Caries Process, Prevention, and Management: The Agent

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Conflict of Interest Disclosure Statement
- Dr. Apoena Aguiar Ribeiro reports no conflicts of interest associated with this course. She has no relevant financial relationships to disclose.

Caries Process, Prevention and Management consists of a series of ten continuing education courses that may be taken individually or as a complete series. This 2025 newly updated series was first developed in conjunction with the American Dental Education Association in 2008 and has now been comprehensively revised by authors who are members of the board of directors of the American Academy of Cariology (AAC) to reflect the current knowledge and best practices and quality depth of knowledge on the caries process, prevention, and management. Procter & Gamble is delighted to provide this resource enabling high quality educational experiences and an up-to-date understanding of both the science and its relevance to all clinicians in practice and we thank the authors for their contributions. Click *here* to learn more about the AAC.

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Overview

This course is part 2 of a 10-part series entitled *Caries Process, Prevention, and Management.* 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.

Learning Objectives

Upon the 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.

Introduction

Dental caries is a very complex disease. It is arguably the most prevalent chronic disease in humans, 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 42% of children between the ages of 2 and 11 having had caries in primary teeth and 23% of children in this same age group having untreated dental caries.[1] Among dentate adults aged 20 to 64, nearly 90% have caries in permanent teeth.[2] Commonly termed "tooth decay," dental caries can be defined as a biofilm-mediated dysbiosis that involves changes in the microbiome composition and function, which leads to the dissolution of tooth tissues (enamel and dentin) by acid produced by select oral bacteria, as a result of the fermentation of dietary carbohydrate.[3] Clinically, it is observed the localized destruction of tooth tissues over time. 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 Snapshot

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 over time can result 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, it is possible for the tooth to be repaired by the action of saliva, which contains buffers that will neutralize the acids produced by bacteria and replace 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 dental plaque as possible.

The decay process starts with the appearance of a white spot on the surface of the tooth in areas that accumulates dental plaque, i.e., close to the gum line and on the occlusal surfaces. Once sufficient mineral has been lost, the white spot turns into a cavity and it is more difficult to control the lesion progression, frequently requiring the repair of the cavity by the dentist to restore tooth function.

In most cases, caries can be controlled by good oral hygiene practices, focusing on biofilm mechanical control (toothbrushing), since the disease is caused by dysbiosis in the biofilm lead by frequent exposure to fermentable carbohydrates, especially between meals.[4] Therefore, the most important action 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. The frequency of intake of sugars should be reduced as much as possible, and, ideally, limited to mealtimes, so that acids in biofilm 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. In addition, the presence of fluoride in toothpaste makes enamel more resistant to acid dissolution and encourages the process of remineralization.

Glossary

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

aciduric – Bacteria 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 is 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.

dysbiosis - an imbalance in the composition and function of the microbial communities, occurring when there is a disruption in the normal balance of beneficial and harmful microorganisms, leading to changes in the overall health and well-being of the host.

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, and adequate buffering. In the presence of fluoride, remineralization is enhanced.

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.

Dental Caries - Historic Perspective

Theories about what cause cavities go as far back as 2500 BC in ancient China when it was thought that “tooth-worms” caused cavities.5 This belief continued for several centuries in many different cultures. Later, in 350 BC, Aristotle and others acknowledged that sweets and figs caused decay.6 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.[7]

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.[8] 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.[8,9]

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. In a 2018 executive summary on fluoridation facts developed by the National Fluoridation Advisory Committee (NFAC) and the American Dental Association (ADA) Council on Advocacy for Access and Prevention (CAAP) noted: "Fluoridation of community water supplies is the single most effective public health measure to prevent tooth decay." In addition, "throughout more than 70 years of research and practical experience, the overwhelming weight of credible scientific evidence has consistently indicated that fluoridation of community water supplies is safe."[10]

Dental Caries

Dental caries is a **biofilm**-mediated 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

