Caries Process and Prevention Strategies: 
The Agent

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Continuing Education Units: 1 hour


**Disclaimer:** Participants must always be aware of the hazards of using limited knowledge in integrating new techniques or procedures into their practice. Only sound evidence-based dentistry should be used in patient therapy.

This is part 2 of a 10-part series entitled *Caries Process and Prevention Strategies*. Dental caries is a multifactorial, infectious disease affecting a significant percentage of the population. This course describes the etiology and pathways of progression of dental caries, including an in-depth review of the role of dental plaque and oral bacteria.

**Conflict of Interest Disclosure Statement**
- The author reports no conflicts of interest associated with this work.

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Overview

Dental caries is arguably the most prevalent disease in man, affecting most of the dentate population at some time in their lives. In the United States, dental caries is the most common chronic disease in childhood, with 41% of children between the ages of 2 and 11 having caries in primary teeth. Among dentate adults aged 20 to 64, 92% have caries in permanent teeth. Commonly termed “tooth decay,” caries is the localized destruction of tooth tissues over time by acid that is produced in the mouth when oral bacteria, such as Streptococcus mutans, ferment dietary carbohydrates. These bacteria aggregate in dental plaque that forms on the outer surface of teeth. In a healthy mouth environment, the bacteria that populate plaque are harmless, but when the environment becomes acidic, the population changes to bacteria that thrive in acidity and are linked to caries. A combination of several factors and sub-factors are required for dental caries to develop, including some that are innate to the oral environment, making caries a multifactorial disease that can be difficult to manage and completely prevent. The caries process, the multiple factors that influence caries development, and plaque as a microbial biofilm ecosystem are discussed.

Clinical Significance Snapshots

Simply put, what causes dental decay? How can I explain it to my patients?

Dental decay is caused when bacteria that accumulate on the surfaces of the teeth feed on sugars in the diet, and convert the sugars into acids that then dissolve the hard tooth material. This results in the loss of minerals, which in turn results in cavities.

Nearly every mouth contains the bacteria that can cause decay. The mouth can withstand several attacks each day from the bacteria that turn sugar into acid. During times between meals (with sugar), it is possible for the tooth to repair itself, replacing the minerals that have been dissolved by the acids. Fluoride in toothpaste helps make teeth more resistant to the acid attack. It is important to clean teeth well to remove as much bacteria as possible, and for those at high risk of developing caries to finish meals with items that are rich in calcium, such as yogurt, milk, or cheese.

Once sufficient mineral has been lost, the tooth forms a cavity that can only be repaired by the dentist placing a filling. The decay process starts with the appearance of a white spot on the surface of the tooth.

Of all the factors listed, what are the most important to control in order to prevent the onset of dental caries?

The frequency of intake of sugars should be reduced as much as possible, and, ideally, limited to mealtimes, so that acids in dental plaque are only produced 3 or 4 times a day, and that there is plenty of time between meals for saliva to act by replacing any minerals that have been dissolved by the acid production during mealtimes. The presence of fluoride makes enamel more resistant to acid dissolution and encourages the process of remineralization. Removal of plaque biofilm is important too, though it is impossible to remove all decay-causing bacteria from the mouth. Therefore, the next most important action, after reducing the frequency of sugars in the diet, is to brush at least twice a day with a fluoride toothpaste that strengthens the enamel against acid attack, encourages remineralization, and removes the plaque biofilm.
Learning Objectives

Upon completion of this course, the dental professional should be able to:
• Define dental caries.
• Discuss the medical history of caries along with its natural history.
• Identify the combination of factors required for caries to develop, and how sub-factors influence this process.
• Define dental plaque as a microbial biofilm.
• Describe the development and maturation of dental plaque.
• Understand the microbial diversity of plaque and recognize it as an ecosystem.
• Discuss the ecological plaque hypothesis.
• Name the bacteria associated with caries.
• Discuss how the acidity in the oral environment is the major determinant of plaque ecology.
• Identify how bacteria convert dietary carbohydrates to acids.

