Caries Process and Prevention Strategies: The Environment

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Continuing Education Units: 1 hour


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

This is part 4 of a 10-part series entitled Caries Process and Prevention Strategies. In this course, the role of fermentable carbohydrates is discussed, paying particular attention to how caries can be influenced by the cariogenic potential of ingested sugars and starches, the physical traits of ingested carbohydrates (such as their adhesiveness), and the frequency of intake and exposure to sugars. The Stephan curve, which illustrates the dental pH changes over time in response to a carbohydrate challenge, is also introduced, with a discussion of how factors such as the type of carbohydrate, the buffering capacity of bacteria, and the type and amount of bacteria present in plaque affect dental plaque pH responses.

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

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Overview

It has been established that the oral environment is one of the primary factors in the caries process. Only when acidity increases in the oral environment does demineralization of enamel, and subsequently caries, occur. In this section, the role of fermentable carbohydrates is discussed, paying particular attention to how caries can be influenced by the cariogenic potential of ingested sugars and starches, the physical traits of ingested carbohydrates (such as their adhesiveness), and the frequency of intake and exposure to sugars. The Stephan curve, which illustrates the dental pH changes over time in response to a carbohydrate challenge, is also introduced, with a discussion of how factors such as the type of carbohydrate, the buffering capacity of bacteria, and the type and amount of bacteria present in plaque affect dental plaque pH responses.

Clinical Significance Snapshots

**Which environmental factors can easily be modified to aid in prevention of dental caries?**

Some factors are much easier to modify than others. The consumption of sugars (as Fermentable carbohydrates) is largely discretionary and therefore can be controlled by the patient. Many foods and beverages have sugar-free forms and can easily be substituted in the daily routine. Likewise, the addition of ‘table sugar’ to meal items such as cereals and hot drinks should be limited and avoided altogether or substituted by non-cariogenic sugar alternatives when possible. Not only should the amount of sugar be limited, but the Stephan curve tells us the frequency should be reduced wherever possible. So, a patient at risk of developing new carious lesions should restrict all exposure to sugars to mealtimes only.

The dental plaque biofilm should be controlled as much as possible by frequent and thorough oral hygiene. As it is simply not possible to remove all acidogenic/cariogenic bacteria from the mouth with conventional oral hygiene, it is not surprising that there are few studies showing any correlation between oral hygiene and the prevention of dental caries.

If demineralization is unavoidable, then the environment can be modified to encourage remineralization. Saliva is the key remineralizing agent, as it is supersaturated with respect to calcium and has good buffering capacity. Saliva production can be stimulated by chewing sugar-free gum. The increase in the flow of saliva also helps to reduce the clearance time, reducing the length of exposure of cariogenic substances in the oral cavity.

**How can I work with my patient in modifying the environmental factors to reduce the risk of developing dental caries?**

If a patient or other family members have signs of caries, it is critically important to look for environmental factors that may be contributing to increased risk of developing the disease. Changing environmental factors is always a challenge, and success is more likely if the family unit’s environment is investigated as well as that of the specific at-risk individual family member. Changing the environmental factors of only one family member is unlikely to succeed.

Fermentable carbohydrates (especially the mono- and disaccharides of glucose and sucrose) are the most important causative factor to change. Eating and drinking habits should be investigated—ideally through a 3- or 4- day diet diary that lists all eating and drinking occasions—to assess the amount and frequency of exposure. Between-meal episodes should be reduced wherever possible, and sugar substitutes should be used. Sugars as part of a meal should be reduced, and fruits or vegetables, or
a sugar substitute, could be used instead of many sugar-containing foods. If obesity is present in the family, referral to a dietitian/nutritionist may be very beneficial for all.

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Learning Objectives

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

• Identify the role of the environment in dental caries etiology.
• Introduce the Stephan curve.
• Explain the impact of various diets on the incidence of caries.
• Describe the concept of frequency versus amount of cariogenic carbohydrates.
• Be familiar with the complex chemical structure of sugars.
• Relate the cause and effect of diet and dental caries to patients.

