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then ionizes. The lactate ions demineralize the hydroxyapatite crystals causing the tooth to degrade. The progression of pit and fissure caries resembles two triangles with their bases meeting along the junction of enamel and dentin. Teeth are bathed in saliva and have a coating of bacteria on them (biofilm) that continually forms. The development of biofilm begins with pellicle formation. Pellicle is an acellular proteinaceous film which covers the teeth. Bacteria colonize on the teeth by adhering to the pellicle-coated surface. Over time, a mature biofilm is formed, creating a cariogenic environment on the tooth surface.[73][74] The minerals in the hard tissues of the teeth – enamel, dentin and cementum – are constantly undergoing demineralization and remineralization. Dental caries result when the demineralization rate is faster than the remineralization, producing net mineral loss, which occurs when there is an ecologic shift within the dental biofilm from a balanced population of microorganisms to a population that produces acids and can survive in an acid environment.[75] Tooth enamel is a highly mineralized acellular tissue, and caries act upon it through a chemical process brought on by the acidic environment produced by bacteria. As the bacteria consume the sugar and use it for their own energy, they produce lactic acid. The effects of this process include the demineralization of crystals in the enamel, caused by acids, over time until the bacteria physically penetrate the dentin. Enamel rods, which are the basic unit of the enamel structure, run perpendicularly from the surface of the tooth to the dentin. Since demineralization of enamel by caries follows the direction of the enamel rods, the different triangular patterns between pit and fissure and smooth-surface caries develop in the enamel because the orientation of enamel rods are different in the two areas of the tooth.[76] As the enamel loses minerals, and dental caries progresses, the enamel develops several distinct zones, visible under a light microscope. From the deepest layer of the enamel to the enamel surface, the identified areas are the: translucent zone, dark zones, body of the lesion, and surface zone.[77] The translucent zone is the first visible sign of caries and coincides with a one to two percent loss of minerals.[78] A slight remineralization of enamel occurs in the dark zone, which serves as an example of how the development of dental caries is an active process with alternating changes.[79] The area of greatest demineralization and destruction is in the body of the lesion itself. The surface zone remains relatively mineralized and is present until the loss of tooth structure results in a cavitation. Unlike enamel, the dentin reacts to the progression of dental caries. After tooth formation, the ameloblasts, which produce enamel, are destroyed once enamel formation is complete and thus cannot later regenerate enamel after its destruction. On the other hand, dentin is produced continuously throughout life by odontoblasts, which reside at the border between the pulp and dentin. Since odontoblasts are present, a stimulus, such as caries, can trigger a biologic response. These defense mechanisms include the formation of sclerotic and tertiary dentin.[80] In dentin from the deepest layer to the enamel, the distinct areas affected by caries are the advancing front, the zone of bacterial penetration, and the zone of destruction.[76] The advancing front represents a zone of demineralized dentin due to acid and has no bacteria present. The zones of bacterial penetration and destruction are the locations of invading bacteria and ultimately the decomposition of dentin. The zone of destruction has a more mixed bacterial population where proteolytic enzymes have destroyed the organic matrix. The innermost dentin caries has been reversibly attacked because the collagen matrix is not severely damaged, giving it potential for repair. The faster spread of caries through dentin creates this triangular appearance in smooth surface caries. The structure of dentin is an arrangement of microscopic channels, called dentinal tubules, which radiate outward from the pulp chamber to the exterior cementum or enamel border.[81] The diameter of the dentinal tubules is largest near the pulp (about 2.5 µm) and smallest (about 900 nm) at the junction of dentin and enamel.[82] The carious process continues through the dentinal tubules, which are responsible for the triangular patterns resulting from the progression of caries deep into the tooth. The tubules also allow caries to progress faster. In response, the fluid inside the tubules brings immunoglobulins from the immune system to fight the bacterial infection. At the same time, there is an increase of mineralization of the surrounding tubules.[83] This results in a constriction of the tubules, which is an attempt to slow the bacterial progression. In addition, as the acid from the bacteria demineralizes the hydroxyapatite crystals, calcium and phosphorus are released, allowing for the precipitation of more crystals which fall deeper into the dentinal tubule. These crystals form a barrier and slow the advancement of caries. After these protective responses, the dentin is considered sclerotic. According to hydrodynamic theory, fluids within dentinal tubules are believed to be the mechanism by which pain receptors are triggered within the pulp of the tooth.