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Glossary
acidogenic – Something that produces acid, such as cariogenic bacteria.

aciduric – Capable of growth in an acidic environment.

allogenic – Denoting individuals of the same species but of different genetic constitution (antigenically distinct).

anaerobic – Living in the absence of air or free oxygen.

biofilm – An aggregation of microorganisms in which cells adhere to each other forming small communities that are held together by an extracellular polymeric matrix. Different communities are co-dependent on each other, and the whole biofilm forms a defensive mechanism requiring much higher concentrations of antimicrobials to control its growth. Dental plaque is a classic biofilm.

buffering capacity – Saliva and the fluid in dental plaque possess the ability to buffer. Buffering adjusts the pH of any solution such as saliva or plaque fluid and can resist changes in pH. Buffering capacity is the degree of buffering that can be brought about.

cariogenic – The ability to cause dental caries. A cariogenic diet contains sugars. Some bacteria in dental plaque (S. mutans) are cariogenic. The mere presence of cariogenic sugars or cariogenic bacteria are not enough to cause the initiation of the caries process. Many other factors play a role, and taken together they may or may not contribute to the process that leads to dental caries.

demineralization – The chemical process by which minerals (mainly calcium) are removed from the dental hard tissues - enamel, dentin, and cementum. The chemical process occurs through dissolution by acids or by chelation, and the rate of demineralization will vary due to the degree of supersaturation of the immediate environment of the tooth and the presence of fluoride. In optimal circumstances, the minerals may be replaced through the process of remineralization.

dental plaque – An organized community of many different microorganisms that forms itself into a biofilm and is found on the surface of the tongue.
and all hard surfaces in the oral cavity. Dental plaque is present in all people and can vary from being comprised of totally healthy microorganisms (commensals) to being very harmful (pathogenic), predisposing the patient to dental caries or periodontal diseases. Note: Dental plaque is not food debris, nor does it contain food debris. Dental plaque can only be completely removed by mechanical means such as toothbrushing or prophylaxis. Food debris can be removed by rinsing.

**disaccharides** – Any group of carbohydrates, such as sucrose or lactose, that yield monosaccharides on hydrolysis; also called double sugars.

**enzyme** – Protein that catalyzes, or facilitates, biochemical reactions.

**fructosyltransferase (FTF)** – An enzyme that catalyzes the breakdown of fructose, liberating glucose.

**glycolysis** – Glycolysis is essential in all living organisms, and is the process whereby energy is released from sugars by the formation of pyruvate.

**glycoprotein** – Any of a group of conjugated proteins that contain a carbohydrate as the non-protein component.

**glycosidic** – Any of a group of organic compounds that yield a sugar and one or more non-sugar substances on hydrolysis.

**invertase** – An enzyme derived from yeast that has the ability to break sucrose down into the simple sugars glucose and fructose.

**lipids** – Any of a group of organic compounds, including the fats, oils, waxes, sterols, and triglycerides, that are insoluble in water but soluble in common organic solvents, are oily to the touch, and together with carbohydrates and proteins constitute the principal structural material of living cells.

**monosaccharides** – The simplest forms of carbohydrates (sugar).

**pellicle** – A thin, acellular membrane of salivary proteins adsorbed to the enamel or cementum.

**phosphoproteins** – Proteins that contain phosphate groups esterified to serine, threonine or tyrosine. The phosphate group usually regulates protein function.

**pili** – A hair-like appendage found on the surface of many bacteria.

**polysaccharides** – Chains of sugar units that are held together by glycosidic bonds.

**prophylaxis** – The clinical procedure that removes plaque, calculus and stain in a procedure carried out by a dental professional.

**remineralization** – The chemical process by which minerals (mainly calcium) are replaced into the substance of the dental hard tissues - enamel, dentin and cementum. The process requires an ideal environment that includes supersaturation with calcium and phosphate ions, the presence of fluoride, and adequate buffering.

**substrate** – Substrate is the material metabolized by specific microorganisms in dental plaque to produce the acids that lead to demineralization. The substrate is typically a sugar such as sucrose, glucose, and fructose occurring in foods and beverages. Substrate is more of a theoretical term; in practice it is sugars that are used by the microorganisms to produce acid in the process of dental caries.

