



Caries Process, Prevention, and Management: The Host



Course Author(s): Amal A. K. Noureldin, BDS, MSD, MS, PhD

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Conflict of Interest Disclosure Statement

• Dr. Noureldin reports no conflicts of interest associated with this course. She has no relevant financial relationships to disclose.

Caries Process, Prevention and Management consists of a series of ten continuing education courses that may be taken individually or as a complete series. This 2025 newly updated series was first developed in conjunction with the American Dental Education Association in 2008 and has now been comprehensively revised by authors who are members of the board of directors of the American Academy of Cariology (AAC) to reflect the current knowledge and best practices and quality depth of knowledge on the caries process, prevention, and management. Procter & Gamble is delighted to provide this resource enabling high quality educational experiences and an up-to-date understanding of both the science and its relevance to all clinicians in practice and we thank the authors for their contributions. Click here to learn more about the AAC.

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Overview

This is part 3 of a 10-part series entitled Caries Process, Prevention, and Management. It has been established that a host must be present for caries to develop. In this course, three host factors - the tooth, saliva, and the oral cavity's immune response - are introduced, and their roles in the caries process are explained.

Learning Objectives

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

- Discuss tooth structure.
- Describe how the mineral composition and structure of enamel relates to caries.
- Be familiar with the concept of enamel maturation.
- Explain what saliva is and how it is produced.
- Identify the major salivary glands.
- Explain the nerve control of saliva secretion.
- List the physical, chemical, and antibacterial properties of saliva.
- Describe the host's immune response in the dental caries process.

Glossary

ameloblasts – The cells in the embryonic tooth germ that produce enamel. Once tooth formation is complete, the ameloblasts are unable to produce further enamel. Therefore, if enamel is lost or damaged by caries, tooth wear, or trauma, it cannot be repaired or replaced.

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.

fluorapatite – A crystal structure in tooth mineral (Ca10 (PO4)6 F2) 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.

hydroxyapatite – Crystals of calcium phosphate - Ca10 (PO4)6 OH2 - 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.

parasympathetic nerves – The part of the nervous system that controls and regulates various organs and glands unconsciously, such as the secretion of salivary and lachrymal fluids.

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.

sympathetic nerves – The part of the nervous system that controls the stress and fight-or-flight response. It controls the force of contraction and rate of the heartbeat, and dilates the pupils and the bronchioles.

Introduction

Caries cannot develop without the presence of a host, which comprises tooth structure, the saliva that surrounds a tooth, and the immune responses of saliva and plasma in the oral cavity. In the caries process, particular attention is paid to the enamel - the hard, outermost layer - because it is the primary contact with cariogenic bacteria, and where demineralization first begins. It is also the only tissue of the tooth that does not have the ability to grow or repair itself after maturation. Saliva is also important to consider in the caries process because it has protective properties that can reduce caries risk: it neutralizes pH in the vicinity of the tooth, assists in remineralization, acts as an antibacterial agent, and plays a role in the immune responses to cariogenic oral bacteria.

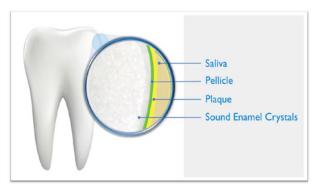


Figure 1. Tooth Surface

Clinical Significance Snapshots

In managing patients at risk of dental caries, how should I approach managing 'host' factors involved in the caries process?

To manage host factors in patients at risk of dental caries, focus should be on protecting enamel and dentin. In younger individuals, enamel is the primary concern, while dentin becomes vulnerable later in life due to gingival recession and root exposure.

Saliva's Role

Saliva plays a critical role by flushing away cariogenic substances and promoting remineralization through its calcium saturation. Reduced saliva flow, often caused by medications or medical conditions, increases caries risk. Dental professionals should assess saliva quantity and quality and collaborate with the patient's physician if concerns arise.

Fluoride Protection

Regular exposure to fluoride is essential. Brushing twice daily with fluoride toothpaste can reduce caries by over 50%. Additional protection can be achieved through fluoride rinses, gels, and professional applications. Preventing gingival recession is key to protecting roots from caries. For exposed roots, fluoride treatments and patient education on meticulous oral hygiene are crucial.

