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DACE DENTAL ACADEMY of CONTINUING EDUCATION

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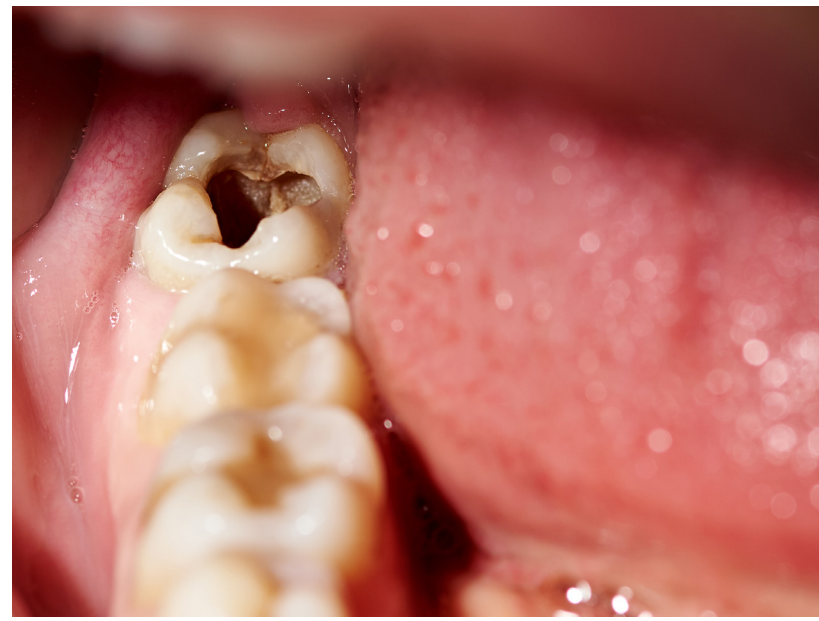
ABSTRACT

Dental caries continues to be the most common transmissible bacterial disease in the world, regardless of age, race, ethnicity, socioeconomic status, geographic location, or any other parameters. Many courses focus on minimally invasive dentistry, including the use of silver diamine fluoride and bioactive restorative materials, which are huge advancements in the preservation and restoration of tooth structure. As dental professionals, it is imperative that we help our patients preserve their healthy natural teeth from one of the most insidious diseases of all time. This course will focus on the primary factors that lead to dental caries disease and will discuss a variety of interventions the dental practitioner can share with patients to prevent and potentially reverse dental caries.

EDUCATIONAL OBJECTIVES

Upon completion of this course, the dental professional will be able to:

1. Recognize the history and extent of dental caries disease
2. Describe the role of pH, saliva, biofilm, diet, and genetics in the dental caries disease process
3. Clarify the role of the Stephan Curve in caries-risk education
4. Develop self-care recommendations based on the current science of the dental caries disease process



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Dental caries: A new protocol for an age-old disease

A PEER-REVIEWED ARTICLE

by Kathryn Gilliam, BA, RDH, MAAOSH, HIAOMT

Dental caries continues to be the most common transmissible polymicrobial disease in the world, regardless of age, race, ethnicity, socioeconomic status, geographic location, or any other parameters.[1] According to the National Institute of Dental and Craniofacial Research, 92% of adults aged 20 to 64 have had dental caries in their permanent teeth.[2] Additionally, 59% of adolescents aged 12 to 19 have had dental caries in their permanent teeth.[3] And perhaps most distressing of all, 42% of children aged 2 to 11 have had dental caries in their primary teeth.[4] Despite the fact that annual dental and oral care expenditures in 2020 reached $142.5 billion, the majority of that money was spent repairing the damage caused by dental caries disease rather than preventing the disease.[5] The prevention of dental caries disease in and of itself is vitally important to health, and now that the cariogenic bacteria, *Streptococcus mutans*, is recognized as an independent risk factor for cardiovascular disease, it is even more critical.[6]