**Introduction**

Dental caries is a biofilm disease that results in the localized destruction of tooth tissues by acid, such as lactic acid, that is produced in the mouth as oral bacteria ferment dietary carbohydrates. If the pH in the environment surrounding tooth tissues becomes too acidic, dropping below a pH of 5.5, then demineralization of tooth enamel—essentially dissolution of tooth structure—begins to occur. The early stages are reversible, because the natural process of remineralization can replace lost enamel. However, if demineralization continues over time, enough mineral content may be lost so that the soft organic matrix left behind disintegrates, forming a cavity.
Dental caries is an infectious disease, but technically, although it is transmissible, one does not “catch” dental caries. The oral bacteria that cause dental caries when they thrive under certain specific conditions populate the oral cavity of all humans, first entering the body when a baby passes through the birth canal. It is more accurate to consider caries as caused, not by an infectious agent, but by a shift in oral microflora to caries-causing bacterial types in response to a shift to an acidic pH caused by metabolism of sugars.

Theories about what cause cavities go as far back as 2500 BC in ancient China when it was thought that “toothworms” caused cavities. This belief continued for several centuries in many different cultures. Later, in 350 BC, Aristotle and others acknowledged that sweets and figs caused decay. It wasn’t until 1819, that Levi Parmly hinted at the real cause of caries: that decay begins on the surface of the teeth by bacteria growing on food particles which lodge around and between teeth, causing destruction of tooth structure.

Caries theory was marked in the 1880s by Miles and Underwood stating in 1881 that acid and “germs” were necessary for decay, while W.D. Miller formulated the concept of caries as a local phenomenon associated with carbohydrate retention and acidogenic bacteria in 1889. In the early to mid-1900s, dental research uncovered several important findings: In 1938, H. Trendley Dean linked fluoride to caries reduction, and in later studies, high sugar consumption was linked to caries, but only in an environment where oral bacteria were present.

In 1955, Procter & Gamble introduced Crest, the first fluoride toothpaste clinically proven to be effective in preventing dental caries. It was hailed as a major scientific breakthrough, and received an endorsement from the American Dental Association (ADA) as an “effective decay-preventive dentifrice that can be of significant value.” In the 1990s, and repeatedly since, the ADA has emphasized the benefit of fluoride, stating: “A comprehensive analysis of the fifty-year history of community water fluoridation in the United States further demonstrated that the inverse relationship between higher fluoride concentration in drinking water and lower levels of dental decay discovered a half-century ago continued to be true.”

Dental Caries
A Multi-factorial Disease

The development of caries is dependent on the interaction of four primary factors. These are a host (tooth surface), a substrate (food), the presence of oral bacteria, and time. Caries will not develop if any of these four primary factors are not present.

(See “Tooth Decay” on pages 15-16)

Each of the four primary factors can be further divided into sub-factors that also influence the likelihood of caries (Figure 1).

1. Host (tooth surface): The sub-factors that influence caries development are age (the enamel of the deciduous teeth of children is more susceptible to acid demineralization), if fluoride has been used, tooth morphology (which varies within the mouth and from person to person), root surface exposure due to gum recession, nutrition (if tooth-strengthening nutrients are consumed), and saliva flow rate and buffering capacity.

A tooth is more susceptible to caries if it has less acid resistant enamel due to age or low fluoride intake, or if the roots have been exposed by gum recession. Caries risk is also higher if the diet is low in nutrients (such as magnesium and vitamin D) that are necessary for healthy tooth development, and/or when an individual’s saliva flow rate is low or has a low buffering capacity. Pit-and-fissure demineralization is more likely to develop in teeth with numerous and exaggerated grooves. Teeth are less prone to caries activity in situations where tooth enamel has been strengthened by fluoride, a diet of tooth-strengthening nutrients is consumed, and/or the buffering capacity of saliva is high.

2. Substrate (food): The sub-factors that influence caries development are oral clearance (if food is retained or not in the mouth after eating), oral hygiene (if, after eating, food is actively removed with a sharp instrument such as a toothpick), eating frequency, food detergency (if consumed food can clean teeth), consumption of
Figure 1. The factors and sub-factors that influence caries development.

carbohydrates, and the cariogenicity of consumed carbohydrates (sucrose is more cariogenic than glucose and fructose).