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Glossary

acidogenic bacteria – Bacteria that have the capability of producing acids through their metabolic pathways. In reference to dental caries, the main acidogenic or acid-producing species of bacteria is Streptococcus mutans. Through the process of glycolysis breaks down mono- and disaccharides into lactic acid. Lactic acid diffuses through the plaque biofilm to reach the enamel surface, where it may cause demineralization, depending upon other factors such as availability of buffering agents and the saturation with calcium.

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

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

clearance time – The interval of time necessary for any substance to be cleared from the mouth by the process of salivary secretion and saliva flow. Factors that affect clearance time, other than saliva flow rate, include the form and ‘stickiness’ of the item to be cleared and the saliva-stimulating potential of the item. A glucose solution will be cleared much faster than sticky caramel.
critical pH – The pH at and below which demineralization of enamel occurs. The research of Stephan and Miller originally demonstrated this critical pH to be approximately 5.5 (see Stephan's curve). Due to other chemical factors, especially the saturation of the immediate environment of the enamel surface with respect to calcium and phosphate, the presence of buffering agents, and the fluoride availability, the critical pH may vary and is considered to be between 4.5 and 5.5.

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.

fermentable carbohydrates – Nearly all carbohydrates in the diet are can be broken down and metabolized by microorganisms. Mono- and disaccharides (sugars such as glucose and sucrose) are most readily metabolized and are therefore the most cariogenic, as they are metabolized to produce lactic acid.

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

‘Stephan Curve’ – The term refers to a graph published by Stephan and Miller in the 1940s. The graph reflected Stephan and Miller’s research demonstrating the fall in pH in the mouth following a glucose rinse. They demonstrated that a pH of 5.5 or less may result in demineralization, and that the pH level may remain below this ‘critical level’ for approximately 20 minutes; with pH completely returning to normal or resting levels in about 45 to 60 minutes.

Introduction
In 1947, a series of human experiments were begun on patients in Vipeholm Mental Hospital in Sweden. Unknown to the Swedish government, but sanctioned by the dental community and the confectionery industry, a group of mental patients were fed copious amounts of sweet foods, like chocolates and caramels, in a full-scale experiment designed to bring about tooth decay. The experiments provided extensive knowledge about dental health, and resulted in the breakthrough findings that the intake of sugar was linked to dental caries, that certain physical qualities of sugars (such as their stickiness) influence caries risk, and that the frequency with which sugary foods are consumed also affects caries development. While, scientifically speaking, the experiment was a success, with more having been learned about dental health and caries than from any previous study, the study would never have taken place today: It violates the principles of medical ethics. Many subjects ended up with their teeth completely ruined to provide fodder for subsequent studies that continued to increase dental knowledge, and to provide much of the information that follows about the oral environment factors that play a role in the dental caries process.

The Role of Fermentable Carbohydrates
The presence of fermentable carbohydrates changes the oral environment. Three main factors play a role in the dental caries process: the cariogenic potential of fermentable carbohydrates, the physical traits of fermentable carbohydrates, and the frequency of intake of, and exposure to, fermentable carbohydrates.

Cariogenic Potential
Simple sugars like sucrose, fructose, lactose,
galactose, and glucose foster colonization and growth of bacteria linked to caries, particularly *Streptococci mutans*. Studies indicate that subjects placed on high-sucrose diets exhibit increased *S mutans* counts and the incidence of early carious lesions. Although starch is recognized to have a lower carcinogenicity than sucrose, frequent consumption of starch has been shown to produce a large number of carious lesions. This is because starch can be broken down to maltose by the enzyme in saliva called amylase. Maltose can then be further metabolized to acids, which in turn lead to demineralization.

Conversely, dietary restriction of fermentable carbohydrates and cooked starches reduce the level of cariogenic organisms in humans. A classic 15-year intervention study, the Hopewood House Study conducted in Australia, evaluated the clinical effects of a sucrose-restricted diet among 81 children, aged 4 to 9 years. At the start of the study, 78% of the children were caries-free, and 53% continued to be caries-free at age 13. This was significantly higher than the proportion of caries-free 13-year-olds within the general residential population—only 0.4%. When the children from Hopewood House were relocated as they became older, they no longer adhered to their strict diet. The result was a steep increase in caries increment, similar to that found in other children, indicating that teeth do not acquire any permanent resistance to dental caries.

