel is the external surface of the tooth that comes into contact with the oral environment, and plaque buildup in these areas may be more easily maintained. Plaque buildup starts on the gingival surface towards the base of the enamel and growth further up the crown as depicted in Figure 6. Additionally, plaque can build up under the gums (gingiva) below the bottom of the enamel and more out of reach of self-treatment techniques. The buildup of plaque under the gums poses significant risk of damaging the tooth root structures and the alveolar bone providing the socket that gives the teeth stability.

Figure 6. Anatomy of the mammalian tooth. The image depicts the internal and external structures of the teeth including the enamel, dentin, and pulp, in addition to the surrounding gingival tissue (Tooth Diagram, n.d.).

Previously, it was discussed that the saliva provides calcium and phosphate ions to the tooth enamel as an additional protector against harsh environments. However, this activity is not continuous, as the saliva gradually deposits a protein film called the dental pellicle on the surface of the enamel, which blocks the supply of ions in the saliva to the enamel. The deposition of the dental pellicle (often called the acquired pellicle) allows bacteria to interact with the pellicle and begin to form a biofilm (Forssten, et al., 2010). The bacteria that adhere to the dental pellicle are termed primary colonizers, and other species such as *Streptococcus mutans* adhere to these primary colonizers, and the colonization of additional species leads to the formation of the plaque biofilm.

Over time, as plaque is allowed to remain on the surfaces of the teeth and within their crevices, a mineralization of the plaque also begins to occur from the minerals calcium and phosphate found in the saliva. The minerals in the saliva begin to invade and solidify the dental plaque that was a viable biofilm for microorganisms (White, 1997). Once hardened, the plaque is then referred to as calculus or tartar and is much more difficult to remove as it is no longer the same soft biofilm as its predecessor, dental plaque.

Streptococcus mutans

Streptococcus mutans as previously described adheres to the dental pellicle and other primary bacteria to form the dental plaque biofilms, but this species can also be found in the pharynx and the intestines. These bacteria function in the conversion of carbohydrates taken in through the diet into acid species. The production of acids in the film directly adjacent to the tooth enamel poses a serious threat to the degradation of the tooth enamel. *Streptococcus mutans* is able to survive in the mouth largely because of the carbohydrate intake through the diet. However, when deprived of carbohydrates, *S. mutans* cannot survive and thus depends on carbohydrate-supplemented saliva for its fuel (Moye et al., 2014). This species relies solely on carbohydrate metabolism for energy through glycolysis. It does so by taking in carbohydrates that are available in the extracellular environment, in this case its biofilm, and converting them to glucose-6-phosphate (G6P). G6P is then isomerized to fructose-6-phosphate (F6P) in the first step of glycolysis, and at this point F6P can continue through the subsequent steps of glycolysis, or it can be converted to glucosamine-6-phosphate (GlcN-6-P) to synthesize more cell wall. However, as the microbe continues through glycolysis, it produces lactic

acid (pKa 3.8) as a byproduct, which is then excreted back into its extracellular environment (Lemos, et al., 2019). Another characteristic of *Streptococcus mutans* is that it is an aciduric species, meaning it can survive at low pH levels created by its production of lactic acid and the intake of other acidic materials. Considering that *Streptococcus mutans* colonizes on the surface of the tooth enamel in the form of plaque, the release of lactic acid through carbohydrate metabolism causes the hydroxyapatite crystals of the enamel to be exposed to acids directly on its surface.

Lactobacillus spp.

Lactobacillus spp. is another bacteria that has been found to be absent in individuals lacking dental caries compared to individuals prone to dental caries. Studies on the presence of *Lactobacilli* of the oral cavity and fecal cells have led researchers to believe that the mouth is a continuous source of *Lactobacillus* to the gastrointestinal tract (Caufield, et al., 2015). Despite the correlation between *Lactobacilli* and individuals with dental caries, Caufield et al. (2015) were unable to identify any *Lactobacillus* species in the dental plaques of those individuals. It was proposed that as *Streptococcus mutans* leads to cariogenesis through its metabolic secretion of lactic acid leading to the breakdown of hydroxyapatite crystals. *Lactobacilli* are then allowed to infiltrate the cavities with *Streptococcus mutans* and bind to structures of the dentin and colonize as the cavity progresses. The cavities produced create suitable habitats for the continual colonization and reproduction of *Lactobacillus* species. This proposed mechanism provides an explanation for the presence of *Lactobacilli* in the gastrointestinal tract as swallowing clears any free microbes from the oral cavity and into the digestive system. Though *Lactobacilli* are not believed to be a direct causative agent of dental caries, their

presence due to *Streptococcus mutans* allows for their residual colonization (Loesche, 1996). This agrees with other findings related to oral bacteria before the eruption of the teeth from the gingival tissue.