When food is retained in the mouth and not actively removed after eating, is consumed more frequently, and/or more sugars, sucrose-containing foods, and sticky foods (like toffee) are consumed, there is higher risk of caries. On the other hand, when remaining food particles are actively removed after eating, food is consumed less frequently, fewer sugars, sucrose-containing foods, and sticky foods are consumed, and/or more tooth-cleaning foods (like apples) are eaten, the likelihood of caries is lower.

3. Oral bacteria: The development of caries depends on microbial load (how much bacteria is present), plaque composition (with some types of plaque microbes being more cariogenic than others), plaque acidogenicity (how much acid can be produced by the plaque that is present), plaque acidoduricity (how well plaque can survive in acidic conditions), oral hygiene (how often microbial load is reduced by brushing or prophylaxis), and if fluoride is present in plaque.

The likelihood of caries development is higher when microbial load is high as indicated by excessive plaque, when more caries-linked bacteria are present in plaque, when plaque produces more acid, when more plaque bacteria can survive in acidic conditions, and/or when plaque is not regularly removed by brushing. The odds that caries will develop are lower when the microbial load is low as indicated by little plaque, present plaque has fewer bacteria associated with caries or that can withstand very acid conditions, plaque acid production is low, and/or plaque is regularly removed by brushing or flossing.

4. Time: While the shift in microflora can occur over a fairly short period, a significant amount of time is needed for demineralization to lead to the development of whitespot and/or carious lesions. Acid production does not instantly trigger tooth decay, and in the early stages, remineralization can restore enamel, keeping the effects of dental caries at bay.

In summary, bacterial fermentation of consumed sugars produces acid in the tooth’s immediate environment. This acid demineralizes tooth enamel, and over time, this dissolution of tooth structure leads to the development of carious
lesions. Because the combination of factors and sub-factors include unavoidable situations, dental caries can be very difficult to prevent.

**Dental Plaque**

**Biofilm**

(See “Plaque and Tartar Control” on pages 17-18)

Bacteria collect on the teeth and along the edge of the gums in a cream-colored mass called plaque (Figure 2). The bacterial deposits that form plaque on teeth differ considerably from that on soft tissues because teeth are a non-shedding surface, allowing more time for the development of a “structure” consisting of multiple layers of bacteria. This plaque “structure” also serves as a biofilm, typically defined as an aggregate of microorganisms in which cells adhere to each other and/or to a solid substrate exposed to an aqueous surface. The bulk of the volume (~90%) of dental plaque biofilm is comprised of gel-like matrix of extracellular polysaccharides produced by oral bacteria—these polysaccharides are what holds the biofilm together and triggers changes that make it increasingly difficult to remove over time: When a cell becomes a component of biofilm, one of the many changes it experiences is a shift in gene expression that makes it up to 1,000 times more resistant to antibodies, antibiotics, and antimicrobial compounds than its planktonic (single cell) counterparts.⁹⁻¹¹

**Microbiology**

All oral bacteria produce acidic byproducts when they metabolize sugar, and so will cause a drop in plaque pH. However, there are two specific types of bacteria most associated with caries: *Streptococci* (most notably *Streptococcus mutans*) and *Lactobacilli*.

*Streptococci* are Gram-positive cocci that form chains, and constitute a relatively large proportion of plaque (~30% – 40%).¹⁰⁻¹³ They have very efficient sugar transport and storage systems and can produce large amounts of lactic acid when excess sugars are available, or they produce formic and acetic acids when they are utilizing their energy reserves. *S. mutans* is the strain most strongly implicated in acid production and caries.¹⁰⁻¹³ This acidogenic bacteria adheres to the biofilm on the tooth by converting sucrose into an extremely adhesive substance called dextran polysaccharide by the enzyme dextranucranase. (Note that *S. mutans* need not be present for caries to occur: Individuals without this strain can still get caries, since they may have other oral bacteria that create acid-demineraling conditions.)