History of dental caries

Dental caries is one of the oldest and most prevalent diseases in humans.[7] The term "dental caries" originates from the Latin word "caries," which means "decay."[8] As long ago as 5000 BC, the Sumerians believed that dental caries were caused by a "tooth worm" that bored into the teeth.[8] In 350 BC, Aristotle noted that sugar and figs caused tooth decay.[9] By the 12th century, the condition of having holes in the teeth was described as caries or cavities.[10] The treatment prescribed was to fill the holes with various compounds such as tobacco ash and other home remedies.[10]

In the 1700s, French physician Pierre Fauchard wrote the first text on dental disease and treatment.[10] He rejected the tooth worm theory and postulated that dental caries were caused by enamel erosion.[10] Fauchard recommended excavation of the cavity and filling it with gold foil, tin, or lead.[10]

In the 1890s, the first oral microbiologist, W. D. Miller, published the chemico-parasitic theory. He postulated that the cause of demineralization of tooth enamel, the first step in dental caries, was bacteria that metabolize fermentable carbohydrates and produce acid.[11] Miller thought that no single bacterium was the cause of dental caries. His focus was on the fermentable carbohydrates that the bacteria consumed. It wasn't until 1954 that researchers were able to prove that it was bacteria, primarily *Streptococcus mutans*, not sugar, that was the main culprit in acid production and demineralization of tooth structures.[11]

Many plaque hypotheses have been proposed to explain the etiology of dental caries. In the 1880s, the nonspecific plaque hypothesis postulated that the quantity of plaque caused dental caries, not the specific bacterial species within the plaque.[10] This idea was commonly accepted and was developed by W. D. Miller and G. V. Black.[10] They believed that the best way to prevent disease was by mechanical plaque removal by toothbrushing or tooth-picking.[10] Once the technology to identify the specific bacteria in the plaque was developed, the nonspecific plaque hypothesis was discarded.[10] However, the idea that mechanical plaque removal is the best way to prevent dental infections persists today.

In 1976, the specific plaque hypothesis was advanced by Loesche.[10] He hypothesized that only a few specific pathogenic bacteria in the oral microbiome were responsible for causing dental caries, and in the absence of those bacteria, there would be no dental caries disease.[10] This hypothesis proposed that targeting specific bacteria such as *Streptococcus mutans* and lactobacilli with antibiotics was the way to cure dental caries.[12] Antibacterial treatment has shown only minor effectiveness, and once the treatment is stopped, the bacteria return to the pretreatment levels.[12] Treatment with antibiotics can lead to antibiotic resistance, as well, so it is not considered the ideal course of treatment today.[12]

In 1994, the ecological plaque hypothesis was developed by Marsh.[12] It combines the previous hypotheses and posits that dental caries is the result of a shift in the microbiota of the oral biofilm from benign bacterial species to cariogenic species.[12] Marsh stated that the environment changes because of the nutrients present and the pH, and that this environment is favorable to certain types of bacteria.[12] For example, the consumption of fermentable carbohydrates results in an acidic oral environment and selects bacteria that thrive in that type of environment and eliminates bacterial species that do not tolerate those acidic conditions.[12] Many acidogenic and aciduric bacteria, such as *Streptococci mutans*, nonmutans streptococci, lactobacilli, actinomyces, and bifidobacteria become dominant in the increasingly acidic oral environment.[12] The acidogenic-aciduric bacterial species *Streptococcus mutans* has long been thought to be the main culprit in dental caries; however, newer research has found that this species is not present in 10%–20% of patients with severe dental caries.[11] This points to the fact that there are other acidogenic-aciduric bacteria that cause dental caries.[11]

Dental caries is a multifactorial disease, affected not only by the bacteria present in the oral microflora, but also the diet, the saliva, the pH of the mouth, and the genetics of the individual.[11] There are many genetic factors that may contribute to caries resistance or risk, such as immune response, salivary protein-rich peptides, tooth morphology including pit and fissure anatomy, enamel composition and structure, taste preferences, and behavior.[13]

The caries process

To understand the process of dental caries, we must first recognize that enamel is the most highly mineralized tissue in the human body. Enamel is made up of minerals, including calcium and phosphate, which combine to form hydroxyapatite (HA) crystals.[14] Dental caries involves demineralization of these crystals, which results in cavitation of the tooth.[14]

