

Caries Process and Prevention Strategies: The Agent



Course Author(s): Susan Higham, BSc, PhD, CBIOL, MRSB; Chris Hope, BSc (Hons), PhD, FHEA; Sabeel Valappil, BSc, MSc, PhD, PGCertEd, FHEA; Phil Smith, BDS, MDS, PhD, FDS, DRD, MRD, FDS (Rest Dent) RCS (Edin), FHEA

Video Course Presenter: Robert V. Faller

CE Credits: 1 hour

Intended Audience: Dentists, Dental Hygienists, Dental Assistants, Dental Students, Dental Hygiene Students, Dental Assistant Students

Date Course Online: 12/09/2010

Last Revision Date: 06/29/2021

Course Expiration Date: 06/28/2024

Cost: Free

Method: Self-instructional

AGD Subject Code(s): 11

Online Course: www.dentalcare.com/en-us/professional-education/ce-courses/ce369

Disclaimers:

- P&G is providing these resource materials to dental professionals. We do not own this content nor are we responsible for any material herein.
- 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.

Conflict of Interest Disclosure Statement

- The authors report no conflicts of interest associated with this course.

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.

Course Contents

- Overview
- Learning Objectives
- Glossary
- Video: The Agent
- Course Test
- References / Additional Resources
- 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.¹ Among dentate adults aged 20 to 64, 91% have caries in permanent teeth.² 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.

Learning Objectives

Upon 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).

anaerobic – Living in the absence of air or free oxygen.

biofilm – An aggregation of microorganisms in which cells adhere to each other forming small communities that are held together by an extracellular polymeric matrix. Different communities are co-dependent on each other, and the whole biofilm forms a defensive mechanism requiring much higher concentrations of antimicrobials to control its growth. Dental plaque is a classic biofilm.

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

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

demineralization – The chemical process by which minerals (mainly calcium) are removed from the dental hard tissues - enamel, dentin, and cementum. The chemical process occurs through dissolution by acids or by chelation, and the rate of demineralization will vary due to the degree of supersaturation of the immediate environment of the tooth and the presence of fluoride. In optimal circumstances, the minerals may be replaced through the process of remineralization.

dental plaque – An organized community of many different microorganisms that forms itself into a biofilm and is found on the surface of the tongue and all hard surfaces in the oral cavity. Dental plaque is present in all people and can vary from being comprised of totally healthy microorganisms (commensals) to being very harmful (pathogenic), predisposing the patient to dental caries or periodontal diseases. Note: Dental plaque is not food debris, nor does it contain food debris. Dental plaque can only be completely removed by mechanical means such as toothbrushing or prophylaxis. Food debris can be removed by rinsing.

disaccharides – Any group of carbohydrates, such as sucrose or lactose, that yield monosaccharides on hydrolysis; also called double sugars.

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.

Video: The Agent



[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/test

- 1. Four primary factors are required in order for caries to occur. These are:**
 - A. an infectious disease caused by oral Host, Substrate, Sugar, Teeth
 - B. *Lactobacilli*, *S. Sanguinis*, *S. mutans* and *P. gingivalis*
 - C. Host, Substrate, Oral Bacteria, Time
 - D. B and C
- 2. The initial stage of biofilm development is formation of a(n) _____ layer.**
 - A. aciduric
 - B. bacterial
 - C. multicellular
 - D. acellular
- 3. Who was the first researcher to link fluoride to caries reduction?**
 - A. Dr. Aristotle Parmly
 - B. Dr. H. Trendley Dean
 - C. Dr. W.D. Miller
 - D. Dr. Frederick McKay
- 4. Saliva helps reduce caries through _____ and _____.**
 - A. abrasion, friction
 - B. oral cleaning, pH buffering
 - C. freshening breath, whitening teeth
 - D. providing fluoride, reducing decay
- 5. One technique for measuring how long the saliva remains below the critical pH for caries after eating is called the _____.**
 - A. Stephan curve
 - B. Ecological plaque hypothesis
 - C. Lactate dehydrogenase pathway
 - D. Late Maturation Stage
- 6. In the Dispersion stage of biofilm development, some bacteria detach from the biofilm in order to _____.**
 - A. fight cavities and gingivitis
 - B. form a reservoir of tartar
 - C. integrate into plaque
 - D. spread and colonize new surfaces in the oral cavity
- 7. The concept of the oral environment being able to cause a shift in dental plaque ecology that can either lead to good health or disease is called the _____.**
 - A. "new plaque hypothesis"
 - B. "ecological plaque hypothesis"
 - C. "dental plaque hypothesis"
 - D. "ecological balance hypothesis"