*Lactobacilli* are Gram-positive rods that are only present in plaque in small numbers (~1%), but they are extremely aciduric, meaning that they can endure very acidic environments. It is quite possible that they do not significantly contribute to caries, but they are frequently isolated from caries lesions due to their ability to thrive at low pH.

**Stages of Development**

There are six stages of plaque biofilm development (Figure 3).

**Stage 1:** Formation of an acellular layer.

Called the acquired pellicle, this layer of salivary glycoproteins, phosphoproteins, and lipids, but no bacteria, forms almost immediately on naked enamel surfaces.

**Stage 2:** Initial attachment.

Free-floating early colonizers of the teeth, such as *Streptococcus sanguinis*, which are normal inhabitants on the mouth, form an initial attachment to the pellicle by weak and reversible van der Waals forces. If these bacteria are not removed, they eventually anchor themselves with adhesive structures, such as pili.
Division decrease. The heterogeneous nature of plaque becomes apparent as a mosaic of microenvironments develop, particularly areas of different pH, oxygen concentrations, and secondary metabolite accumulations around and within microcolonies. The plaque microbial ecology reaches a pseudo-steady-state climax community, where there is a constant turnover of cells, but the overall composition remains roughly the same. At this point, a thick, three-dimensional layer of dental plaque biofilm has formed.

Stage 6: Dispersion. Enzymes that degrade the biofilm (such as dispersin B) allow some bacteria to detach themselves from the biofilm—sometimes in response to deleterious environmental conditions—in order to spread and colonize new surfaces in the oral cavity.

Ecology in Health and Disease
Mature dental plaque is composed of a highly complex community of microbes, with the population of microbes varying from person to person.
person and between different sites within the mouth. Classical microbiological techniques estimated that plaque contained 800 distinct oral species, with a healthy individual possessing 50 to 100 different species at any one time. However, a powerful new molecular technique tool called pyrosequencing, which analyses ribosomal RNA, has estimated at least 19,000 phylotypes (assuming a 6% difference in RNA sequence to constitute a new species). These populations of bacteria form their own microbial ecosystem in dental plaque. Just like any other ecosystem, the plaque microbial ecosystem can both influence its environment and be influenced by its environment, which in this case is the mouth.

Production of acid by the microbes in dental plaque as they ferment consumed sugars lowers plaque pH, which causes the localized environment to change. The lowering of plaque pH causes a corresponding shift in plaque ecology, in which acid-sensitive bacteria such as S. sanguinis are less able to survive, but aciduric bacteria such as S. mutans and Lactobacilli will thrive. The end result is disruption in the natural balance between dental plaque and the tooth surface, more acid production and increased demineralization. On the other hand, when pH remains neutral, acid-sensitive bacteria like S. sanguinis can survive, keeping acid production low and increasing remineralization. This concept of the oral environment being able to cause a shift in dental plaque ecology that can either lead to good oral health or disease, such as caries and gingivitis, is referred to as the "ecological plaque hypothesis.

What drives the shift in plaque ecology is not the presence of sugars per se, but rather the acid formed by their fermentation that can cause pH to drop from a neutral 7 to a pH of lower than 5.5. At a pH of 5.5, the plaque community remains stable, but as pH drops lower to 4.5, the numbers of S. mutans and Lactobacillus increase. When plaque pH drops below 4.5, this is considered an environmental catastrophe for plaque microflora, like S. sanguinis, that normally inhabit a healthy mouth. That is because these acid-sensitive species can be inhibited or killed, while acid-tolerant species proliferate (Figure 4).

### Oral Bacteria

#### Sugar Metabolism

Dietary sugars, starches, and fermentable carbohydrates (usually collectively referred to

![Figure 4. Ecological Plaque Hypothesis.
1. Extracellular invertase cleaves the energy rich α(1-2) glycosidic bond between the glucose and fructose moieties.

2. The bacterial cell transports the sucrose across the cell membrane and cleaves the glycosidic bond using an intracellular invertase.

3. Extracellular glycosyltransferases polymerize the glucose molecule while liberating the sugars.

4. Enzymes in bacteria and saliva break down sugars' polysaccharides and disaccharides to monosaccharides. There are five main mechanisms by which oral Streptococci hydrolyse (break down) sucrose (Figure 5).