The Tooth

The four major tissues of the teeth are enamel, dentin, dental pulp, and cementum (Figure 2).

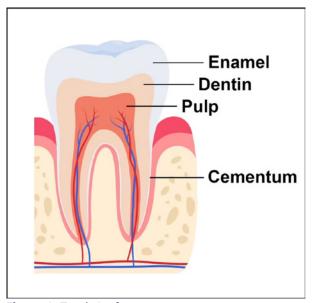


Figure 2. Tooth Surface

Enamel Structure and Composition

Enamel is the most mineralized tissue of the body, forming a very hard, thin, translucent

layer of calcified tissue that covers the entire anatomic crown of the tooth. It can vary in thickness and hardness on each tooth, from tooth to tooth and from person to person. It can also vary in color (typically from yellowish to grayish white) depending on variations in the thickness, quality of its mineral structure and surface stains. Enamel has no blood or nerve supply within it. It is enamel's hardness that enables teeth to withstand blunt, heavy masticatory forces. Enamel is so hard because it is composed primarily of inorganic materials: roughly 95% to 98% of it is calcium and phosphate ions that make up strong hydroxyapatite crystals. Yet, these are not pure crystals, because they are carbonated and contain trace minerals such as strontium, magnesium, lead, and fluoride. These factors make "biological hydroxyapatite" more soluble than pure hydroxyapatite. 1-3

Approximately 1% to 2% of enamel is made up of organic materials, particularly enamel-specific proteins called enamelin, which have a high affinity for binding hydroxyapatite crystals. Water makes up the remainder of enamel, accounting for about 4% of its composition.

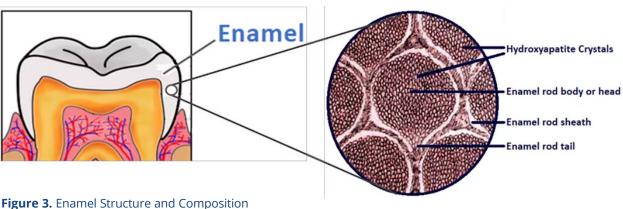
The inorganic, organic, and water components of enamel are highly organized: millions of carbonated hydroxyapatite crystals are arranged in long, thin structures called rods that are 4 µm to 8 µm in diameter. It is estimated that the number of rods in a tooth ranges from 5 million in the lower lateral incisor to 12 million in the upper first molar. In general, rods extend at right angles from the dento-enamel junction (the junction between enamel and the layer below it

called dentin) to the tooth surface. Surrounding each rod is a rod sheath made up of a protein matrix of enamelins. The area in between rods is called interrod enamel, or interrod cement. While it has the same crystal composition, crystal orientation is different, distinguishing rods from interrod enamel.¹⁻³

Minute spaces exist where crystals do not form between rods. Typically called pores, they contribute to enamel's permeability, which allows fluid movement and diffusion to occur, but they also cause variations in density and hardness in the tooth, which can create spots that are more prone to demineralization – the loss of calcium and phosphate ions – when oral pH becomes too acidic and drops below 5.5. In demineralization, the crystalline structure shrinks in size, while pores enlarge.^{3,4}

Enamel is formed by epithelial cells called ameloblasts. Just before a tooth erupts from the gums, the ameloblasts are broken down, removing enamel's ability to regenerate or repair itself. This means that when enamel is damaged by injury or decay, it cannot be restored beyond the normal course of remineralization. When a tooth erupts, it is also not fully mineralized. To completely mineralize the tooth, calcium, phosphorous, and fluoride ions are taken up from saliva to add a layer of 10 µm to 100 µm of enamel over time.³

There are conditions that can affect the formation of enamel and thus increase the risk of caries. These include the genetic disorder amelogenesis imperfecta, in which enamel is



never completely mineralized and flakes off easily, exposing softer dentin to cariogenic bacteria.4 Other conditions are linked with increased enamel demineralization, such as gastroesophageal reflux disease (GERD) and celiac disease.^{5,6}