Figure 1. 1955, Procter & Gamble, Crest® toothpaste

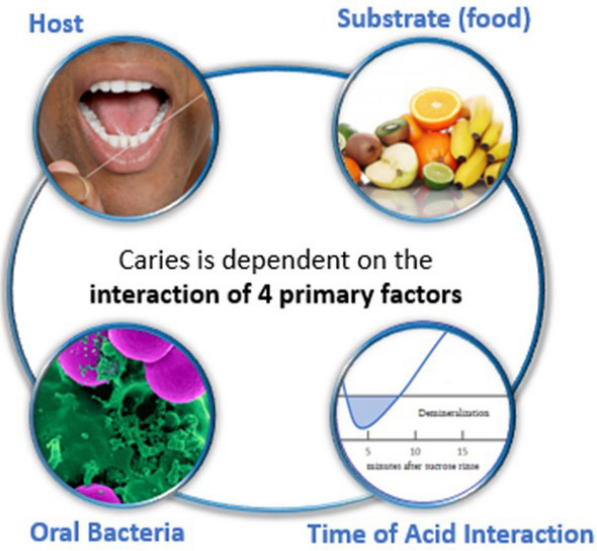

Figure 2. Primary factors of dental caries.

enamel–essentially dissolution of tooth structure–begins to occur. The early stages are reversible, because the natural process of **remineralization** can replace lost mineral from enamel crystals. However, if demineralization continues over time, enough mineral content may be lost so that the soft organic matrix left behind disintegrates, forming a cavity.

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 (Figure 2). Caries will not develop if any of these four primary factors are not present.

Each of the four primary factors can be further divided into secondary (yellow circle) and tertiary factors (purple circle) that also influence the likelihood of caries (Figure 3).

Host (tooth surface): The sub-factors that influence caries development are **age** (the