5. As sugars (as sugars) are present in the diet, and are in direct contact with plaque during eating, and for some time afterwards. The breakdown of sugars is an important step that influences the plaque environment. Enzymes in bacteria and saliva break down sugars' polysaccharides and disaccharides to monosaccharides. There are five main mechanisms by which oral Streptococci hydrolyse (break down) sucrose (Figure 5).

Figure 5. Mechanisms of sucrose hydrolysis by oral bacteria.
fructose molecule so it is free to enter the bacterial cell. *Streptococci* are particularly proficient at this.

4. Extracellular fructosyltransferases polymerize the fructose while the glucose molecule is liberated, so it is free to enter the cell.

5. Salivary amylase cleaves the polysaccharides.

**Acid Production**

Bacteria in a person’s mouth convert glucose, fructose, and sucrose into acids through a process called glycolysis, which is the main energy generating pathway in all bacteria, including *S. mutans*. The monosaccharides glucose, galactose, and fructose can enter the glycolysis pathway at the points shown in the diagram (Figure 6). The dotted lines in the pathways indicate that there are additional intermediate steps. *S. mutans* is capable of metabolizing pyruvate (pyruvic acid) further to generate yet more energy and more acid byproducts. When excess sugars are available they favor the lactate dehydrogenase pathway to produce lactic acid; between meals, they utilize their energy reserves and produce formic and acetic acid instead.

**Conclusion**

Dental caries is a multifactorial, infectious disease affecting a significant percentage of the population. It is more accurate to consider caries as caused, not by an infectious agent, but by a shift in oral microflora to caries-causing types in response to acidity resulting from metabolism of sugars. The development of caries is dependent on the interaction of four primary factors. These are a host (tooth surface), a substrate (food), the presence of oral bacteria, and time. Caries will not develop if any of these four primary factors are not present. Understanding the etiology and pathways of progression of dental caries will enable the profession to strive toward early intervention and, hopefully, prevention (Figures 7-11).

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![Diagram of Glycolytic Pathway](image-url)

**Figure 6.** Glycolytic pathway of *Streptococcus mutans*, from monosaccharides to acid. Adapted from: Marsh PD, Martin MV. Oral Microbiology. 5th ed. Edinburgh: Churchill Livingstone Elsevier; 2009.
Figure 7. Caries Lesion Initiation and Progression Pellicle Formation.

Figure 8. Caries Lesion Initiation and Progression Biofilm Formation.
Figure 9. Caries Lesion Initiation and Progression Dietary Sugars Diffuse into the Biofilm.

Figure 10. Caries Lesion Initiation and Progression Fermentation Produces Acid Leading to Demineralization.
Figure 11. Caries Lesion Initiation and Progression Demineralization and Remineralization.

Video 3: Caries Lesion Initiation and Progression (Animation)
Click here to view this video on dentalcare.com.
WHAT IS TOOTH DECAY?

Tooth decay happens when acids wear away the tooth’s hard surface layer, called enamel. These acids are made by a sticky film called plaque. Plaque has germs that feed on sugary foods. The process of digesting these sugars makes acids that attack tooth surfaces.

Over time, tooth decay can cause holes in the tooth surface. These are called cavities. If left untreated, cavities can get bigger. They can even destroy the tooth.

If you think you have a cavity, see your dental team. Your dentist is likely to put in a filling. Fillings may stop the cavity from getting bigger.

Acids constantly attack your tooth surfaces, but tooth decay doesn’t happen all at once. That’s because other elements in your mouth work to strengthen your teeth and stop the tooth decay process. One of these elements is saliva. Saliva has minerals that help strengthen tooth surfaces. Fluoride, a natural mineral that is often added to water and found in toothpaste, also helps to make teeth stronger.

**4 stages of tooth decay**

**Stage 1**
The dull spot on the tooth’s surface may be decay. Brushing with a fluoride toothpaste and flossing may prevent it from becoming a cavity.