Dentin Structure and Composition

Dentin is a hard, light yellow, porous layer of tissue directly underneath enamel and cementum. Dentin constitutes the largest portion of the tooth and consists of approximately 70% inorganic matter and 30% organic matter and water. Its organic matter is calcium and phosphate ions that form hydroxyapatite crystals as in enamel, but the crystals are 30 times smaller, making dentin somewhat softer than enamel.¹

Another way in which dentin is different than enamel is that it is living tissue with the ability for constant growth and repair. Tiny dentinal tubules that run between the cementoenamel junction (the interface of crown enamel and the tooth root cementum) and the pulp layer beneath it assist in this regeneration process. Cell processes in the pulp layer reach into the tubules, creating new dentin and mineralizing it. Nerves also pass through these dentinal tubules allowing dentin to transmit pain, unlike enamel.¹

Pulp Chamber

The dental pulp is the soft tissue of the tooth. The chamber containing the dental pulp that underlies, and is connected to, the dentin layer is called the pulp chamber. It contains the coronal pulp within the crown that is continuous with the radicular pulp within the root. Pulp contains odontoblasts, cells whose biological function is the creation of dentin.7 It also contains neurons, vascular tissues, fibroblasts and macrophages. (It was long thought that pulp contained lymphatic vessels but a 2010 immunohistochemical staining study found that, under normal conditions, dental pulp does not contain true lymphatic vessels.)8 A recent 2022 study found a moderate correlation between pulp inflammation and the formation of new blood and lymphatic vessels.9

One area of note is the apical foramen at the end of the radicular pulp. Blood vessels, nerves, and connective tissue pass through this area to reach the interior of the tooth.

Cementum

This is a thin, light yellow layer of bonelike tissue that covers the roots of the teeth. Its main function is to anchor teeth to the bony walls of the tooth sockets in the periodontium by attaching to the periodontal ligament. The

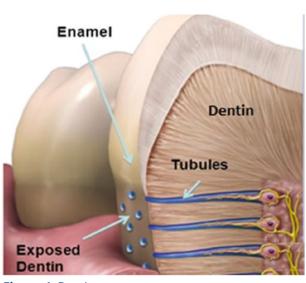


Figure 4. Dentin

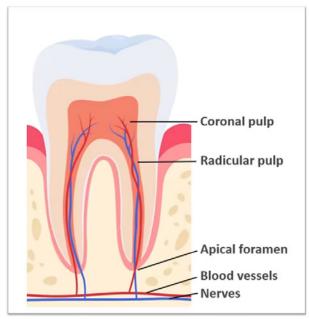


Figure 5. Pulp Chamber

cementum is composed of approximately 55% organic material and 45% inorganic material, mainly calcium salts. It joins the enamel at the cervix of the tooth at the cementoenamel junction. In most teeth, the cementum overlaps the enamel for a short distance; in some, enamel meets the cementum in a sharp line; and in a few teeth, there is a gap between the enamel and the cementum, exposing a narrow area of root dentin. Such areas may be very sensitive to thermal, chemical, or mechanical stimuli. Cementum is formed continuously to make up for the loss of tissue due to wear, and to allow for the attachment of new fibers of the periodontal ligament to the surface of the root.^{9,10}

Saliva

Saliva is a complex biological fluid composed of both mucous and serous secretions, which are pale yellow, transparent, and physiologically benign. It is primarily produced by the salivary glands, whose anatomical structure and functional mechanisms play a crucial role in maintaining oral homeostasis. Beyond its role in facilitating oral lubrication, saliva is an essential component in the prevention and modulation of oral diseases, particularly dental caries.

Saliva contains a diverse array of biochemical constituents, including calcium, phosphate, fluoride, proteins, antibacterial agents, lipids, and buffering compounds, each of which contributes to its protective and reparative functions. One of its primary mechanisms in caries prevention is its buffering capacity, which helps neutralize acids produced by bacterial metabolism. This acid neutralization is critical in mitigating enamel demineralization. Additionally, saliva serves as a reservoir of calcium and phosphate, essential ions that not only slow the dissolution of enamel minerals but also facilitate remineralization processes. Notably, fluoride efficacy in enhancing enamel remineralization is contingent upon the availability of calcium and phosphate within the oral environment.