(See "Children's Teeth" on pages 12-13)

**Physical Traits**

These include the adhesiveness (stickiness) and clearance time of dietary carbohydrates, as well as the frequency of exposure to them. In general, those dietary carbohydrates that are sticky confer the highest potential for caries. This was first demonstrated in the Vipeholm Study, which had study subjects consume sucrose in toffee, chocolate, caramel, bread, or in a liquid form. It was shown that the intake of sticky foods like toffees and caramels produced higher caries rates among monitored subjects than those who consumed rapidly swallowed sugars. The increase in caries activity disappeared when sugar-rich foods were reduced or removed from the diet. However, the cariogenicity of liquid sugars should not be discounted given the high incidence of caries with soft-drink consumption and the occurrence of early childhood ("baby bottle") caries.

It is also important to consider the clearance rate of dietary carbohydrates in the caries process. Different foods are cleared from the oral cavity at different rates. For example, sticky, retentive...
foods such as toffees, or foods that can compact in the pits-and-fissures of the teeth, such as biscuits and cakes, have increased clearance times. In general, refined carbohydrates that are retained for long periods tend to be the most cariogenic. Also, bacterial acid production can persist after the carbohydrate has cleared from the oral cavity.

The link between the frequency of carbohydrate intake and caries incidence was also investigated in The Vipeholm Study. When study subjects ingested 300 g of sugar with meals, a significantly lower caries rate was observed (0.43 new carious lesions per year) compared to subjects ingesting the same amount of sugar as snacks between meals (4.02 new carious lesions annually).

Increased snacking increases the risk of caries because increasing the frequency of sugar intake extends the duration of acid production and exposure, thereby tipping the scale toward the development of caries. This can be demonstrated simply by measuring plaque pH (which would be the immediate environment of the tooth) throughout the day. In the example below it can be clearly seen that in A, increasing the frequency of eating and drinking increases the episodes when the pH of plaque falls below 5.5. In B, restricting between-meal snacks and drinking non-sugared drinks reduces the time that plaque pH falls below 5.5.

An interesting observation is that it can be less beneficial to eat one sweet than it is to eat five sweets in immediate succession. With five in succession, the levels of sucrose may be toxic to bacteria and there may be a greater salivary stimulatory effect. Furthermore, if five sweets are spread out throughout the day, oral pH would be depressed for more episodes. The message for patients: Consume all sweets in one episode, and preferably following a meal, rather than spreading them throughout the day.

The Stephan Curve
Acidogenic bacteria in dental plaque rapidly metabolize fermentable carbohydrates producing acidic end products. In the mouth, these changes over time in response to a challenge (usually a cariogenic food) are known as Stephan responses or Stephan curves. The pH of dental plaque under resting conditions (i.e., when no food or drink has been consumed), is fairly constant. Differences do exist, however, between individuals and in different sites within an individual.

The response after exposure of dental plaque to a fermentable carbohydrate is that pH decreases rapidly, reaching a minimum in approximately 5 to 20 minutes. This is followed by a gradual recovery to its starting value, usually over 30 to 60 minutes, although this can be longer in some individuals.

The following sections discuss some of the mechanisms underlying each stage of the Stephan curve:

Frequency of Intake or Exposure
Increased snacking increases the risk of caries because increasing the frequency of sugar intake extends the duration of acid production and exposure, thereby tipping the scale toward the development of caries. This can be demonstrated simply by measuring plaque pH (which would be the immediate environment of the tooth) throughout the day. In the example below it can be clearly seen that in A, increasing the frequency of eating and drinking increases the episodes when the pH of plaque falls below 5.5. In B, restricting between-meal snacks and drinking non-sugared drinks reduces the time that plaque pH falls below 5.5.

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Resting Plaque pH
This describes plaque that has not been exposed to fermentable carbohydrates for approximately 2 hours and generally has a pH of between
Figure 4. Frequent sugar-containing snacks between meals cause more episodes of the oral pH falling under 5.5, increasing caries risk. (B.) Fewer sugar-containing snacks between meals cause fewer episodes of the oral pH under 5.5, reducing caries risk.

