



# Caries Process and Prevention Strategies: Demineralization/Remineralization



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**CE Credits:** 1 hour

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

**Assistant Students** 

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  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 5 of a 10-part series entitled *Caries Process and Prevention Strategies*. In this course, the dynamic process of demineralization and remineralization is discussed, paying particular attention to tooth hard tissue structure, the role of acid production by cariogenic bacteria, and the critical pH at which tooth enamel begins to dissolve. The role of acid-reducing bacteria, saliva, and fluoride in tooth hard tissue remineralization will also be explained.

#### **Course Contents**

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#### **Overview**

Caries is the chemical dissolution of the hard tooth structures - enamel and dentin - by the acid created as the bacteria in dental plaque ferment carbohydrates. The development of caries is dependent on the interplay between processes that cause demineralization of tooth enamel, and those which cause remineralization: Only when factors favor the high acidity that leads to demineralization does caries occur. In this section, the dynamic process of demineralization and remineralization is discussed, paying particular attention to tooth hard tissue structure, the role of acid production by cariogenic bacteria, and the critical pH at which tooth enamel begins to dissolve. The role of acid-reducing bacteria, saliva, and fluoride in tooth hard tissue remineralization will also be explained.

# **Learning Objectives**

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

- Discuss the difference in how tooth enamel and dentin structure are affected by demineralization.
- Explain the role of bacterial acid production in demineralization.
- Understand the relationship between critical pH and demineralization.
- Identify five zones of carious dentin in the advanced lesion.
- Describe how demineralization affects young children, the elderly, and other special populations.
- Be familiar with the factors that promote remineralization.

#### **Glossarv**

**acidogenic** – Something that produces acid, such as cariogenic bacteria.

**aciduric** – Capable of growth in an acidic environment.

**buffering agent** – Adjusts the pH of any solution such as saliva or plaque fluid and can resist changes in pH. Beneficial in the prevention of dental caries.

**carbonated hydroxyapatite** – The hydroxyapatite in human enamel is not pure, and contains carbonate ions. The presence of carbonate ions makes the enamel structure much more soluble and less resistant to acid dissolution. Chemically, the hydroxyapatite that comprises enamel is often described as a calcium-deficient carbonated hydroxyapatite.

**cariogenic bacteria** – Bacteria present in the oral biofilm of dental plaque that will lead to the occurrence of carious lesions when all other necessary factors are present.

**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.

**fluorapatite** – A crystal structure in tooth mineral ( $Ca_{10}$  ( $PO_4$ )<sub>6</sub>  $F_2$ ) resulting from the replacement of hydroxyl ions (OH-) in the hydroxyapatite structure with fluoride ions (F-). Fluorapatite (also commonly referred to as fluoroapatite, fluorhydroxyapatite or fluorohydroxyapatite) is stronger and more acid resistant than hydroxyapatite.

**GERD** – Gastroesophageal reflux disease; the reflux of hydrochloric acid generated in the stomach that travels to the mouth. Erosion will occur upon the acid's contact with enamel surfaces.

**glycolysis** – Glycolysis is essential to all living organisms, and is the process whereby energy is released from sugars by the formation of pyruvate.

**hydroxyapatite** – Crystals of calcium phosphate –  $(Ca_{10} (PO_4)_6 OH_2)$  that form the mineral structure of teeth and bone. Enamel comprises approximately 98% hydroxyapatite (by weight). Much of the hydroxyapatite in enamel, however, is a calcium-deficient carbonated hydroxyapatite, the crystals of which are readily dissolved by acids. The addition of fluoride creates fluorapatite, which is less soluble and more acid-resistant.

**ions** – Atoms or molecules that carry either a positive or a negative electric charge in a solution. For example, sodium chloride (NaCl, common table salt) in water dissociates into Na+ and Cl– ions.

**pellicle** – A layer of salivary glyco-proteins that forms on the tooth surface and is present within minutes of oral hygiene or professional prophylaxis. The pellicle layer is protective against caries, as it slows the diffusion of calcium and phosphate ions away from the tooth surface. Sometimes referred to as the Acquired Pellicle, it varies in thickness in different parts of the mouth and is reduced during oral hygiene or by dietary acids. In addition to protecting against caries, it is the layer to which microorganisms first attach to the tooth surface in the formation of the dental plaque biofilm.

**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.

**translucent** – Permitting the passage of light; especially: transmitting and diffusing light so that objects beyond cannot be seen clearly.

**white spot lesion** – One of the early clinical signs of dental caries, before cavitation has occurred. The stage at which the disease can be reversed by remineralization.