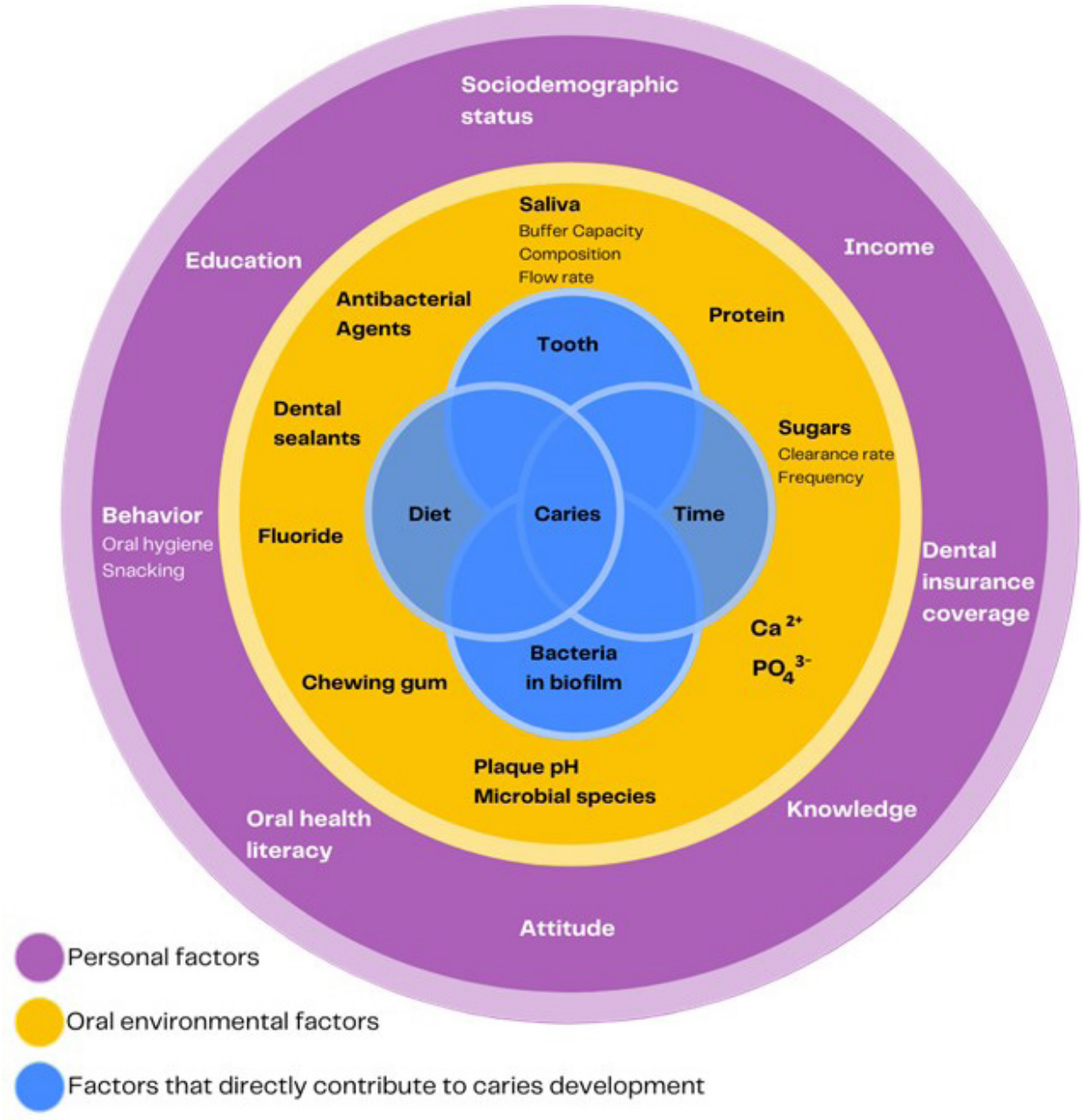

Figure 3. The factors and sub-factors that influence caries development.
Adapted from: Selwitz RH, Ismail AL, Pitts NB. Dental caries. Lancet. 2007;369:51-59.

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), enamel defects, **root surface exposure** due to gum recession, nutrition (if tooth-strengthening nutrients are consumed).

A tooth is more susceptible to caries if it has less acid resistant enamel due to age, low fluoride intake, hypomineralization, 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. 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 and a diet of tooth-strengthening nutrients is consumed. Enamel defects can be caused by genetics, environmental factors, or developmental issues, and these defects may result in uneven surfaces or pitting allowing plaque and bacteria to build up and increase the risk of cavities.

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 aciduricity** (how well plaque can survive in acidic conditions), **oral hygiene** (how often the microbial load is reduced by brushing or **prophylaxis**), and if **fluoride is present in plaque**.

The likelihood of caries development is higher when the 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

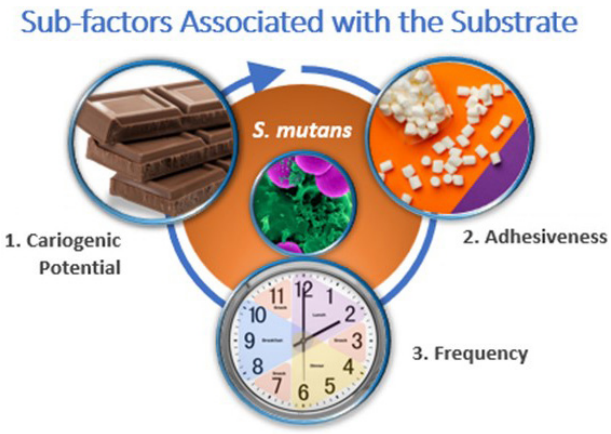

Figure 4. Sub-factors Associated with the Substrate (food)

highly acidic conditions, plaque acid production is low, and/or plaque is regularly removed by brushing or flossing.

Substrate (food): The sub-factors that influence caries development are **oral clearance** (if food is retained or not in the mouth after eating), **eating frequency, food detergency** (if consumed food can clean teeth), **consumption of 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. Certain foods can help protect against dental caries by neutralizing acids in the mouth, such as cheese, yogurt, and milk, which are high in calcium and phosphate, which help remineralize and strengthen tooth enamel; vegetables like spinach, kale, and other leafy greens promote the production of saliva, which helps neutralize acids; and green tea contains compounds called catechins, which have antimicrobial properties.

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 white-spot 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

Bacteria collect on specific sites of the teeth and along the edge of the gums in a cream-colored mass called plaque (Figure 5). 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 a 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.[11-13] The extracellular polysaccharides also form a hydrophobic layer that helps bacteria in biofilm to evade antibodies and protect the microorganisms from antibiotics, fluorides, and antimicrobials.

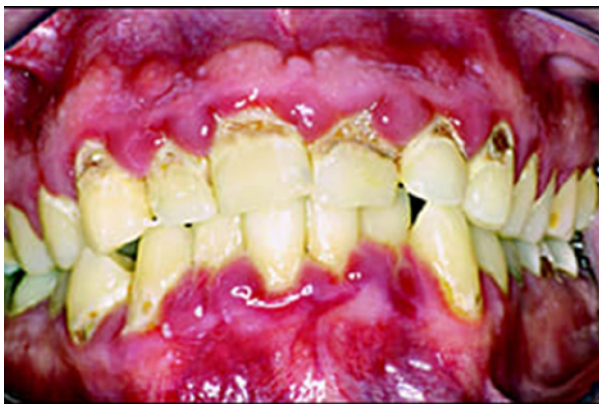

Figure 5. Dental Plaque Deposits.
From: V. Kim Kutsch, DDS. Originally published in Inside Dentistry, 2009;5(5):60-65.