Figure 5. Plaque pH after eating 1 sweet or 5 sweets in succession.
also present. These metabolic products are present in plaque in much higher concentrations than in saliva, partly because they are constantly produced from the intra- and extracellular metabolism of bacterial carbohydrate stores, as well as from the breakdown of salivary glycoprotein.6

**Decrease in Plaque pH**

After exposure of dental plaque to fermentable carbohydrates, the pH decreases rapidly. This is due primarily to the production of lactic acid in plaque, with acetic and propionic acids being simultaneously lost from the plaque.6,10

The rate at which the pH decreases is due in part to the microbial composition of dental plaque. In general, if more acidogenic, aciduric bacteria is present in plaque, the pH would lower more rapidly. The rate of pH decrease is also dependent on the speed with which plaque bacteria are able to metabolize the dietary carbohydrate. While sucrose would be metabolized quickly, prompting a more rapid decrease, larger molecules, like starch, would diffuse into plaque more slowly because it would need to be broken down before it can be assimilated by plaque microbes.6 Another factor that affects the rate of pH decrease is the buffering capacity of unstimulated saliva.6,11

The rate at which plaque pH decreases is also influenced by the density of plaque. Less dense plaque can be penetrated more easily by buffering saliva and oxygen causing slower pH decreases than very dense plaque, which cannot be accessed by saliva and oxygen.6,12

**Increase in Plaque pH**

The gradual recovery of the plaque pH is influenced by various factors. These include the buffering capacity of saliva, whether fermentable carbohydrate remains in the mouth, the pH value (which may be unfavorable to bacterial enzyme systems) and the diffusion of acids from plaque into saliva or teeth. It is also influenced by base production in plaque. Ammonia from the deamination of amino acids and breakdown of urea in saliva are examples of reactions that contribute to the pH rise. These bases are important to neutralize acid when carbohydrate intake is moderate.13 The rise in pH may also be assisted by the removal of acids by bacteria, such as those from the genus Veillonella that

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**Video 1. Explain the significance of the Stephan Curve in the initiation and prevention of dental caries?**

Click here to view this video on dentalcare.com.

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**Figure 6. The Stephan Curve**

Salivary Stimulation and Plaque pH
The fact that saliva is so beneficial in terms of buffering and neutralizing acidic plaque pH values has stimulated much interest in agents that increase salivary flow rates. Chewing gum or unflavored materials such as paraffin wax after consuming fermentable carbohydrates leads to an increase in salivary flow with a concurrent rapid rise in plaque pH. This rise has been shown to be closely associated with a rise in bicarbonate buffering capacity, as well as an increased supply of nitrogenous substrates, which are metabolized to basic (less acidic) end products. The chewing of cheeses rich in nitrogenous compounds gives rise to similar pH increases found with paraffin wax, despite the cheese itself being acidic. This is probably due to the breakdown of casein and other cheese proteins, as well as the fact that cheese is a strong sialogogue, an agent that increases the flow of saliva. Cheese has the added advantage of raising the plaque concentrations of calcium and phosphate and, therefore, increasing the chance of remineralizing teeth.

Critical pH
The critical pH is the pH at which saliva and plaque fluid cease to be saturated with calcium and phosphate, thereby permitting the hydroxyapatite in dental enamel to dissolve. It is the highest pH at which there is a net loss of enamel from the teeth, which is generally accepted to be about 5.5 for enamel. The solubility of acid varies with pH but it is also complicated by the fact that teeth are bathed in saliva, which is constantly replenished and supersaturated with apatite, whose composition varies. By increasing the concentration of calcium and/or phosphate, it is possible to reduce the effective critical pH so that teeth are able to withstand lower pH values before demineralizing.

Conclusion
Understanding how fermentable carbohydrates influence the oral environment, and in turn, caries risk, is key to helping the dentist teach the patient about effective caries prevention. To that end, it is important for the dentist to understand the factors that affect food’s cariogenicity and the Stephan responses of plaque pH to challenges by fermentable carbohydrates.
Figure 8. Plaque pH responses by a sugar-rich snack alone, and followed by sugared or sugar-free chewing gum.
Adapted from: Manning RH, Edgar WM. pH changes in plaque after eating snacks and meals, and there modification by chewing sugared- or sugar-free gum. Br Dent J. 1993;174(7):241-244.