[84] Since sclerotic dentin prevents the passage of such fluids, pain that would otherwise serve as a warning of the invading bacteria may not develop at first. See also: Tertiary dentin In response to dental caries, there may be production of more dentin toward the direction of the pulp. This new dentin is referred to as tertiary dentin.[82] Tertiary dentin is produced to protect the pulp for as long as possible from the advancing bacteria. As more tertiary dentin is produced, the size of the pulp decreases. This type of dentin has been subdivided according to the presence or absence of the original odontoblasts.[85] If the odontoblasts survive long enough to react to the dental caries, then the dentin produced is called "reactionary" dentin. If the odontoblasts are killed, the dentin produced is called "reparative" dentin. In the case of reparative dentin, other cells are needed to assume the role of the destroyed odontoblasts. Growth factors, especially TGF-β,[85] are thought to initiate the production of reparative dentin by fibroblasts and mesenchymal cells of the pulp.[86] Reparative dentin is produced at an average of 1.5 µm/day, but can be increased to 3.5 µm/day. The resulting dentin contains irregularly shaped dentinal tubules that may not line up with existing dentinal tubules. This diminishes the ability for dental caries to progress within the dentinal tubules. The incidence of cemental caries increases in older adults as gingival recession occurs from either trauma or periodontal disease. It is a chronic condition that forms a large, shallow lesion and slowly invades first the root's cementum and then dentin to cause a chronic infection of the pulp (see further discussion under classification by affected hard tissue). Because dental pain is a late finding, many lesions are not detected early, resulting in restorative challenges and increased tooth loss.[87] The tip of a dental explorer, which is used for caries diagnosis A dental infection resulting in an abscess and inflammation of the maxillary sinus Tooth samples imaged with a non-coherent continuous light source (row 1), LSI (row 2) and pseudo-color visualization of LSI (row 3)[88] The presentation of caries is highly variable. However, the risk factors for dental caries are similar. Initially, it may appear as a small chalky area (smooth surface caries), which may eventually develop into a large cavitation. Sometimes caries may be directly visible. However other methods of detection such as X-rays are used for less visible areas of teeth and to judge the extent of destruction. Lasers for detecting caries allow detection without ionizing radiation and are now used for detection of interproximal decay (between the teeth). Primary diagnosis involves inspection of all visible tooth surfaces using a good light source, dental mirror and explorer. Dental radiographs (X-rays) may show dental caries before it is otherwise visible, in particular caries between the teeth. Large areas of dental caries are often apparent to the naked eye, but smaller lesions can be difficult to identify. Visual and tactile inspection along with radiographs are employed frequently among dentists, in particular to diagnose pit and fissure caries.[89] Early, uncavitated caries is often diagnosed by blowing air across the suspect surface, which removes moisture and changes the optical properties of the unmineralized enamel. Some dental researchers have cautioned against the use of dental explorers to find caries,[90] in particular sharp ended explorers. In cases where a small area of tooth has begun demineralizing but has not yet cavitated, the pressure from the dental explorer could cause a cavity. Since the carious process is reversible before a cavity is present, it may be possible to arrest caries with fluoride and remineralize the tooth surface. When a cavity is present, a restoration will be needed to replace the lost tooth structure. At times, pit and fissure caries may be difficult to detect. Bacteria can penetrate the enamel to reach dentin, but then the outer surface may remineralize, especially if fluoride is present.[91] These caries, sometimes referred to as "hidden caries", will still be visible on X-ray radiographs, but visual examination of the tooth would show the enamel intact or minimally perforated. The differential diagnosis for dental caries includes dental fluorosis and developmental defects of the tooth including hypomineralization of the tooth and hypoplasia of the tooth.[92] The early carious lesion is characterized by demineralization of the tooth surface, altering the tooth's optical properties. Technology using laser speckle image (LSI) techniques may provide a diagnostic aid to detect early carious lesions.[88] G. V. Black Classification of Restorations Caries can be classified by location, etiology, rate of progression, and affected hard tissues.