Furthermore, saliva exhibits a robust antimicrobial function, containing multiple antibacterial agents that inhibit bacterial proliferation and modulate the oral microbiome. In conditions where salivary flow is significantly reduced or compromised, such as in xerostomia, the absence of these protective factors results in a heightened susceptibility to

dental caries (10-20 times as great as a normal individual), even in the presence of fluoride treatment. This underscores the indispensable role of saliva in maintaining oral health and preventing the pathological consequences of bacterial activity.¹¹

Anatomy of Salivary Glands

These are made up of an acinus (a berry-shaped cluster of excretory cells) and ductal systems. Saliva is formed in the acini with the serous (watery) secretion formed by serous cells, and the mucous (viscous) secretion formed by the mucous cells. There are three major bilateral salivary glands: the parotid, the sublingual and the submandibular(Figures 7-8).^{1,12}

The parotid is the largest pair of glands, occupying the parotid facial space, an area posterior to the mandibular ramus, and anterior and inferior to the ear. It secretes saliva through the Stensen's ducts into the oral cavity to facilitate mastication and swallowing. The submandibular are a pair of glands located beneath the lower jaws. Even though they are smaller than the parotid, they produce 70% of saliva, secreted via the Wharton's ducts. These two glands produce "true" saliva – the combination of serous fluid and mucous. They also have acini connected to intercalated ducts (essentially, transitional tissue) which then connect to striated ducts that have the capacity to modify the mineral content of saliva. On the other hand, the third major set of glands – the sublingual glands which are located beneath the tongue – secretes mainly mucous, and does not have striated ducts, releasing its mucous directly from acini via excretory ducts.^{1,12}

Innervation of Salivary Glands

The salivary glands are innervated by the **parasympathetic** and **sympathetic** branches of the autonomic nervous system.

Parasympathetic stimulation favors serous (watery) secretion and occurs via cranial nerves, with the glossopharyngeal nerve innervating the parotid, and the facial nerve innervating the submandibular and the sublingual glands. These release acetylcholine and substance P, neurotransmitters that bind to receptors on acinar and ductal cells of the salivary glands.^{1,13}

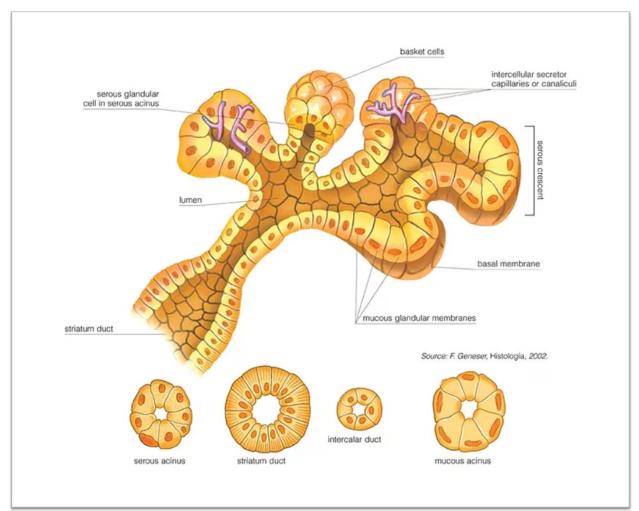


Figure 6. Anatomy of Salivary Gland. Image source: F. Geneser, Histologia, 2002

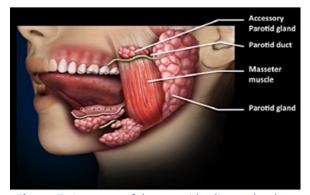


Figure 7. Anatomy of the parotid salivary glands.

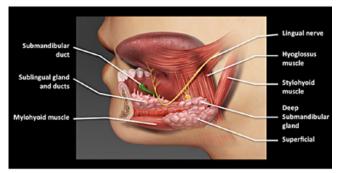


Figure 8. Anatomy of the sublingual and the submandibular salivary glands. Images used with permission from AEGIS Communications, LLC.