Microbiology

The bacterial microbiome from dental biofilms can harbor more than 720 unique species. However, 800 to 1000 different oral bacterial taxa can be identified with the more modern techniques with differences in abundance and diversity patterns across age, sample quality and origin, and health status.[14]

Traditionally, *Streptococcus mutans* and *Lactobacillus acidophilus* have been the most studied bacterial species due to its virulent factors associated with caries development and progression. However, the latest evidence shows that, in addition to *Streptococcus mutans*, other bacteria are linked to the onset and progression of caries, including species such as *Actinomyces* and *Bifidobacterium*, *Streptococcus salivarius*, *Streptococcus sobrinus*, *Streptococcus parasanguinis*, *Scardovia wiggsiae, Slackia exigua, Lactobacillus salivarius, Parascardovia denticolens, and* various species of *Porphyromonas and Veillonella.*[15-19]

This multi-specie cariogenic biofilm is usually made up of bacterial species that possess the following key characteristics: (1) the ability to adhere to the saliva-coated tooth surface, (2) the production of an exopolysaccharide (EPS)-rich matrix, which limits the spread of acidic by-products from carbohydrate fermentation, and (3) the ability to survive in an acidic environment. These traits ultimately lead to enamel dissolution due to the localized acidic microenvironments within the biofilm and at the tooth-biofilm interface.

Stages of Development

Traditionally, plaque biofilm development was described in six stages:

- Stage 1: Formation of an acellular layer called the acquired **pellicle**, composed of **glycoproteins**, **phosphoproteins**, and **lipids**.
- **Stage 2: Initial attachment**, when free-floating early colonizers of the teeth, such as Streptococcus sanguinis form an initial attachment to the pellicle by weak and reversible van der Waals forces.
- **Stage 3: Irreversible attachment**, where organisms that were unable to attach to the pellicle begin to adhere to the first layer of

colonizers with irreversible attachments via specific adhesion-receptor interactions and form microcolonies embedded in an extracellular matrix.

- **Stage 4: Early Maturation** (also called Maturation I), with early colonizers becoming established.
- **Stage 5: Late Maturation** (also called Maturation II), a thick, three-dimensional layer of dental plaque biofilm formed.
- **Stage 6: Dispersion**, when enzymes that degrade the biofilm (such as dispersin B) allow some bacteria to detach themselves from the biofilm and travel to new sites, to start the cicle again.[20]

However, latest technology has allowed the observation that 90% of the microbes in saliva can be found as multicellular structures, instead of free living bacterial cells (planktonic; fluid phase). Bacteria are also found coadhered with human epithelial cells, forming a highly complex and structurally diverse community. Notably, it is now understood that (Figure 6):

- **Stage 1 - Pellicle formation:** is characterized by the formation of an acellular layer called enamel pellicle formation.
- **Stage 2 - Attachment:** this stage is marked by both aggregates and single cells can bind to the tooth surface (solid phase), and these microbial aggregates in saliva harbor different bacterial taxa, including those traditionally considered early and late colonizers. Thus, the polymicrobial community can adhere to the surface collectively as structured colonizing units without an orderly attachment in a sequential fashion.
- **Stage 3 - Biofilm initiation and development:** Actively growing aggregates expand tridimensionally, often engulfing the single cells and merging with other active growers to build the oral biofilm, forming spatially and compositionally heterogeneous superstructures but with reduced microbial diversity.
- **Stage 4 – Dispersion:** when microbial aggregates detach from the biofilm and a new cycle starts.[21]