Ultimately, it was discovered that for dental caries to occur, one must have a tooth, tooth-adherent bacteria, and fermentable carbohydrates, which the bacteria metabolize to produce acids.[7] It is these acids that demineralize the tooth structure over time, causing dental caries.[7] The oral microbiota changes from benign homeostasis to a cariogenic, acidogenic, and aciduric population as a result of frequent exposures to fermentable carbohydrates.[7] As bacteria metabolize fermentable carbohydrates, the end products are organic acids and lactic acid in particular.[7] The result of this acid production is a drop in pH, which leads to the demineralization of tooth enamel.[15]

Remineralization is the body's natural reconstruction mechanism in which the body uses calcium and phosphate ions to rebuild the tooth structure.[16] Recurring cycles of demineralization followed by remineralization followed again by demineralization make up the continuum of the dental caries process.[16] The end point of this process, if allowed to progress, is the deterioration of the tooth.[16]

One factor that has been discovered to be of paramount significance in dental caries is the pH of the plaque biofilm.[17] Low salivary pH drives the production of aciduric bacteria, which enables the acidogenic bacteria to proliferate, resulting in an oral microflora that is unfavorable to more protective nonacidogenic oral bacteria.[17] This further lowers the salivary pH, continuing the cycle.[17]

In the early 20th century, Dr. Robert Stephan postulated that there is a continuous change in salivary pH following the consumption of foods and beverages.[18] The Stephan Curve is a graphic representation that explains the rapid drop in pH to a level that can cause demineralization of tooth enamel as fermentable carbohydrates are consumed.[18] Cementum demineralizes at a pH of 6.7. Dentin demineralizes at a pH of 6.5.[18] Enamel demineralizes at a pH of 5.5.[18] In a healthy mouth, after about five to 10 minutes, the saliva begins to buffer the pH, and within an hour, the mouth generally returns to a normal alkaline level.[19] When the biofilm is acidic, the minerals from the enamel dissolve into the saliva and the teeth are left weakened and at risk of caries.[19]

Saliva consists of electrolytes, proteins, enzymes, immunoglobulins, mucins, ammonia, and urea.[20] These salivary components help to modulate demineralization and remineralization of tooth surfaces, buffering capacity and pH of the saliva, bacterial attachment in the plaque biofilm, and antibacterial properties of the saliva.[20] The saliva in contact with the teeth is supersaturated with calcium and phosphate ions that are leached out of the teeth.[15] The saliva responds by increasing the oral pH to encourage the reuptake of minerals back into the enamel.[15] A cycle of low salivary pH provides an acidogenic environment for the growth of aciduric bacteria; this leads to dental caries, which further lowers the salivary pH.[15] Our job is to guide patients on how to interrupt that cycle and help them maintain a balanced oral microbiome to lower their risk of dental caries.

Biofilm disruption

Dental professionals have not been very successful in interrupting that cycle, as evidenced by the current epidemic level of caries disease. That might be due to the fact that many experiments on the efficacy of fluoride have been done in vitro, which means they have not been conducted in the presence of biofilm. On a tooth uncoated in biofilm, fluoride is able to penetrate the outer layers of enamel and strengthen them. In the natural oral environment, however, teeth are coated in biofilm, whose exopolysaccharide layer makes penetration by fluoride extremely challenging. Once fluoride was studied in vivo, on natural teeth with biofilm, it was found that those biofilms significantly inhibit fluoride from reaching the enamel surfaces.[21] Thus, our patients are not getting the full benefit of fluoride, and we continue to see the physiologic breakdown of tooth structure. The question then becomes how we can best penetrate oral biofilm, increase the pH, increase calcium ion availability, deliver remineralizing agents to the tooth surface, and make a significant impact on our patients' oral health.