[93] These forms of classification can be used to characterize a particular case of tooth decay to more accurately represent the condition to others and also indicate the severity of tooth destruction. In some instances, caries is described in other ways that might indicate the cause. The G. V. Black classification is as follows: Class I: occlusal surfaces of posterior teeth, buccal or lingual pits on molars, lingual pit near cingulum of maxillary incisors Class II: proximal surfaces of posterior teeth Class III: interproximal surfaces of anterior teeth without incisal edge involvement Class IV: interproximal surfaces of anterior teeth with incisal edge involvement Class V: cervical third of facial or lingual surface of tooth Class VI: incisal or occlusal edge is worn away due to attrition Rampant caries caused by methamphetamine abuse Early childhood caries (ECC), also known as "baby bottle caries," "baby bottle tooth decay" or "bottle rot," is a pattern of decay found in young children with their deciduous (baby) teeth. This must include the presence of at least one carious lesion on a primary tooth in a child under the age of 6 years.[94] The teeth most likely affected are the maxillary anterior teeth, but all teeth can be affected.[95] The name for this type of caries comes from the fact that the decay usually is a result of allowing children to fall asleep with sweetened liquids in their bottles or feeding children sweetened liquids multiple times during the day.[96] Another pattern of decay is "rampant caries", which signifies advanced or severe decay on multiple surfaces of many teeth.[97] Rampant caries may be seen in individuals with xerostomia, poor oral hygiene, stimulant use (due to drug-induced dry mouth[98]), and/or large sugar intake. If rampant caries is a result of previous radiation to the head and neck, it may be described as radiation-induced caries. Problems can also be caused by the self-destruction of roots and whole tooth resorption when new teeth erupt or later from unknown causes. Children at 6–12 months are at increased risk of developing dental caries.[99] A range of studies have reported that there is a correlation between caries in primary teeth and caries in permanent teeth.[100][101] This section needs additional citations for verification. Please help improve this article by adding citations to reliable sources in this section. Unsourced material may be challenged and removed. (November 2016) (Learn how and when to remove this message) Temporal descriptions can be applied to caries to indicate the progression rate and previous history. "Acute" signifies a quickly developing condition, whereas "chronic" describes a condition that has taken an extended time to develop, in which thousands of meals and snacks, many causing some acid demineralization that is not remineralized, eventually result in cavities. Recurrent caries, also described as secondary, are caries that appear at a location with a previous history of caries. This is frequently found on the margins of fillings and other dental restorations. On the other hand, incipient caries describes decay at a location that has not experienced previous decay. Arrested caries describes a lesion on a tooth that was previously demineralized but was remineralized before causing a cavitation. Fluoride treatment can help recalcification of tooth enamel as well as the use of amorphous calcium phosphate. Micro-invasive interventions (such as dental sealant or resin infiltration) have been shown to slow down the progression of proximal decay.[102] Depending on which hard tissues are affected, it is possible to describe caries as involving enamel, dentin, or cementum. Early in its development, caries may affect only enamel. Once the extent of decay reaches the deeper layer of dentin, the term "dentinal caries" is used. Since cementum is the hard tissue that covers the roots of teeth, it is not often affected by decay unless the roots of teeth are exposed to the mouth. Although the term "cementum caries" may be used to describe the decay on roots of teeth, very rarely does caries affect the cementum alone. Toothbrushes are commonly used to clean teeth. The primary approach to dental hygiene care consists of tooth-brushing and flossing. The purpose of oral hygiene is to remove and prevent the formation of plaque or dental biofilm,[103] although studies have shown this effect on caries is limited.[104] While there is no evidence that flossing prevents tooth decay,[105] the practice is still generally recommended.[5] A toothbrush can be used to remove plaque on accessible surfaces, but not between teeth or inside pits and fissures on chewing surfaces. When used correctly, dental floss removes plaque from areas that could otherwise develop proximal caries but only if the depth of sulcus has not been compromised. Additional aids include interdental brushes, water picks, and mouthwashes. The use of rotational electric toothbrushes might reduce the risk of plaque and gingivitis, though it is unclear whether they are of clinical importance.[106] However, oral hygiene is effective at preventing gum disease (gingivitis / periodontal disease). Food is forced inside pits and fissures under chewing pressure, leading to carbohydrate-fuelled acid demineralisation where the brush, fluoride toothpaste, and saliva have no access to remove trapped food, neutralise acid, or remineralise tooth enamel. (Occlusal caries accounts for between 80 and 90% of caries in children (Weintraub, 2001).) Unlike brushing, fluoride leads to proven reduction in caries incidence by approximately 25%: higher concentrations of fluoride (>1,000 ppm) in toothpaste also helps prevents tooth decay, with the effect increasing with concentration up to a plateau.