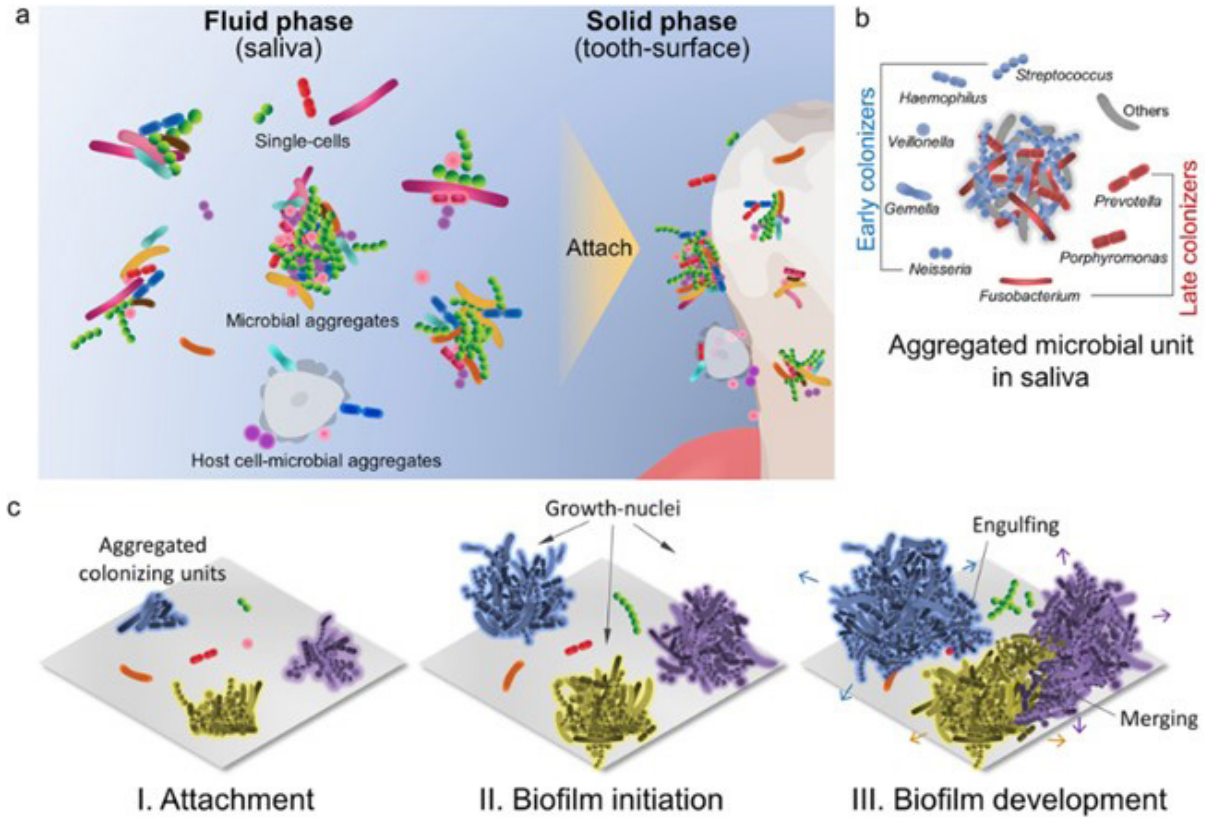

Figure 6. A) Saliva is dominated by polymicrobial aggregates harboring different species together in addition to free-living bacterial cells. Bacteria are also found coadhered with human epithelial cells. **B)** Bacteria that traditionally have been considered early and late colonizers are found together in the adhering aggregates and in early formed human plaque samples, indicating that they bind concomitantly to the surface as a colonizing unit. **C)** Microbial aggregates attached to the enamel pellicle act as growth nuclei that grow more actively and faster than the attached single cells, which stay mostly static. Actively growing aggregates expand tridimensionally, often engulfing the static cells and merging with active growers, forming superstructures.

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 and between different sites within the mouth. Classical microbiological techniques have estimated that plaque contains 800 distinct oral species, with a healthy individual possessing 50 to 100 different species at any one time.[12,19,22] 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).[22] 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.[19,23,24] On the other hand, when pH remains neutral, acid-sensitive bacteria like *S. sanguinis* can survive, keeping acid production low and increasing remineralization.[25] 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."[12,19,24]