According to a white paper published by Callister et al. in 2019, the majority of dental products on the market are acidic and contain preservatives and supplemental ingredients such as alcohol, sodium lauryl sulfate, and parabens, which have been shown to be detrimental to oral tissues.[22] The authors contend that while fluoride has been shown to be taken up under acidic conditions, these conditions ultimately contribute to an acidic oral environment preferred by cariogenic organisms, thus perpetuating the cariogenic cycle. Products that penetrate the biofilm without creating an acidic environment and enhance the uptake of calcium ions from saliva would be of great benefit. Fluoride has some beneficial effects; however, fluoride has also been associated with multiple negative health effects.[23] According to Lamont et al., current antimicrobials have limited clinical efficacy to prevent dental caries.[24] There is a need for alternative antimicrobial anticaries agents.[24]

Patient management

Due to the multifactorial nature of dental caries, a multifaceted treatment approach is required to treat and prevent this disease. The objective is to prevent new pathogenic biofilm accumulation, disrupt an established biofilm matrix, interrupt the polymicrobial synergies associated with acidogenesis, rebalance the salivary pH, diminish the dissolution of the minerals in the enamel, and augment the action of remineralizing agents.[24]

Mechanically dislodging supragingival biofilms with manual or power toothbrushing is part of the equation; however, it is impossible to completely remove these deposits this way, especially in difficult-to-access areas such as interproximal and sulcular spaces.[24] Additionally, many individuals who lack the dexterity to perform optimal mechanical hygiene techniques, such as young children, the elderly, and people with certain disabilities, may need supplemental assistance and/or special devices to aid in thorough biofilm removal.[24] One of the most effective mechanical biofilm disruption

devices, especially in difficult-to-reach, protected retentive sites, is a shear-generating, high-velocity water spray.[25] This type of "water flosser" is a tool that most people are able to manipulate more easily than traditional string dental floss. It is possible that caregivers of those who may not be able to perform oral hygiene procedures for themselves may be more likely to utilize this type of device than traditional floss due to its ease of application.

Hydroxyapatite is a calcium-phosphate compound that occurs naturally and is found in dental enamel, bone, and saliva. Nano-hydroxyapatite (nano-HA) is a synthetic compound that is derived chemically or mechanically and is nearly identical to natural HA.[26] Nano-HA has an extremely small particle size and has been shown to have significant remineralization effects on both enamel and dentin. Nano-HA is found in toothpaste and mouthwash in Japan, Canada, and the Netherlands. The FDA has not approved the use of nano-HA in the United States, although it is considered the gold standard of oral health ingredients in other countries. Research has shown that nano-HA demonstrates significant remineralization of both dentin and enamel due to its small particle size. Studies don't show a significant difference between the effectiveness of fluoride and nano-HA in remineralization; however, there is concern over the potentially harmful effects of fluoride. The biocompatibility of nano-HA makes it an excellent choice of remineralizing agent.

The goal of biofilm management is homeostasis, not the destruction or elimination of the biofilm, which can happen with antimicrobial agents. Nano-HA is also proven to have anti-adhesion properties that prevent bacterial adhesion. In addition, nano-HA binds to plaque and proteins, reducing plaque accumulation.[27]

In addition to mechanical biofilm disruption and enamel remineralization, other adjunctive therapies have emerged to address the complex issue of treating and preventing dental caries. Significant research has been undertaken to study plants with the goal of isolating novel biocompatible active drugs that could replace synthetic drugs. These plants would provide a source of natural drugs for nontraditional or alternative modern medicine.[28] Several natural compounds including the plant extracts catechol, emetine, quinine, and flavone have been proven effective in the treatment of dental caries in clinical trials.[29]

Phytochemicals and plant extracts have been shown to inhibit all the stages of the development of dental caries.[28] This includes demonstrating bactericidal activity against cariogenic microbes, suppression of bacterial adherence, inhibition of microbial aggregation, disruption of biofilm formation, and blockage of the production of glycolytic acid.[30]

Many plant products, including herbs and spices, have been proven to be toxic to cells; therefore, the cytotoxic potential of medicinal plants must be evaluated against host cells.[31] The safety of plants intended for therapeutic use must be established and the potential side effects must be ascertained in order for these alternatives to synthetic drugs to be acceptable. The best candidates for the formulation of natural remedies are bioactive compounds with few or no toxic effects on the host.[30]

There is a great deal of interest in the effectiveness of these natural compounds against dental caries. Usha et al. published a randomized controlled clinical trial that demonstrated a 0.5% extract of *Stevia rebaudiana* leaves bolstered the buffering capacity of the saliva and substantially diminished the number of cariogenic microorganisms in patients at high risk for dental caries disease.[32]

Another plant product that has proven effective against dental caries is xylitol, a naturally occurring five-carbon sugar polyol. Xylitol is a white crystalline carbohydrate that has been widely studied during the last 40 years.[22] It is found naturally in berries, fruits, and vegetables, and it is manufactured artificially from xylan-rich plants such as birch and beechwood trees.[22] Xylitol is available in a crystalized form that is very similar in volume and sweetness to sugar and can be substituted for sugar in cooking and baking and for sweetening beverages.