Direct sympathetic stimulation favors viscous (mucoid) secretion and takes place via preganglionic nerves in the thoracic segment of the spinal cord which synapse with postganglionic neurons. These release the neurotransmitter norepinephrine that binds to receptors on the salivary gland acinar and ductal cells. There is also indirect sympathetic stimulation of the salivary glands via innervation of the blood vessels that supply the glands.^{1,13}

With both types of stimulation, the binding of neurotransmitters to salivary gland receptors leads to increases in intracellular calcium and alterations in membrane permeability, and a corresponding increase of saliva, as organic molecules, electrolytes, water and mucus are excreted into the acinar lumen.^{1,13}

Host Protective Properties of Saliva

It has been established that saliva plays a crucial role in reducing caries risk. This is due in large part to saliva's **physical**, **chemical** and **antibacterial** properties.

Physical Protective Qualities

Due to its water content and flow rate, saliva physically cleanses the oral cavity of food and debris. 14 Unstimulated flow rates are approximately 0.3 to 0.4 ml/min This rate decreases to 0.1 ml / min during sleep. While stimulated flows are approximately 1.5 to 2 ml/min, although there is great variability in individual salivary flow rates. 10

Most humans produce roughly 0.5 to 1 liter of saliva per day with 90% secreted from the major glands. Saliva also dilutes and removes organic acids from dental plaque.¹²

Chemical Protective Qualities

Saliva contains a number of electrolytes and organic molecules that minimize decreases in local pH, creating an environment that favors remineralization. For example, sodium bicarbonate and phosphates, along with other salivary components, act as buffers or neutralizing agents in saliva. In addition, one salivary protein called sialin tends to raise salivary pH to neutral levels.

Saliva is also supersaturated with hydroxyapatite, fluorapatite, and calcium and phosphate ions compared to the carbonated hydroxyapatite in enamel. This supersaturation is maintained by the proline-rich proteins and statherins in saliva, and it increases the likelihood of remineralization via the incorporation of calcium and phosphate into enamel.^{10,12}

Antibacterial Properties

Saliva contains several proteins with different types of antibacterial properties: The **mucins** are sulfated glycoproteins that trap, aggregate, and clear bacteria. The enzymes called **amylases** break down food particles that stick to teeth, reducing the bacterial build-up that can lead to decay.

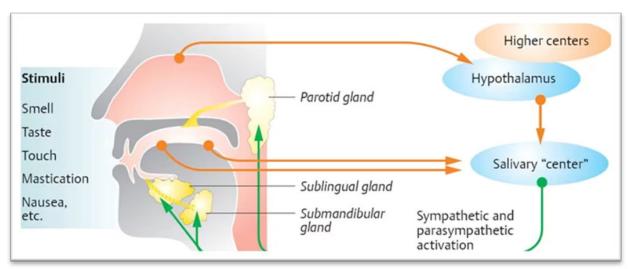


Figure 9. Innervation of Salivary Glands.

Image source: Gastrointestinal Secretion - Physiology - An Illustrated Review (doctorlib.info)

Lysozyme is a cationic protein that lyses (damages) the cell walls of bacteria, rendering them inactive. Lactoferrin is an iron-binding glycoprotein that deprives bacteria of energygenerating iron so that they cannot survive.

Peroxidase enzyme is a protein that forms free radical compounds in bacterial cells which cause them to self-destruct. Is Immunoglobulins are antigen-binding proteins that block the adherence of bacteria to the tooth surface and/or promote the clearance of bacteria from tooth structure.

Immune Response

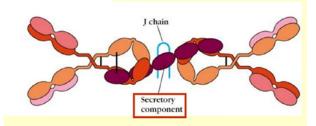
Saliva plays a pivotal role in the health of the mouth and contributes to host defense mechanisms through physio-chemical barriers, non-specific (innate immunity) factors, and specific (adaptive immunity) factors. Of key importance are the specific factors, which include the immunoglobulins that increase in number when there is exposure to cariogenic bacteria.