Differences in the oral microbiota can be observed after the eruption of primary teeth compared to the microbiota before the eruption of the teeth. *Lactobacilli* can be observed in infants, which has been traced to the mothers vaginal *Lactobacilli*, but they are not retained after one month (Caufield et al., 2015). However, the eruption of the teeth provides suitable sites of colonization for *Lactobacilli*. Oral disease-related microbes have been found to be absent before the eruption of teeth, but following their eruption, it is believed that these microbes have greater ability to colonize on the tooth structures. Without the appropriate tissue structures to colonize on, certain microbes may be unable to colonize residually. These results suggest that *Streptococcus mutans* is the primary culprit for the development of dental caries, while other microorganisms, such as *Lactobacilli*, benefit from the structural changes produced by *Streptococcus mutans*.

Applying the Understanding of Dental Deterioration

It is understood that along with the dissolution of the tooth enamel and underlying structures, the enamel becomes softened when exposed to acidic substances. This occurs through the lactic acid production by dental plaque bacteria, such as *Streptococcus mutans*, but erosion can result through an acidic diet as well (Honório et al., 2010). It is important to note that there is a distinction between dental caries and dental erosion, but both are related to the weakness of hydroxyapatite crystals in the presence of acids. As a result, there are other contributors to the softening of the enamel and the loss of enamel mass. The softening of the tooth enamel creates concern as the characteristic hardness of

the tooth is reduced. As a result, the tooth becomes susceptible to further erosion and damage due to outside factors. For example, softening of tooth structures can reduce the ability of the teeth to withstand the forces of chewing hard foods, and they may be prone to chipping. Softening of the enamel also makes the teeth susceptible to additional erosion on the lingual surfaces from abrasion with the tongue, thus worsening the condition of the durability of the teeth.

Erosion Related to Dietary Intake

Related to tooth erosion, there are many contributing factors including the oral microbiota, salivary content, and physiological conditions that in turn create suboptimal conditions for the protection and maintenance of the tooth structures. Yet, these factors are not controllable by the individual, and preventative responses are necessary to counteract their effects. However, another highly important factor that is under the control of the individual is the dietary intake and the understanding of its significance to the oral health. The intake of highly acidic beverages, such as soft drinks and sports drinks, creates a low pH environment while also supplying oral microbes that are dependent on carbohydrates with fuel to produce more acids. Carbohydrate-dependent microbes like *Streptococcus mutans* are aciduric microbes and thus can survive in acidic conditions resulting from low salivary flow and acid intake (Loesche, 1996). To provide a reference for the acidity of common beverages containing various acids, it is known that Diet Coke for instance contains phosphoric acid (pKa 2.2), citric acid (pKa 2.8), and tartaric acid (pKa 3.0), which are all more acidic than the lactic acid that is produced by *Streptococcus mutans.* Common sports drinks, such as Gatorade, also contain citric acid. Although the teeth are exposed to these acids for a relatively short period of time through

each sip, remnants of the ingredients remain interspersed in the saliva and thus remain in close contact with the tooth enamel.

As discussed previously, the saliva produced possesses buffering capability because of its composition of bicarbonate, but the flood of acidic fluid quickly overwhelms the buffering capacity of the saliva. The mention of sports drinks in particular carries some sense of irony because, as described by a study on dental erosion in athletes consuming sports drinks, the focus of those individuals is a healthy lifestyle through the consumption of nutritious foods and exercise, but the effects of these beverages are often overlooked (Sirimaharaj, et al., 2002). It has been considered whether the carbonation of some drinks is a factor in tooth erosion, but it is not believed that tooth erosion can be progressed by carbonation alone but by the added ingredients to the beverages. As such, carbonated water is a positive substitute for soft drinks as they contain minimal additives that have the potential to advance the erosion of the teeth. Carbonated water does still contain some acidic materials, such as carbonic acid (pKa 6.4) which arises from the infusion of the beverage with carbon dioxide under pressure. Compared to the acidic substances in soft drinks, consumption of carbonated water would be expected to have a lesser impact on the durability of the enamel, and the saliva would be more effective in buffering the pH and keeping it between the optimal 6.2 to 7.6 range.