Xylitol is alkaline, practically nonfermentable by oral bacteria, and able to reduce the number of cariogenic strains of bacteria in the oral microbiome. Xylitol reduces the levels of *Streptococcus mutans* in plaque and saliva by disrupting their energy production. Interfering with this process leads to a futile energy cycle and cell death.[33] Xylitol reduces the acid production potential of *Streptococcus mutans* and reduces the bacteria's ability to adhere to the surface of the teeth.[33]

Unlike any other sweetener, xylitol promotes mineralization by increasing the salivary flow when used as a pastille or chewing gum.[33] Additionally, there is a decrease in the amount of plaque biofilm in people who habitually consume xylitol.[33] The human body tolerates xylitol well as a sweetener, and its absorption rate in the small intestine is very slow.[33] There is an upper daily limit to the amount of xylitol that can be tolerated as excess amounts are known to induce osmotic diarrhea.[33]

Nanotechnology that utilizes xylitol is being developed.[22] These technologies also use calcium salts, which act as buffering agents to prevent demineralization and are crucial in the remineralization process.[22] Until we have access to that technology, xylitol is also readily available in the form of candy, mints, chewing gum, and toothpaste. The recommended optimal dose of xylitol for the prevention of dental caries

is 6–10 grams per day at a frequency of three or more times per day.[22]

Another approach has been to manipulate the oral biofilm by modulating the salivary pH with arginine.[24] Arginine can be metabolized to produce alkali, inhibiting the biofilm acidification process, and creating pH homeostasis.[24] The result is the prevention of acidogenic-aciduric bacterial overgrowth.[24]

The use of beneficial bacteria found naturally in the oral microbiome, such as *Streptococcus dentisani* or *Streptococcus A12*, to inhibit the growth of cariogenic species is yet another novel approach.[34] These beneficial bacteria utilized in this way are called probiotics. Probiotics are defined as "living microorganisms, which upon ingestion in certain numbers, exert health benefits beyond inherent basic nutrition."[35] Antimicrobial peptides can augment targeting specificity against caries pathogens such as *Streptococcus mutans* and can increase the numbers of commensal streptococci to rebalance the microbiome.[36]

Rodríguez et al. published a 2016 study comparing milk supplemented with the probiotic *Lactobacillus rhamnosus* with standard milk to determine the effect on patients at high risk for dental caries.[37] The results demonstrated that the group receiving the probiotic milk had fewer new lesions than the control group who received the standard milk, thus confirming the potential benefits of using the probiotic *Lactobacillus rhamnosus* for the control of dental caries.[37] Also, probiotics not only serve as potential antimicrobial agents but also maintain the stability of the oral ecosystem.[37] Many different probiotic supplements are available to patients without a prescription.

Koo et al. reviewed surface modification and antibiofilm coatings as potential biofilm-specific targeting approaches.[25] New drug delivery nanotechnologies have been introduced that can infiltrate biofilms and facilitate drug release in response to acidic pH.[23] This may enhance the effectiveness of chemical treatments that target cariogenic biofilms.[23]

Many research studies have shown the potential anticaries activity of several natural products, and research is ongoing into developing alternative, biological approaches to prevent, reduce, or reverse dental caries disease.[38] Natural products with anticaries benefits include food, beverages, flowers, and traditional herbs.[38] Polyphenol compounds have been proven to be the effective components of most of these products.[37] Some natural products have been found to be effective in altering the demineralization/remineralization balance while others have been studied as antibacterial agents.[38] Ongoing research will further elucidate new alternatives to traditional methods of dental caries control.