8. **Classical microbiological methods estimate that plaque contains ~ 800 distinct oral species, with healthy individuals possessing _____ different species at any one time.**
- A. 10-20
 - B. 50-100
 - C. 200-300
 - D. 25-60
9. **Although there are many plaque bacteria, which bacteria are most associated with caries?**
- A. *S. mutans* and *Lactobacilli*
 - B. *S. sanguinis* and *P. gingivalis*
 - C. *P. gingivalis* and *S. mutans*
 - D. *S. oralis* and *P. cavitalis*
10. ***S. mutans* adheres to the tooth biofilm by converting sucrose into an extremely adhesive substance known as _____.**
- A. Glucose – 6 phosphate
 - B. α (1-2) glycoside
 - C. Dispersin B
 - D. Dextran polysaccharide
11. **Which of the following is the more accurate description of caries?**
- A. It is more accurate to consider caries as caused solely by an infectious agent, such as *S. mutans*.
 - B. 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.
 - C. It is more accurate to consider caries as a disease that everyone is likely to get, regardless of what preventive measures are used.
 - D. It is more accurate to consider caries as being a direct result of the amount of fluoride ingested during the developmental stages of the teeth.
12. **What impact does the production of acids by sugar fermentation have on plaque pH?**
- A. It lowers the plaque pH.
 - B. It raises the plaque pH.
 - C. Its impact on plaque pH is dependent on the salivary flow rate of the individual.
 - D. It has no effect on plaque pH.
13. **During the Hydrolysis of Sugar process, extracellular invertase cleaves the energy rich α (1-2) glycosidic bond between which moieties in order to initiate the acid-forming process?**
- A. glucose – 6 phosphate and ATP
 - B. glucose and lactic acid
 - C. xylitol and mannitol
 - D. glucose and fructose
14. **Extracellular polysaccharides make up what percentage of plaque volume?**
- A. 15
 - B. 30
 - C. 60
 - D. 90

15. When a cell becomes a component of a biofilm, it experiences a shift in gene expression, making it up to how many times more resistant to antibodies, antibiotics, and antimicrobials than its planktonic counterparts?
- A. 100
 - B. 1,000
 - C. 10,000
 - D. 25