**Stage 2**
The decay is now a cavity. It has gone through the tooth’s hard surface layer.

**Stage 3**
Now that the cavity has reached the softer layer of the tooth, it will get bigger faster.

**Stage 4**
If the cavity is not filled, it can cause bigger problems deeper in the tooth. This is why it’s important to see your dental team regularly.

Dentists check for tooth decay and cavities

If you do have tooth decay, your dental team may talk to you about fillings, fluoride, or other treatment choices.
Here are some tips to help prevent tooth decay:

Don’t eat a lot of sugary foods.
Cut down on snacks between meals.
• This will help prevent plaque from making acid.
  It will also reduce the number of times your teeth are exposed to acids.

Eat a diet high in calcium.
• Calcium helps strengthen tooth surfaces.

Drink plenty of water, especially if you take certain medicines.
• Some medicines can decrease the amount of saliva your body makes. This may put you at greater risk for tooth decay.

Use a toothpaste and mouthwash with fluoride.
• Fluoride helps make tooth surfaces harder and stronger.

Visit your dental team at least twice a year.
• They will clean your teeth and check for cavities.

Quick facts about tooth decay

• Tooth decay happens when acids wear away the tooth’s hard surface layer.

• Tooth decay can cause holes in your teeth. These are called cavities.

• Tooth decay can be avoided by brushing twice a day with a fluoride toothpaste and flossing between teeth.

• Toothpastes and mouthwash with fluoride can also help strengthen teeth and help fight tooth decay.

For more tips on how to prevent tooth decay, talk to your dental team or visit oralb.com.
PLAQUE AND TARTAR CONTROL

Patients often confuse plaque and tartar and how they are related to each other

Plaque is a sticky, colorless deposit of bacteria that is constantly forming on the tooth surface. Saliva, food, and fluids combine to produce these deposits that collect on teeth and where teeth and gums meet.

The buildup of plaque can trap stains on the teeth, and it is also the primary factor in gum disease. Fighting plaque is a life-long part of good oral care.

Plaque begins forming on teeth 4 to 12 hours after brushing, which is why it is so important to brush at least twice a day and floss daily.

Tartar, also called calculus, is a crusty deposit that can trap stains on the teeth and cause discoloration. It creates a strong bond that can only be removed by a dental professional. Tartar formation may also make it more difficult to remove new plaque and bacteria.

Individuals vary greatly in their susceptibility to plaque and tartar. For many of us, these deposits build up faster as we age.

The photographs show the degrees of tartar (or calculus) formation.

There are many stages and forms of periodontal disease, including:

Understanding Calculus

Calcium and phosphate bind to form crystals on the teeth. These calcium phosphate crystals eventually harden within plaque, forming calculus. Certain types of chemicals called pyrophosphates help to decrease calculus buildup by stopping the growth of crystals on the tooth surface and preventing new crystals from forming.
You can help prevent the buildup of tartar by:

- Having your teeth cleaned professionally every 6 months, or more frequently as recommended by your dentist or hygienist

- Brushing with a toothpaste that contains pyrophosphate, such as Crest® Tartar Protection, which adheres to the tooth surface and inhibits the formation or growth of calculus crystals

- Brushing with Crest® Pro-Health or Crest® 3D White Advanced Vivid, which contain sodium hexametaphosphate, a pyrophosphate specially formulated to not only inhibit calculus, but also loosen and break the bonds of extrinsic stains for powerful whitening and a protective barrier to prevent future stains

For more tips on plaque and tartar control, talk to your dental team or visit oralb.com.
Course Test Preview
To receive Continuing Education credit for this course, you must complete the online test. Please go to:

1. Which of the following best describes the etiology of caries? Caries is ____________.
   a. an infectious disease caused by oral bacteria
   b. caused when acidic byproducts of oral bacteria come into contact with tooth enamel
   c. a disease caused by snacking frequently and not brushing the teeth
   d. entirely preventable

2. At what pH does tooth enamel begin to demineralize?
   a. 8.3
   b. 7.5
   c. 5.5
   d. 3.2

3. Which researcher(s) first suggested an association between acid production and caries?
   a. Miles and Underwood (1881)
   b. Miller (1889)
   c. Viperholm (1945-1954)
   d. Orland and Keyes (1954)

4. Which factors play an essential role in caries development?
   a. A food substrate
   b. Oral bacteria
   c. Time
   d. All of the above.