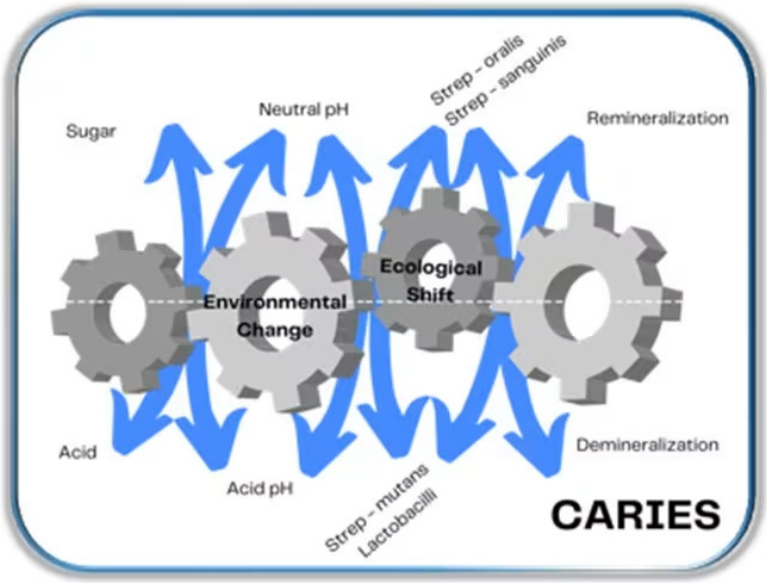

Figure 7. Ecological Plaque Hypothesis.
Adapted from: Marsh, PD. Microbial Ecology of Dental Plaque and its Significance in Health and Diseases. Adv Dent Res. 1994:8(2):263-271.

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, Lactobacilli* and other aciduric bacteria 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 7).

Oral Bacteria

Sugar Metabolism

Dietary sugars, starches, and fermentable carbohydrates (usually collectively referred to 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**. Sucrose is the specific substrate for the bacterial enzymes, glucosyltransferases, that convert the sucrose to water insoluble extracellular glucose polymers, glucans. There are five main mechanisms by which oral Streptococci hydrolyze (break down) sucrose (Figure 8).

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 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.

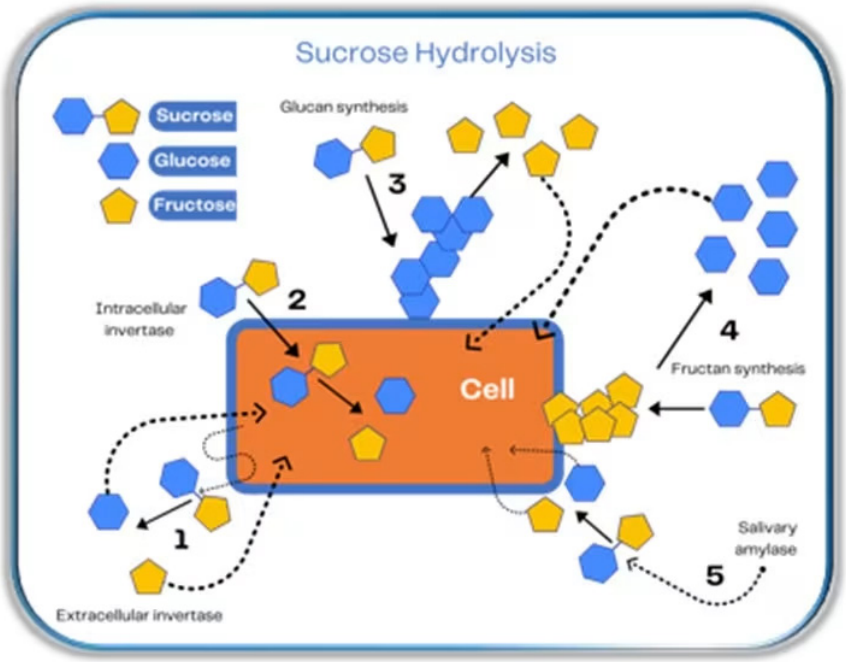

Figure 8. Mechanisms of sucrose hydrolysis by oral bacteria.
Adapted from: Marsh PD, Lewis MAO, Rogers H, et al. Oral Microbiology. 6th ed. 2016; Edinburgh: Churchill Livingstone Elsevier.

Among the full range of fermentable carbohydrates, sucrose is the most cariogenic. It is a relatively small molecule that can penetrate the biofilm, and the bacteria use sucrose for energy production through fermentation, which produces acid. During the process, the bacteria can use sucrose to make a key carbohydrate called dextrins.

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 9). 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 disease affecting a significant percentage of the population. It is a dysbiosis, meaning that it is caused by a shift in oral commensal microflora to a selection of caries-causing types in response to acidity resulting from the 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.