Figure 9. Plaque pH responses to a sucrose mouthrinse alone, and followed by paraffin or cheese.
Figure 10. Caries Lesion Initiation and Progression - Fermentation Produces Acid Leading to Demineralization

Figure 11. Caries Lesion Initiation and Progression - Demineralization
HOW DO I CARE FOR MY CHILD’S TEETH?

Good oral care begins before a baby’s first tooth.

Babies are born with all their teeth. You can’t see them because they are hidden in the gums. Baby teeth start to break through the gums around 6 months. But it is important to start good oral care even before the first tooth comes in. From healthy gums come healthy teeth.

Kids have all their baby teeth by age 3. These are called primary teeth. Baby teeth start falling out around age 6; that’s when the permanent, or adult, teeth start coming in. Gaps between baby teeth are normal. They make room for the permanent teeth. Most permanent teeth come in by age 13.

Bottle Tooth Decay is a serious problem.

Bottle Tooth Decay can happen if babies drink milk, formula, or juice out of bottles over long periods of time.

To avoid it:
• Take the bottle away after your baby is done drinking.
• Don’t put your baby to bed with a bottle.

Here are some tips to keep kids’ teeth healthy and strong

0–2 years
• Wipe gums with a washcloth after feeding. This will help get rid of the sticky coating called plaque that can cause tooth decay.
• Brush teeth twice a day with water and a soft-bristle toothbrush. If there is a high rate of cavities in your family you may apply a “smear” of fluoride toothpaste to the teeth with your finger.
• Start using fluoride toothpaste at age 2. Use only a pea-sized amount. Make sure your child spits it out after brushing.
• Begin flossing as soon as teeth touch.
• Ensure infant receives an oral health risk assessment from primary health care provider by 6 months of age. Schedule first dental appointment before first birthday.

3–5 years
• Continue using fluoride toothpaste. Use only a pea-sized amount. Make sure your child spits it out after brushing.
• Try to break thumb-sucking and pacifier habits by age 4.
• Continue visiting your dental team every 6 months.
HOW DO I CARE FOR MY CHILD’S TEETH?

6–9 years
• Let your child know that it’s normal for baby teeth to fall out. That’s how “grown-up” teeth come in.
• Until children are able to practice proper oral health habits alone, parents should help their child brush and floss twice a day.
• Always pay special attention to the back teeth, which may have more plaque.

10–12 years
• Require children who play sports to wear a mouthguard to protect their smile.

13+ years
Parents can make the most of their teen’s interest in how they look by reminding them that a healthy smile and fresh breath will help them look and feel their best.

Here are some tips
• Encourage teens who wear braces to brush and floss thoroughly.
• How teeth look when braces come off depends on how they’re treated while the braces are on.
• Suggest that teens carry a toothbrush, toothpaste, and floss in their purse or gym bag for use during the day.

Quick tips for better oral health for kids
• Start practicing good oral care even before the first tooth comes in. From healthy gums come healthy teeth.
• Parents should schedule their child’s first dental appointment before the first birthday and every 6 months starting at age 3.
• It is important that children brush twice a day with a fluoride toothpaste and begin flossing as soon as two teeth touch.
• Children should limit sugary and sticky foods and drinks to protect against tooth decay.

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

1. Which of the following is true about the cariogenic potential of carbohydrates?
   a. Starch has been shown to produce a large number of caries.
   b. Starch and sucrose have the same cariogenic potential.
   c. Of all the sugars, only sucrose, fructose and glucose are cariogenic.
   d. Maltose sugars are not cariogenic.

2. What is one main finding of the Australian Hopewood House study?
   a. Restricting sugar intake only reduces caries risk.
   b. Dietary restriction of fermentable carbohydrates and cooked starches reduce the level of cariogenic
      organisms.
   c. After years of sugar restriction, one can develop resistance to caries.
   d. After returning to a normal diet, caries incidence in the study subjects did not increase.