In a similar way, the consumption of carbohydrate-rich foods provides carbohydrate-dependent microorganisms with fuel to produce lactic acid and cause dental caries. Carbohydrate-rich foods are particularly troublesome because remnants are commonly left behind following the mastication and swallowing of these food. Put simply, foods can become packed densely on the occlusal (biting) surfaces of bicuspids

and molars, and remnants can be hidden in the small spaces between teeth, allowing bacteria to colonize and damage the enamel. Additionally, many dentists also state that hard foods such as sunflower seeds, nuts, or even the practice of chewing ice, can have an abrasive effect on the occlusal surface of the teeth (Abrasion–Preston Dental Centre, n.d.). Similar to cases where individuals have a habit of clenching or grinding their teeth, the chewing of hard foods can forcefully wear away the occlusal. Therefore, careful consideration must be applied to the potential effects of prolonged consumption of foods and beverages on the teeth as well as the permanent damage that can result.

Tongue Abrasion

An *in vitro* study conducted by Gregg et al. (2004) proposed that the softening of tooth structures by acids taken in through the diet (citric acid used in the study; pKa 2.8) combined with the abrasive effects of contact with the tongue through licking showed additional erosion of the enamel. This study used a method of brief citric acid immersion to elicit softening and erosion, followed by tongue abrasion, and the results were positive for a loss of enamel substance as a result of tongue abrasion (Gregg et al., 2004).

An additional study published in 2017 measured effects of tongue abrasion on softened tooth enamel *in vivo* using an intraoral appliance. It was observed that the tongue aids in reducing the thickness of the dental pellicle, which is understood to be a precursor to dental plaque (Ablal, 2017). However, it was also observed that, after a 4-week study period, the anterior teeth exhibited a greater loss of enamel than posterior teeth, and it was observed that the maxillary teeth do not have any variation from the mandibular teeth in the degree of enamel loss. The data from these studies both suggest

that tongue abrasion in conjunction with enamel softening from dietary acids leads to the additional loss of enamel substance.

Preventative Measures

The structures of the enamel serve an important role as a protective coat for the underlying structures and as effective provider of stability to the teeth as tools for mastication and digestion. However, the enamel does not have the capability to reproduce itself when damage is afflicted, highlighting the importance of personal hygiene and professional monitoring.

Individually, the practice of brushing teeth following every meal can ensure that bacteria like *Streptococcus mutans* are not supplied with fuel for acid synthesis for prolonged periods of time by physically removing remnants and debris from the teeth. Additionally, this practice can disrupt the existing plaque deposits on the surface of the teeth, and thus disrupt the activity of the bacteria present. Dental plaque is easily recognizable to individuals as it results in a fuzzy feeling on the surface or the teeth, and this can be apparent after prolonged periods without brushing teeth. It is also recommended to use a toothpaste containing minerals such as fluoride. As discussed, fluorapatite crystallites are present within the crystal structure of the tooth enamel along with hydroxyapatite crystallites, and they serve to enhance the hardness and durability of the tooth enamel. Both hydroxyapatite and fluorapatite crystals are believed to have about the same hardness as they both are given a score of five on the Mohs hardness scale, comparable to diamond which has a hardness of ten and bone with also has a hardness of five (Fluorapatite, n.d.; Hydroxylapatite, n.d.; Mohs hardness, n.d.) In the same way, a

regular supply of fluoride ions through fluoride-containing toothpastes can assist in maintaining the durability of the enamel.

Similar to brushing, the practice of flossing serves to remove excess debris from the crevices that are unreachable with tooth brushing. As discussed, plaque can build up on the surface of the teeth nearest to the gingival lining, and it can also build up underneath the gingival tissue. Regular flossing can work to clear significant amounts of debris and plaque formations in those types of hidden places. Insufficient flossing schedules are evident based on the gingival tissue's response to flossing, as the tissues can become inflamed shortly after flossing. A case study conducted by Suhana et al. (2020) described a patient dealing with bleeding of the gums and inflammation upon brushing and stated that the most likely diagnosis was plaque-induced gingivitis because of residual plaque buildup. Many dental professionals also advise the use of a tool called a water pick, which is a small water pump linked to a reservoir. It serves to aggressively flush out debris from hard-to-reach places, while also preventing tissue damage from improper flossing. This flossing technique is also highly convenient for individuals with braces or dental appliances as it eliminates the need for a threading mechanism to floss.

