Caries Process and Prevention Strategies: The Agent

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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.

Introduction
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 authors report no conflicts of interest associated with this course.

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Course Contents
- Overview
- Learning Objectives
- Glossary
- Introduction
- Dental Caries
  - A Multi Factorial Disease
- Dental Plaque
  - Biofilm
  - Microbiology
  - Stages of Development
  - Ecology in Health and Disease
- Oral Bacteria
  - Sugar Metabolism
  - Acid Production
- Conclusion
- Course Test
- References
- About the Author

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 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. Among dentate adults aged 20 to 64, 91% 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 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.

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).
an aerobic – 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.

**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.

**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 “tooth-worms” 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. In its statement commemorating the 60th anniversary of community water fluoridation, the ADA noted: “Studies conducted throughout the past 60 years have consistently indicated that fluoridation of community water supplies is safe and effective in preventing dental decay in both children and adults. It is the most efficient way to prevent one of the most common childhood diseases – tooth decay (5 times as common as asthma and 7 times as common as hay fever in 5- to 17-yearolds).”

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.

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.

![Figure 1](https://via.placeholder.com/150)

**Figure 1.** The factors and sub-factors that influence caries development. Adapted from: Selwitz RH, Ismail AL, Pitts NB. Dental caries. Lancet. 2007;369:51-59.
(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 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 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 highly acidic 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 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 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 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.\textsuperscript{9-11}

**Microbiology**

All oral bacteria produce acidic byproducts when they metabolize sugar, and this causes a drop in plaque pH. Although there are numerous bacteria present in plaque, 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%).\textsuperscript{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.\textsuperscript{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 dextransucrase. (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-deminalizing 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.

**Stage 3: Irreversible attachment.** 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. The
bacteria replicate and form microcolonies embedded in an extracellular matrix.

**Stage 4: Early Maturation (also called Maturation I).** As a result of the previous steps in which bacteria form attachments, early colonizers become established. This leads to increased dental plaque complexity due to allogenic factors, such as oxygen consumption within plaque creating anaerobic zones, food chains becoming established, and an increased range of receptor sites for bacterial attachments. Cell division and recruitment of new bacteria also allows the bacterial population to increase.

**Stage 5: Late Maturation (also called Maturation II).** In this stage, microbial diversity continues to increase, while rates of cell 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 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. 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 *Lactobacilli* 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 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 hydrolyze (break down) sucrose (Figure 5).

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.

**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 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 (Figures 7-11).
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 on image to view video online.
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/professional-education/ce-courses/ce369/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. **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 Authors

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Dr. Higham is a Professor of Oral Biology in the Department of Health Services Research and School of Dentistry, University of Liverpool, United Kingdom. She is Director of Postgraduate Research in her University Research Institute and is the National Institute of Health Research, Comprehensive Research Network Oral and Dental Specialty Research Group Lead for North West Coast and National Industry Lead.

Dr. Higham has a background in microbiology and biochemistry, a PhD focused on dental plaque metabolism from the University of Liverpool, Chartered Biologist status and a member of the Royal Society of Biology. She was appointed as a Research Fellow in the Department of Clinical Dental Sciences at the University of Liverpool and later promoted to Senior Lecturer and then to Professor.

Dr. Higham has supervised more than 40 postgraduate students and has published more than 370 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 has been a scientific advisor for the European organization for caries research (ORCA) for many years and is a dentistry panel member for the Research Excellence Framework (REF) in the UK. She has been elected as Vice-President of the Cariology Group of the International Association of Dental Research (IADR) and will serve as President in 2020.

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Chris Hope, BSc (Hons), PhD, FHEA
Chris graduated with a degree in Microbiology at the University of Liverpool in 1994 and then went on to study for a PhD in Chemical Engineering at The University of Birmingham. This somewhat unconventional entry into dental research came via biofilm modeling which led to his appointment at the Eastman Dental Institute – University College London as a research fellow between 2000 and 2005.

In 2005, Chris was appointed as Lecturer in Oral Biology at the University of Liverpool where his experience of biofilm modeling complimented the research group themes of caries and plaque-related disease. Chris developed a biological model of dental caries which acquires enamel lesions in less than two weeks and continued his interests in imaging by studying the natural fluorescence of dental plaque. More recently, he has revisited photodynamic therapy by looking at the lethal photosensitization of periodontal pathogens by means of their intrinsic porphyrins.

Chris served on the British Society for Oral and Dental Research (BSODR) Oral Microbiology and Immunology Group (OMIG) management committee from 2008 to 2011 and then again for a second term from 2015 to 2018. Chris was also elected onto the management board of the BSODR in 2017. He is also on the editorial board of the Journal of Medical Microbiology.

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Sabeel Valappil, BSc, MSc, PhD, PGCertEd, FHEA
Dr. Valappil is a lecturer in dental sciences in the School of Dentistry at the University of Liverpool, United Kingdom. He is a deputy Director of Postgraduate Research in his University Research Institute. Dr. Valappil is a microbiologist with special interests in bacteriology and biomaterials. Following his PhD, Dr. Valappil worked at Imperial College London and the University of Westminster on developing tissue engineering composites. He then worked on controlled antibacterial agent delivery systems and bacterial biofilms at Eastman Dental Institute, University College London. Since moving to Liverpool, Dr. Valappil focused his research in the development of novel antibacterial materials for dental applications in treating periodontitis and caries. Dr. Valappil has published over 100 book chapters, peer-reviewed papers and peer reviewed abstracts. Dr. Valappil is an associate editor of BMC Oral Health and Review Editorial Board Member of the journal Frontiers in Antimicrobials, Resistance and Chemotherapy. He is a peer reviewer for over 40 scientific journals and act as grant reviewer for national and international research councils including Medical Research Council, UK; Chilean Science Agency, CONICYT and Italian Cystic Fibrosis Research Foundation.

Dr. Valappil has been involved in University teaching at all undergraduate and postgraduate levels for over 10 years and so far, supervised 20 undergraduate and postgraduate project students. Dr. Valappil was the chair of the Dental Biomaterials session for the European Society for Biomaterials 2014 conference and chair of the Microbiology session for the European organisation for caries research 2016 conference.

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Phil Smith, BDS, MDS, PhD, FDS, DRD, MRD, FDS (Rest Dent) RCS (Edin), FHEA
Phil is currently Senior Lecturer and Honorary Consultant in Restorative Dentistry at Liverpool University Dental Hospital and he has been an NHS Consultant since 1998. He has been actively involved in teaching, research and clinical service, and is lead clinician for restorative care of CLP patients in Liverpool and North West (West) Region. He has also gained experience in managing clefts from time spent at Oslo. He has published widely including authoring/co-author of 3 textbooks and has been supervisor, mentor and advisor for a number of postgraduate students and trainees. He is a reviewer for Journal of Dental Research, Journal of Dentistry, British Dental Journal, Dental Materials, Journal of The European Journal of Prosthodontics and Restorative Dentistry, and Dental Update. He is also part of a team from Liverpool that has been commended in the recent Medical Futures Innovation awards and is currently President of the British Society of Prosthodontics.

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