3. Which of the following physical traits of food confers the highest potential for dental caries?
   a. Soft and mushy
   b. Liquid
   c. Sticky
   d. Hard and brittle

4. What is one main finding of the Swedish Vipeholm study?
   a. Liquid foods cause little to no caries.
   b. When study subjects ingested sugar with meals, a lower caries rate was observed than when study
      subjects ingested the same amount of sugar as snacks between meals.
   c. Retentive foods are not significantly cariogenic.
   d. Only teeth with pits and fissures are prone to caries.

5. Why does snacking more often increase caries risk?
   a. Just one snack acidifies oral pH for a long period of time.
   b. The snacks must only be of the retentive type to cause caries.
   c. The mechanism is not known.
   d. Because increasing the frequency of sugar intake extends the duration of acid production and
      exposure.

6. What explains the phenomenon that eating five sweets in succession is better than having
   just one?
   a. Five sweets in a row causes dental plaque pH to fall below 5.5.
   b. The pH of saliva becomes less acid.
   c. The levels of sucrose may be toxic to bacteria and there may be a greater salivary stimulatory effect.
   d. All of the above.

7. Once exposed to fermentable carbohydrates, how long does it take on average for plaque pH
   to reach its minimum?
   a. 5 to 20 minutes
   b. 1 to 3 minutes
   c. 30 to 60 minutes
   d. There is no average; it depends entirely on the individual.
8. After exposure to fermentable carbohydrates, how long does it take on average for pH levels to return to its starting value?
   a. 15 to 20 minutes
   b. 30 to 60 minutes
   c. 90 minutes
   d. There is no average; it depends entirely on the individual.

9. Which of the following best describes plaque in resting pH?
   a. This is plaque that has not been exposed to fermentable carbohydrates for approximately 2 hours and generally has a pH between 6 and 7.
   b. Plaque in resting pH has a pH of 8 to 9.
   c. Plaque in resting pH is not very stable.
   d. None of the above.

10. Which of the following is true about the chemical composition of resting plaque?
    a. Ammonia is not present.
    b. There are relatively high concentrations of (less acidic) acetate compared to (more acidic) lactate.
    c. The amino acids glutamate and proline are not present.
    d. There is more lactate than acetate.

11. Which factor below affects the rate of decrease in plaque pH?
    a. The rate of pH decrease is affected by the buffering capacity of unstimulated saliva.
    b. The rate of pH decrease is influenced by the density of plaque.
    c. The rate of pH decrease is dependent on the speed with which plaque bacteria are able to metabolize dietary carbohydrates.
    d. All of the above.

12. What factors affect the recovery of plaque pH?
    a. The buffering capacity of saliva and whether fermentable carbohydrates remain in the mouth.
    b. The speed with which plaque bacteria are able to metabolize dietary carbohydrates.
    c. The source of the acid attack.
    d. The frequency with which the oral environment comes under attack.

13. What is the importance of *Veillonella* bacteria?
    a. *Veillonella* use lactate as a substrate, metabolizing it to less acidic products, raising plaque pH.
    b. The presence of *Veillonella* reduces salivary flow.
    c. The presence of *Veillonella* increases caries risk.
    d. *Veillonella* increases the acidity of plaque.

14. Why does cheese have a beneficial effect on saliva?
    a. Cheese has the advantage of raising the plaque concentrations of calcium and phosphate, increasing the chance of remineralizing teeth.
    b. Cheese is a strong sialogogue, an agent that increases the flow of saliva.
    c. The chewing of cheeses rich in nitrogenous compounds gives rise to pH increases, despite the cheese itself being acidic.
    d. All of the above.

15. What is the critical pH at which saliva and plaque fluid cease to be saturated with calcium and phosphate, permitting the hydroxyapatite in dental enamel to dissolve?
    a. It is generally accepted to be 6.5.
    b. It is the highest pH at which there is a net loss of enamel from the teeth, which is generally accepted to be about 5.5 for enamel.
    c. It is generally accepted to be 4.5.
    d. Enamel can dissolve at any pH.
References

About the Author

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

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 membership in the Institute of Biology. She was appointed a research fellow in the Department of Clinical Dental Sciences at the University of Liverpool, where she was promoted later to senior lecturer and then to a professor.

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

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