To combat against the harmful effects of some members of the oral microbiota, many mouthwashes are available that serve as antiseptics that prevent the continual growth of bacteria. In the presence of antiseptics, bacteria are not able to grow, so mouthwash use after teeth brushing delays the growth of bacteria for the duration of the antiseptic. This practice can be effective in reducing the quantity of harmful bacteria, but it can also reduce the growth of the many bacteria that are important to maintaining the condition of the mouth. One study conducted in 2015 by Haerian-Ardakani et al.

researched the antimicrobial effects of various common mouth rinses and observed effective bacteria viability reduction after two weeks of consistent use which was said to be a necessary factor for effective results. However, the study tested against both positive and negative contributors to oral health, and reduced bacteria levels were observed in both categories (Haerian-Ardakani et al., 2015). This result highlights the debate of whether the reduction of the overall oral microflora is necessary for promoting oral health.

A similar effect can be observed with the use of antibiotic treatments in an attempt to reduce harmful bacteria. Studies have shown that the use of probiotics are a positive substitute for antibiotics and antiseptics such that, instead of reducing the growth of bacteria and potentially altering the microbial community, specific probiotics promote the growth of bacteria that are known to have positive contributions in the mouth (Ferrer et al., 2020). Ferrer et al. recognized the counterproductivity of common antiseptics against oral disease as they disrupt the activity of positive contributors in the mouth. To identify a probiotic substitute, researchers experimented with *Streptococcus dentisani* as a probiotic in this clinical trial and found that *S. dentisani* probiotics improve the mineral content of salivary excretions promoting pH buffering and inhibiting demineralization. Further, increased levels of *S. dentisani* led to reduced levels of bacteria commonly serving as structural contributors to plaque formation. As shown from this clinical trial, the promotion of the growth of positive bacteria amplifies its activity and improves the condition of the mouth by inhibiting the activity of bacteria associated with oral diseases.

Dental professionals are also able to provide unique services to prevent and counteract the effects of deterioration. Through persistence in attending biannual dental

visits, evaluations are given on the quality of personal oral hygiene, and incomplete practices can be improved. Additionally, regular professional supervision is necessary for the monitoring for progressive diseases that are unable to be stopped alone. In addition to the evaluative benefits of visiting the dentist, dental professionals are also able to administer dental sealants to the teeth. Dental sealants are a type of ceramic and plastic compound that coats the crown of the teeth, and it serves as a shield for the enamel against acidic environments and harmful bacteria (Dental Sealant FAQs, n. d.). These sealants, however, are not permanent and must be replaced periodically. Additionally, fluoride treatments are available, which coats the tooth in a fluoride-rich gel that supplies the enamel with fluoride ions and provides additional durability in the same manner as the fluoride-rich toothpastes.

To prevent against teeth grinding and a deteriorated occlusal service, dental care providers are able to construct specialized plastic retainers that take the impact of excessive biting and the abrasion of tooth-to-tooth contact. Dental professionals have a wide range of techniques available for the prevention of tooth decay. However, the effectiveness of their practices is largely dependent on the cooperativity of the patient.

Conclusion

The development of the adolescent and adult dentition is a highly intricate process that is carried out in three main stages: the bud stage, cap stage, and bell stage. Through these stages, the various layers of tissue undergo specific changes in shape, orientation and composition, and their activity is controlled by the expression of various proteins, making tooth development a highly regulated process. In the late stages of tooth development, the protective layer known as the enamel is excreted in the form of a

gelatinous matrix rich in proteins and ions. They are excreted and arranged in a specific orientation so that through the deposition of the hydroxyapatite and fluorapatite crystallites, a highly ordered tissue structure is achieved. Through the deposition and expansion of these crystallites, the mature enamel is formed with a durability that makes it the hardest tissue structure in the human body. The basic nature of these crystals makes them susceptible to dissolution by acidic substances that can come from several sources.

The bacteria *Streptococcus mutans*, which is a carbohydrate-dependent organism that obtains its energy through glycolysis, can thrive in the oral cavity due to the continual source of carbohydrates, but a byproduct of its carbohydrate metabolism is lactic acid. This acid is excreted, and the basic enamel becomes susceptible to the acid, and its crystals are prone to dissolve. The same effects arise through controllable factors such as an individual's diet. The intake of acidic and carbohydrate-rich foods and beverages causes the tooth enamel to progressively erode, and abrasion from the tongue also causes additional deterioration. The tooth enamel is incapable of regenerating, and therefore, great care must be taken to protect it so that it can last the duration of one's life. Personal practices, such as thorough brushing and flossing can reduce the effects of deterioration, and coupled with professional intervention, the durability and lifespan of the tooth enamel can be significantly prolonged.

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