[107] A randomized clinical trial demonstrated that toothpastes that contain arginine have greater protection against tooth cavitation than the regular fluoride toothpastes containing 1450 ppm alone.[108] A Cochrane review has confirmed that the use of fluoride gels, normally applied by a dental professional from once to several times a year, assists in the prevention of tooth decay in children and adolescents, reiterating the importance of fluoride as the principal means of caries prevention.[109] Another review concluded that the supervised regular use of a fluoride mouthwash greatly reduced the onset of decay in the permanent teeth of children.[110] Professional hygiene care consists of regular dental examinations and professional prophylaxis (cleaning). Sometimes, complete plaque removal is difficult, and a dentist or dental hygienist may be needed. Along with oral hygiene, radiographs may be taken at dental visits to detect possible dental caries development in high-risk areas of the mouth (e.g. "bitewing" X-rays which visualize the crowns of the back teeth). Alternative methods of oral hygiene also exist around the world, such as the use of teeth cleaning twigs such as miswak in some Middle Eastern and African cultures. There is some limited evidence demonstrating the efficacy of these alternative methods of oral hygiene.[111] Annual caries incidence increases exponentially with annual per capita sugar consumption. Data based on 10,553 Japanese children whose individual lower first molar teeth were monitored yearly from the age of 6 to 11 years of age. Caries plotted on a logarithmic scale, so line is straight. People who eat more free sugars get more cavities, with cavities increasing exponentially with increasing sugar intake. Populations with less sugar intake have fewer cavities. In one population, in Nigeria, where sugar consumption was about 2g/day, only two percent of the population, of any age, had had a cavity.[112] Chewy and sticky foods (such as candy, cookies, potato chips, and crackers) tend to adhere to teeth longer. However, dried fruits such as raisins and fresh fruit such as apples and bananas disappear from the mouth quickly, and do not appear to be a risk factor. Consumers are not good at guessing which foods stick around in the mouth.[113] For children, the American Dental Association and the European Academy of Paediatric Dentistry recommend limiting the frequency of consumption of drinks with sugar, and not giving baby bottles to infants during sleep (see earlier discussion).[114][115] Parents are also recommended to avoid sharing utensils and cups with their infants to prevent transferring bacteria from the parent's mouth.[116] Xylitol is a naturally occurring sugar alcohol that is used in different products as an alternative to sucrose (table sugar). As of 2015 the evidence concerning the use of xylitol in chewing gum was insufficient to determine if it is effective at preventing caries.[117][118][119] Common dentistry trays used to deliver fluoride Fluoride is sold in tablets for cavity prevention. The use of dental sealants is a means of prevention.[120] A sealant is a thin plastic-like coating applied to the chewing surfaces of the molars to prevent food from being trapped inside pits and fissures. This deprives resident plaque bacteria of carbohydrate, preventing the formation of pit and fissure caries. Sealants are usually applied on the teeth of children, as soon as the teeth erupt but adults are receiving them if not previously performed. Sealants can wear out and fail to prevent access of food and plaque bacteria inside pits and fissures and need to be replaced so they must be checked regularly by dental professionals. Dental sealants have been shown to be more effective at preventing occlusal decay when compared to fluoride varnish applications.[121][needs update] Calcium, as found in food such as milk and green vegetables, is often recommended to protect against dental caries. Fluoride helps prevent decay of a tooth by binding to the hydroxyapatite crystals in enamel.[122] Streptococcus mutans is the leading cause of tooth decay. Low concentration fluoride ions act as bacteriostatic therapeutic agent and high concentration fluoride ions are bactericidal.[123] The incorporated fluorine makes enamel more resistant to demineralization and, thus, resistant to decay.[124] Fluoride can be found in either topical or systemic form.[125] Topical fluoride is more highly recommended than systemic intake to protect the surface of the teeth.[126] Topical fluoride is used in toothpaste, mouthwash and fluoride varnish.[125] Standard fluoride toothpaste (1,000–1,500 ppm) is more effective than low fluoride toothpaste (< 600ppm) to prevent dental caries.[127] It is recommended that all adult patients to use fluoridated toothpaste with at least 1350ppm fluoride content, brushing at least 2 times per day and brush right before bed. For children and young adults, use fluoridated toothpaste with 1350ppm to 1500ppm fluoride content, brushing 2 times per day and also brush right before bed. American Dental Association Council suggest that for children