Conclusion

Dental caries is one of the most prevalent microbe-mediated oral diseases in humans. The recognized etiology of dental caries is a five-point theory that includes oral microorganisms, the oral environment, which includes saliva and pH, the diet or exposure to dietary carbohydrates, the time and frequency of those exposures, and genetics, the last of which we cannot yet control.

Because dental caries is a multifactorial disease, multiple modes of action must be utilized in the efforts to control it. Excessive exposure to simple carbohydrates induces the accumulation of acid-producing and acid-resistant microorganisms in the mouth, so it is of paramount importance to educate patients about the role of excess exposure to these foods.

Dysbiosis of the oral biofilm drives dental caries. Due to the nature of oral biofilm and its ability to become pathogenic as it matures, disruption of this cycle is a priority. A combination of mechanical debridement with a power toothbrush and water flosser as well as the use of products that can penetrate biofilm can be effective.

It is possible to alter the oral microbiome, remineralize existing lesions, and potentially prevent dental caries disease. This could entail enhancing mechanical biofilm disruption through antimicrobial agents, elevating the pH of the oral environment, and implementing alternative natural strategies to control other environmental variables.

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1. According to the National Institute of Dental and Craniofacial Research, ___% of adults, ages 20 to 64, have had dental caries in their permanent teeth.

A. 52
B. 72
C. 92
D. None of the above

2. According to the National Institute of Dental and Craniofacial Research, ___% of adolescents, ages 12 to 19, have had dental caries in their permanent teeth.

A. 39
B. 59
C. 99
D. None of the above

3. According to the National Institute of Dental and Craniofacial Research, ___% of children, ages 2 to 11, have dental caries in their primary teeth.

A. 22
B. 42
C. 62
D. None of the above

4. Which cariogenic bacterium is recognized as an independent risk factor for cardiovascular disease?

A. *Fusobacterium nucleatum*
B. *Streptococcus mutans*
C. *Porphyromonas gingivalis*
D. All of the above

5. The term "dental caries" originated from the Latin word "caries," which means:

A. Tooth worm
B. Decay
C. Hole
D. None of the above

6. The first text on dental disease and treatment was written by:

A. Pierre Fauchard
B. Underwood and Miles
C. W. D. Miller
D. None of the above

7. W. D. Miller is credited with being:

A. The first American chemist
B. The first dentist
C. The first oral microbiologist
D. All of the above

8. W. D. Miller believed:

A. Bacteria metabolize fermentable carbohydrates and produce acids
B. Acids cause demineralization of tooth enamel
C. No single bacterium is the cause of dental caries
D. All of the above

9. In the 1880s, the nonspecific plaque hypothesis postulated that it was the ___ of the plaque, not the specific bacterial species in the plaque, which causes dental caries.

A. Quality
B. Quantity
C. Thickness
D. All of the above

10. The idea that mechanical plaque removal is the best way to prevent dental caries originated:

A. From the chemico-parasitic theory
B. From the specific plaque hypothesis
C. From the nonspecific plaque hypothesis
D. None of the above

11. The specific plaque hypothesis states that it is ___ in the oral microbiome that is responsible for causing dental caries.

A. Specific bacterial pathogens
B. Benign bacteria
C. Quantity of plaque
D. None of the above

12. Loesche believed:

A. Targeting specific bacteria with antibiotics would cure dental caries
B. *Streptococcus mutans* and lactobacillus are caries-causing bacteria
C. In the absence of specific caries-inducing bacteria, there would be no dental caries disease
D. All of the above

13. Antibiotic treatment for dental caries:

A. Is only minimally effective
B. Is beneficial until stopped
C. Contributes to antibiotic resistance
D. All of the above

14. The ecological plaque hypothesis posits that dental caries disease is a result of:

A. A shift of the oral microbiota to cariogenic species
B. Heavy plaque buildup
C. Not flossing daily
D. None of the above

15. Aciduric and acidogenic bacteria such as ___ become dominant in an acidic oral environment.

A. *Streptococcus mutans* and nonmutans streptococci
B. Lactobacilli and bifidobacteria
C. Actinomyces and yeasts
D. All of the above