5. Which trio of factors listed below increases the risk of caries?
   a. Eating frequently, high proportion of acidogenic bacteria, lower fluoride levels.
   b. Brushing only once daily, eating often, high flow of saliva.
   c. Eating apples, higher fluoride levels, do not brush teeth in the evening.
   d. Snacking between meals, high counts of oral streptococci, using a toothpick after eating.

6. Which trio of factors listed below reduces the risk of caries development?
   a. Presence of more bacteria that thrive in very acidic conditions, using a toothpick to remove food particles, having adult (permanent teeth).
   b. Presence of bacteria that do not thrive in very acid conditions, infrequent snacking, and little consumption of sucrose.
   c. High presence of acidogenic bacteria, high saliva flow rate, infrequent snacking.
   d. All of the above.

7. Which of the following best describes biofilm?
   a. It is composed mostly of extracellular polysaccharides.
   b. It can develop on shedding surfaces.
   c. Bacterial cells join it only by sticking to the tooth surface.
   d. All of the above.

8. Which bacteria are linked to caries development?
   a. *S. mutans* and *S. oralis*
   b. *S. mutans* and *Lactobacilli*
   c. *S. sanguinis* and *S. mutans*
   d. All of the above.
9. Which of the following best describes *S. mutans*?
   a. The first colonizer to form biofilm.
   b. Present in all humans.
   c. The strain of bacteria most strongly implicated in acid production and caries.
   d. Does not produce acids.

10. In the late maturation phase, biofilm is ____________.
    a. homogenous
    b. two-dimensional
    c. made up of several microenvironments
    d. characterized by increase rates of cell division

11. Which of the following is not true about biofilm?
    a. In the late maturation stage of development there is no turnover of cells.
    b. Biofilm always forms on the acquired pellicle.
    c. Bacteria can become detached from the biofilm in order to spread to new surfaces of the oral cavity.
    d. Biofilm is a microbial system.

12. Which of the following describes the plaque ecosystem?
    a. It contains no known species of bacteria.
    b. It contains only one species of bacteria.
    c. Once established, it cannot be removed.
    d. The plaque ecosystem can influence its environment, and the environment can influence the plaque ecosystem.

13. According to the ecological plaque hypothesis:
    a. A neutral pH is linked to proliferation of *S. mutans* in plaque and demineralization.
    b. Sugar drives the shift in plaque ecology that leads to caries.
    c. A neutral pH is linked to proliferation of *S. sanguinis* and remineralization.
    d. A pH of 5.5 can destabilize plaque ecology, leading to demineralization.

14. Which of the following is not a mechanism of sucrose metabolism?
    a. Enzymes in saliva cleave sucrose polysaccharides.
    b. Glucose is polymerized by glycosyltransferases.
    c. Fructose is polymerized by fructosyltransferases.
    d. Sucrose is transported across the cell membrane and cleaved by extracellular invertase.

15. Which of the following is not true about glycolysis:
    a. It an energy-producing mechanism.
    b. It is an acid-producing mechanism.
    c. All bacteria use glycolysis to break down sugars.
    d. It produces only lactic acid.
References

About the Author

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Dr. Higham is a Professor of Oral Biology, Department of Health Services Research and School of Dentistry, University of Liverpool, United Kingdom and is Director of postgraduate research in her University Research Institute.

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Dr. Higham has supervised 24 Doctoral students and 10 Master’s degree candidates and has published more than 300 book chapters, peer-reviewed papers, and peer-reviewed abstracts. Her main research interests are in the use of in vitro and in situ models and clinical trials to study dental diseases, together with the development of optical technologies for the quantification of mineral loss/gain in vivo. She has been involved in University teaching at all undergraduate and postgraduate levels for over 30 years. Dr Higham is a scientific advisor for the European Organisation for Caries Research (ORCA) and is a dentistry panel member for the Research Excellence Framework (REF 2014) in the UK.

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