Caries Process and Prevention Strategies: The Environment

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Conflict of Interest Disclosure Statement
- The authors report no conflicts of interest associated with this course.

Introduction
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 saliva, and the type and amount of bacteria present in plaque affect dental plaque pH responses.

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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 saliva, and the type and quantity of bacteria present in plaque affect dental plaque pH responses.

Learning Objectives

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

- Identify the role of the environment in dental caries etiology.
- Understand and discuss 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.

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, acidogenic bacteria break 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 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.

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

'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 returning to normal or resting levels in about 30 to 60 minutes.

Video: The Environment

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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/ce371/test*

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. B and C

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. There is no explanation for this phenomenon.

7. **Once exposed to fermentable carbohydrates, how long does it take on average for plaque pH to reach its minimum?**
   A. 5 to 10 minutes
   B. 20 to 30 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. Resting plaque is not likely to respond to a carbohydrate challenge.

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 number of active caries present.
   B. The rate of pH decrease is influenced by the daily quantity of water ingested.
   C. The rate of pH decrease is dependent on the speed with which plaque bacteria are able to metabolize dietary carbohydrates.
   D. The age of the individual.

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 fluoride, increasing the chance of remineralizing teeth.
   B. Cheese contains key ingredients that decrease 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. Cheese has not been shown to have a beneficial effect on saliva.

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

1. Gustafsson BE, Quensel CE, Lanke LS, et al. The Vipeholm dental caries study; the effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years. Acta Odontol Scand. 1954 Sep;11(3-4):232-64. doi: 10.3109/00016355308993925.
2. Burne RA. Oral streptococci... products of their environment. J Dent Res. 1998 Mar;77(3):445-52. doi: 10.1177/00220345980770030301.
3. Kashket S, Zhang J, Van Houte J. Accumulation of fermentable sugars and metabolic acids in food particles that become entrapped on the dentition. J Dent Res. 1996 Nov;75(11):1885-91. doi: 10.1177/00220345960750111101.
4. Harris R. The biology of the children of Hopewood House, Bowral, N.S.W. VI. The pattern of dental caries experience. Aust Dent J. 1967 Jun;12(3):220-7. doi: 10.1111/j.1834-7819.1967.tb04263.x.
5. Vadiakas G. Case definition, aetiology and risk assessment of early childhood caries (ECC): a revisited review. Eur Arch Paediatr Dent. 2008 Sep;9(3):114-25. doi: 10.1007/BF03262622.
6. Marsh PD, Lewis MAO, Rogers H, et al. Oral Microbiology. 6th ed. 2016; Edinburgh: Churchill Livingstone Elsevier.
7. Edgar WM. Duration of response and stimulus sequence in the interpretation of plaque pH data. J Dent Res. 1982 Oct;61(10):1126-9. doi: 10.1177/00220345820610100301.
8. Jenkins GN. Dental caries in relation to nutrition: environmental effects. Proc Nutr Soc. 1959;18(1):85-91. doi: 10.1079/pns19590021.
9. Sissons CH, Wong L, Shu M. Factors affecting the resting pH of in vitro human microcosm dental plaque and Streptococcus mutans biofilms. Arch Oral Biol. 1998 Feb;43(2):93-102. doi: 10.1016/s0003-9969(97)00113-1.
10. Higham SM, Edgar WM. Human dental plaque pH, and the organic acid and free amino acid profiles in plaque fluid, after sucrose rinsing. Arch Oral Biol. 1989;34(5):329-34. doi: 10.1016/0003-9969(89)90105-2.
11. Stookey GK. The effect of saliva on dental caries. J Am Dent Assoc. 2008 May;139 Suppl:11S-17S. doi: 10.14219/jada.archive.2008.0347.
12. Ahn SJ, Ahn SJ, Browngardt CM, Burne RA. Changes in biochemical and phenotypic properties of Streptococcus mutans during growth with aeration. Appl Environ Microbiol. 2009 Apr;75(8):2517-27. doi: 10.1128/AEM.02367-08. Epub 2009 Feb 27.
13. Shu M, Morou-Bermudez E, Suárez-Pérez E, Rivera-Miranda C, Browngardt CM, Chen YY, Magnusson I, Burne RA. The relationship between dental caries status and dental plaque urease activity. Oral Microbiol Immunol. 2007 Feb;22(1):61-6. doi: 10.1111/j.1399-302X.2007.00325.x.
14. Edgar WM, Dawes C and O'Mullane D. Saliva and Oral Health. 3rd ed. BDJ Books, London, UK. 2004.
15. Manning RH, Edgar WM. pH changes in plaque after eating snacks and meals, and their modification by chewing sugared- or sugar-free gum. Br Dent J. 1993 Apr 10;174(7):241-4. doi: 10.1038/sj.bdj.4808141.
16. Higham SM, Edgar WM. Effects of Parafilm and cheese chewing on human dental plaque pH and metabolism. Caries Res. 1989;23(1):42-8. doi: 10.1159/000261153.
17. Featherstone JD. Dental caries: a dynamic disease process. Aust Dent J. 2008 Sep;53(3):286-91. doi: 10.1111/j.1834-7819.2008.00064.x.

Additional Resources

- No Additional Resources Available.

About the Authors

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Sue is currently Honorary Emeritus Professor in the School of Dentistry in the Institute of Life Course and Medical Sciences and Honorary Senior Research Fellow in the Institute of Population Health, University of Liverpool, United Kingdom. She 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.

Dr. Higham has supervised more than 50 postgraduate students and has published widely with approximately 400 peer-reviewed papers and book chapters. Her main research interests have been 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 since 1983. Dr. Higham was a scientific advisor for the European organization for caries research (ORCA) for many years and was a dentistry panel member for the Research Excellence Framework (REF) in the UK.

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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 acquired enamel lesions in less than two weeks and continued his interests in imaging by studying the natural fluorescence of dental plaque and the lethal photosensitization of periodontal pathogens by means of their intrinsic porphyrins.

Chris served two terms on the British Society for Oral and Dental Research (BSODR) Oral Microbiology and Immunology Group (OMIG) management committee and was elected onto the management board of the BSODR in 2017. He has also previously served on the editorial board of the Journal of Medical Microbiology. Chris left academia in 2018.

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Video Course Presenter

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Robert Faller has in excess of 40 years in the Oral Care Research field. He retired from P&G after more than 31 years in Oral Care, where he focused on caries and enamel related research as P&G's chief cariologist. He is editor of *Volume 17 – Monographs in Oral Science: Assessment of Oral Health – Diagnostic Techniques and Validation Criteria*. He has written 3 book chapters, published 34 papers in peer-reviewed journals and has over 100 published abstracts on fluoride, caries, dental erosion, and various oral care technologies, along with 5 patents related to Oral Care and 6 Continuing Education courses. He currently resides in the UK and is a consultant to the Oral Care industry.

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