16. It was ultimately discovered that for dental caries to occur, one must have:

A. A tooth
B. Tooth-adherent bacteria
C. Fermentable carbohydrates
D. All of the above

17. As a result of frequent exposures to fermentable carbohydrates, the oral microbiota changes to:

A. Cariogenic
B. Acidogenic
C. Aciduric
D. All of the above

18. Acidogenic means:

A. Acid-attracted
B. Acid-forming
C. Acid-opposed
D. All of the above

19. Aciduric means:

A. Capable of tolerating an acidic environment
B. Incapable of tolerating an acidic environment
C. Elevating the pH
D. None of the above

20. As bacteria metabolize fermentable carbohydrates, the result is:

A. A drop in pH
B. An increase in pH
C. A shift to neutral pH
D. None of the above

21. A decrease in the oral pH results in:

A. Remineralization of enamel
B. Demineralization of enamel
C. Tooth mobility
D. None of the above

22. Remineralization is:

A. The body's natural reconstruction process
B. The use of calcium ions to rebuild tooth structure
C. The use of phosphate ions to rebuild tooth structure
D. All of the above

23. The Stephan Curve is:

A. A picture of caries formation over time
B. A graphic representation to explain the rapid drop in pH as foods and beverages are consumed
C. An explanation of remineralization of tooth structure
D. None of the above

24. The pH at which enamel demineralizes is:

A. 7.0
B. 6.5
C. 5.5
D. None of the above

25. Patient education must include information regarding:

A. The Stephan Curve
B. The importance of neutral pH
C. Frequent applications of antimicrobial agents such as xylitol
D. All of the above

26. Saliva is supersaturated with:

A. Iron and calcium
B. Potassium and phosphate
C. Calcium and phosphate
D. All of the above

27. Many experiments on the efficacy of fluoride have been done in vitro, meaning:

A. On a tooth in a lab
B. On a tooth uncoated in biofilm
C. On a tooth with no exopolysaccharide layer
D. All of the above

28. The exopolysaccharide layer of the biofilm:

A. Makes fluoride penetration extremely challenging
B. Inhibits fluoride from reaching enamel surfaces
C. Makes fluoride less effective than previously understood
D. All of the above

29. A shear-generating, high-velocity water spray:

A. Is one of the most effective mechanical biofilm disruption devices
B. Is a device only for children
C. Is not recommended in place of traditional string floss
D. All of the above

30. Several natural compounds have been proven effective in the treatment and prevention of dental caries. These include:

A. Phytochemicals
B. Plant extracts
C. Probiotics
D. All of the above

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ENDEAVOR BUSINESS MEDIA

PUBLISHED: SEPTEMBER 2023
EXPIRES: AUGUST 2026

Dental caries: A new protocol for an age-old disease

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EDUCATIONAL OBJECTIVES

1. Recognize the history and extent of dental caries disease
2. Describe the role of pH, saliva, biofilm, diet, and genetics in the dental caries disease process
3. Clarify the role of the Stephan curve in caries risk education
4. Develop self-care recommendations based on the current science of the dental caries disease process

COURSE EVALUATION

1. Were the individual course objectives met?
   Objective #1: Yes No Objective #3: Yes No
   Objective #2: Yes No Objective #4: Yes No

Please evaluate this course by responding to the following statements, using a scale of Excellent = 5 to Poor = 0.

2. To what extent were the course objectives accomplished overall? 5 4 3 2 1 0
3. Please rate your personal mastery of the course objectives. 5 4 3 2 1 0
4. How would you rate the objectives and educational methods? 5 4 3 2 1 0
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7. Was the overall administration of the course effective? 5 4 3 2 1 0
8. Please rate the usefulness and clinical applicability of this course. 5 4 3 2 1 0
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10. Do you feel that the references were adequate? Yes No
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12. If any of the continuing education questions were unclear or ambiguous, please list them.

    _____

13. Was there any subject matter you found confusing? Please describe.

    _____

14. How long did it take you to complete this course?

    _____

15. What additional dental continuing education topics would you like to see?

    _____

    _____

Mail/fax completed answer sheet to:

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7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	22.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	23.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	24.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	25.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	26.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	27.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	28.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	29.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
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