References

1. National Institute of Dental and Craniofacial Research. Dental Caries (Tooth Decay) in Children Age 2 to 11. Updated July 2018. Accessed June 3, 2021.
2. Dye B, Thornton-Evans G, Li X, Iafolla T. Dental caries and tooth loss in adults in the United States, 2011-2012. NCHS Data Brief. 2015 May;(197):197.
3. Gerabek WE. The tooth-worm: historical aspects of a popular medical belief. *Clin Oral Investig*. 1999 Mar;3(1):1-6. doi: 10.1007/s007840050070.
4. Newbrun E. Sugar and dental caries: a review of human studies. *Science*. 1982 Jul 30;217(4558):418-23. doi: 10.1126/science.7046052.
5. Christen AG. Sumter Smith Arnim, DDS, PhD (1904-1990): a pioneer in preventive dentistry. *J Dent Res*. 1995 Oct;74(10):1630-5. doi: 10.1177/00220345950740100201.
6. Ismail AI, Hasson H, Sohn W. Dental caries in the second millennium. *J Dent Educ*. 2001 Oct;65(10):953-9.
7. Ripa LW. A half-century of community water fluoridation in the United States: review and commentary. *J Public Health Dent*. 1993 Winter;53(1):17-44. doi: 10.1111/j.1752-7325.1993.tb02666.x.
8. American Dental Association. Fluoridation Facts 2018. Accessed June 3, 2021.
9. Filoche S, Wong L, Sissons CH. Oral biofilms: emerging concepts in microbial ecology. *J Dent Res*. 2010 Jan;89(1):8-18. doi: 10.1177/0022034509351812.
10. Marsh PD, Martin M. *Oral Microbiology*, 6th ed. ENew York, NY. Elsevier. 2016.
11. Ochiai K, Kurita-Ochiai T, Kamino Y, Ikeda T. Effect of co-aggregation on the pathogenicity of oral bacteria. *J Med Microbiol*. 1993 Sep;39(3):183-90. doi: 10.1099/00222615-39-3-183.
12. Beighton D. The complex oral microflora of high-risk individuals and groups and its role in the caries process. *Community Dent Oral Epidemiol*. 2005 Aug;33(4):248-55. doi: 10.1111/j.1600-0528.2005.00232.x.
13. Burne RA. Oral streptococci... products of their environment. *J Dent Res*. 1998 Mar;77(3):445-52. doi: 10.1177/00220345980770030301.
14. Marsh PD. Microbial ecology of dental plaque and its significance in health and disease. *Adv Dent Res*. 1994 Jul;8(2):263-71. doi: 10.1177/08959374940080022001.
15. Keijser BJ, Zaura E, Huse SM, van der Vossen JM, Schuren FH, Montijn RC, ten Cate JM, Crielaard W. Pyrosequencing analysis of the oral microflora of healthy adults. *J Dent Res*. 2008 Nov;87(11):1016-20. doi: 10.1177/154405910808701104.
16. Touger-Decker R, van Loveren C. Sugars and dental caries. *Am J Clin Nutr*. 2003 Oct;78(4):881S-892S. doi: 10.1093/ajcn/78.4.881S.
17. Marsh PD. Are dental diseases examples of ecological catastrophes? *Microbiology (Reading)*. 2003 Feb;149(Pt 2):279-294. doi: 10.1099/mic.0.26082-0.
18. Hicks J, Garcia-Godoy F, Flaitz C. Biological factors in dental caries: role of saliva and dental plaque in the dynamic process of demineralization and remineralization (part 1). *J Clin Pediatr Dent*. 2003 Fall;28(1):47-52. doi: 10.17796/jcpd.28.1.yg6m443046k50u20.

Additional Resources

- No Additional Resources Available.

About the Authors

Susan Higham, BSc, PhD, CBIol, MRSB



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.

Email: S.M.Higham@liverpool.ac.uk

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.

Email: ckhope@gmail.com

Sabeel Valappil, BSc, MSc, PhD, PGCertEd, FHEA



Dr. Valappil is a lecturer in dental sciences and Understanding Clinical Practice Year 1 Lead in the School of Dentistry at the University of Liverpool, United Kingdom. He is a Postgraduate Research Lead 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 25 undergraduate and postgraduate project students.

Email: S.Valappil@liverpool.ac.uk

Phil Smith, BDS, MDS, PhD, FDS, DRD, MRD, FDS (Rest Dent) RCS (Edin), FHEA



Phil is currently Honorary Senior Research Fellow and formerly 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 was lead clinician for restorative care of CLP patients in Liverpool and North West (West) Region. He has gained experience in managing clefts from time spent in 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 has acted as external examiner for Universities of Birmingham, Cork, Dundee, Glasgow, Manchester and Newcastle. He was also part of a team from Liverpool commended in the recent Medical Futures Innovation awards and was President of the British Society of Prosthodontics.

Email: P.W.Smith@liverpool.ac.uk

Video Course Presenter

Robert V. Faller, BS



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.

Email: robert.faller@yourencore.com