# **Video: Demin/Remin**



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#### **Course Test Preview**

To receive Continuing Education credit for this course, you must complete the online test. Please go to: <a href="https://www.dentalcare.com/en-us/professional-education/ce-courses/ce372/test">www.dentalcare.com/en-us/professional-education/ce-courses/ce372/test</a>

# 1. Which of the following is true about enamel?

- A. It has a blood and nerve supply.
- B. It contains no pores.
- C. It is comprised mostly of inorganic materials: 95% of it is calcium and phosphate ions combined to make up strong hydroxyapatite crystals.
- D. Water makes up 12% of its composition.

# 2. How is the biological hydroxyapatite of tooth enamel different than pure hydroxyapatite?

- A. It readily incorporates trace minerals, such as fluoride and carbonate into its crystal lattice.
- B. It is stronger.
- C. It has the following formula: Ca<sub>12</sub>(PO<sub>4</sub>)<sub>8</sub>(OH)<sub>4</sub>
- D. A and C

### 3. What differentiates dentin from enamel?

- A. There are no significant differences.
- B. Enamel can repair and regenerate, while dentin cannot.
- C. Unlike enamel, dentin is living tissue with the ability for constant growth and repair, thanks to cells called odontoblasts that create new dentin.
- D. Dentin is harder than enamel.

# 4. What acid is *Streptococcus mutans* capable of metabolizing, and in the process, further promoting demineralization?

- A. lactic acid
- B. acetic acid
- C. pyruvate acid
- D. formic acid

#### 5. Which of the following does NOT effect the rate at which acid is produced in plaque?

- A. The microbial composition of the dental plague.
- B. The density of plaque.
- C. The speed at which bacteria are able to metabolize the dietary carbohydrate.
- D. The number of cavities present.

# 6. What prevents hydroxyapatite from continuously growing out of control?

- A. Hydroxyapatite crystal growth-inhibitors in saliva.
- B. p-rich proteins in saliva that coat enamel to prevent seeding by exposed crystals.
- C. Fluoride prevents seeding by exposed crystals.
- D. A and B

#### 7. What is the effect of sucrose on interdental plaque ion stores?

- A. Frequent sucrose exposure depletes calcium and phosphate reservoirs in plaque.
- B. Sucrose increases calcium stores in interdental plaque.
- C. Sucrose increases fluoride stores in interdental plaque.
- D. Sucrose has little impact on calcium and phosphate reservoirs in plague.

## 8. At what pH does tooth enamel begin to demineralize?

- A. 8.3
- B. 7.5
- C. 5.5
- D. 3.2

# 9. What is the clinical appearance of the initial stage of a carious lesion?

- A. A large cavitation that extends into the dentin.
- B. A chalky white and softened spot on the tooth surface.
- C. Evidence of tooth erosion caused by acid attack.
- D. Completely demineralized tissue.

# 10. How does *Veillonella* bacteria affect the process of demineralization/remineralization?

- A. *Veillonella* use lactate as a substrate, metabolizing it to less acidic products, helping to create an environment that promotes remineralization.
- B. *Veillonella* increases the acidity of plaque, increasing demineralization.
- C. Veillonella causes xerostomia which increases demineralization.
- D. Veillonella attacks pathogenic bacteria, promoting remineralization.

# 11. Which of the following is true about the remineralization of a carious lesion?

- A. Deeper layers of enamel remineralize first and more fully.
- B. Surface layers of enamel remineralize last and completely.
- C. The lesion body in deeper layers of enamel does not remineralize because slow diffusion doesn't allow supersaturation in deeper layers.
- D. B and C

# 12. What is Ostwald ripening?

- A. It is the name given to the maturing of bacteria in interdental plaque.
- B. It is the name given to the maturing of dental enamel.
- C. It is the name given to the process in which small, imperfect hydroxyapatite crystals re-form and grow to reach their maximum size in the presence of a large volume of saturated oral solution.
- D. It is the name given to the regeneration of dentin.

# 13. What is the initial clinical appearance of a remineralized carious lesion?

- A. It appears as a black cavitation.
- B. It appears as a white scar with a shiny, hard surface.
- C. It appears as a brown spot that feels soft and sticky with dental probing.
- D. It appears as a white chalky soft spot that flakes with dental probing.

# 14. Which of the following is a remineralization-promoting characteristic of saliva?

- A. Saliva stimulates odontoblasts to promote enamel remineralization.
- B. Saliva does not promote remineralization.
- C. Saliva is supersaturated with calcium and phosphate ions.
- D. Saliva is slightly acidic, which helps to stimulate the remineralization process.

# 15. Which of the following is true about fluorapatite?

- A. It is not very stable, making it more prone to demineralization.
- B. It binds with calcium, making it less likely that calcium ions are pulled out of the tooth and into the solution.
- C. It can change the critical pH level to 4.5.
- D. B and C

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#### **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.

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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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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.

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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

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# **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

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