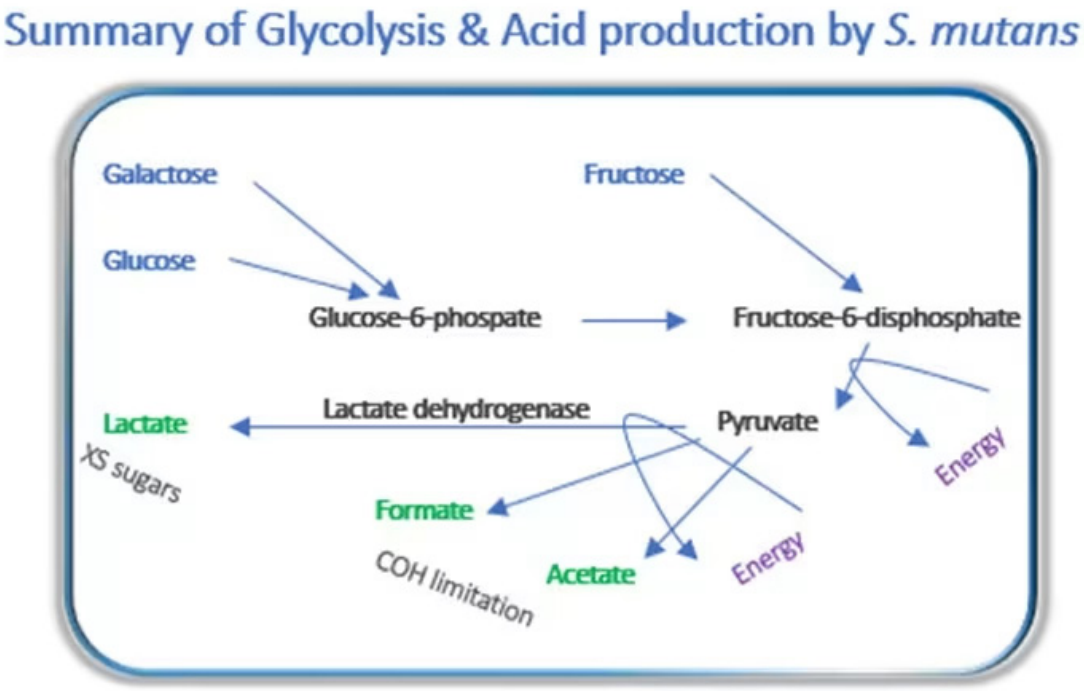

Figure 9. Glycolytic pathway of Streptococcus mutans, from monosaccharides to acid. *Adapted from: Marsh PD, Lewis MAO, Rogers H, et al. Oral Microbiology. 6th ed. 2016; Edinburgh: Churchill Livingstone Elsevier.*

Course Test Preview

To receive Continuing Education credit for this course, you must complete the online test. Please go to: *www.dentalcare.com/en-us/ce-courses/ce711/start-test*

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. Dietary sucrose is associated with the initiation of enamel surface caries. This sucrose dependence is attributed to which of the following.

A. Sucrose is the only sugar that the mutans streptococci are capable of fermenting to lactic acid.
B. Sucrose inhibits the non-cariogenic bacteria in plaque.
C. Sucrose is the specific substrate for the bacterial enzymes, glucosyltransferases, that convert the sucrose to water insoluble extracellular glucose polymers, glucans.
D. Sucrose is the only sugar that is converted to intracellular polysaccharide that promotes sustained acid production.

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 traditionally 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 specie 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. The initial colonizers that attach to the enamel pellicle are single, planktonic cells.
B. Biofilm always forms on the acquired pellicle.
C. Bacterial complexes 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.

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Additional Resources

- No Additional Resources Available

About the Author

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Apoena De Aguiar Ribeiro is a Pediatric Dentist and Microbiologist. She was born in Rio de Janeiro, Brazil. Among her 25 years of teaching, Ribeiro most recently served as a tenured associate professor in the Fluminense Federal University, Department of Pediatric Dentistry and Cariology for 12 years, with a joint appointment at the Department of Microbiology for 5 years, in Rio de Janeiro State, Brazil. Prior to that role, she was an associate professor in the Grande Rio University Department of Pediatric Dentistry and Cariology for 8 years. During this time, she has also maintained a part-time role in a solo dental practice for 22 years, devoted to provide oral health to infants, children, adolescents and children with special needs. She is the author of a full book of Pediatric Dentistry (Pediatric Dentistry – A contemporary approach, GEN Editor, Brazil) and has published more than 50 papers in peer-reviewed journals. Dr. Ribeiro had successfully obtained 5 grants as a Principal Investigator in Brazil.

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