Secretory Immunoglobulin A (IgA)

This is the dominant immunoglobulin in the healthy mouth.¹ SigA is produced by gland-associated immunocytes that are scattered in acini and in clusters adjacent to salivary ducts. SigA is composed of two molecules of heavy and light chains, a secretory component that protects the immunoglobulin from being degraded by proteolytic enzymes, and a J chain. This is a unique joining chain not found in any other immunoglobin that connects the two IgA molecules into a dimeric structure.¹6

SigA can agglutinate oral bacteria, such as S. mutans, modulate enzyme activity, and inhibit the adherence of bacteria to the buccal epithelium and to enamel.¹⁷⁻¹⁹ It does well at interfering with the initial colonization of caries-associated microflora on the tooth surface, but being a salivary protein, it does not always have access to bacteria that are deeper in periodontal pockets. SigA is also a poor activator of the complement system, the biochemical cascade that helps antibodies physically clear pathogens. It is also a poor opsonizer that does not reliably make bacterial cells susceptible to phagocytosis. A recent study identified sIgA as a potential biomarker for early childhood caries, demonstrating high sensitivity and specificity.20

Secretory IgA



Dimers and tetramers in secretion with a secretory component

Figure 10. Secretory Immunoglobulin A (IgA)

Image Source: PPT - Chapter 4 Immunoglobulins: Structure and Function PowerPoint Presentation -ID:4524596 (slideserve.comage from: PPT - Chapter 4 Immunoglobulins: Structure and Function PowerPoint Presentation - ID:4524596 (slideserve.com)

Immunoglobulin G (IgG)

This type is almost entirely derived from gingival crevicular fluid, now more commonly called gingival fluid. This is a blood exudate (a protein-rich fluid that has escaped from blood vessels) that emerges from the crevice between the gingiva and the tooth. It contains immunoglobulins such as IgG, which can be produced by plasma in periodontal pockets. An increase in gingival fluid and IgG to a variety of oral microbial agents, including S mutans, has been seen in response to periodontal irritation and inflammation. Yet, IgG to oral microbial agents are also present in the healthy mouth, suggesting that it prevents early stages of plaque development from worsening.²¹ Compared to SigA, IgG is strong in complement-activating and opsonizing that can lead to antibodymediated phagocytosis. In the absence of inflammation, the naturally low levels of complement would reduce IgG, and may play a role in modulating oral microflora.²²

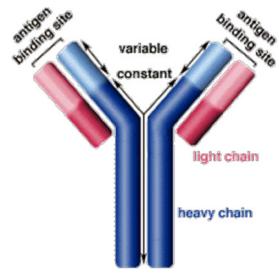


Figure 11. Immunoglobulin G (IgG)

Conclusion

A primary factor in the development of caries is the presence of a host. A number of host-related subfactors can influence the caries process. These include tooth structure, the physical, chemical, and antibacterial properties of the saliva that surrounds the tooth, and the immunity conferred by the immunoglobulins in the host environment, the mouth. Understanding tooth anatomy and the composition of all the tooth tissues, the protective role of saliva, and the two main different types of immune responses by secretory Immunoglobulin A and Immunoglobulin G will help dental professionals reduce caries risk in their patients.

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/ce-courses/ce712/start-test

1. Which of the following is not a major tissue of teeth?

- A. Enamel
- B. Hydroxyapatite crystals
- C. Pulp
- D. Cementum

2. Which of the following is true about enamel?

- A. It has a blood and nerve supply.
- B. It contain no pores.
- C. Its hydroxyapatite crystals are highly organized.
- D. Water makes up 12% of its composition.

3. When a tooth erupts, it is not fully mineralized. Which ions are taken up from saliva to complete mineralization?

- A. Calcium
- B. Phosphorous
- C. Fluoride
- D. All of the above

4. What are the main differences between dentin and enamel?

- A. Dentin has more organic matter, dentin is softer, and dentin is living tissue that can grow and repair.
- B. Dentin has more organic matter, dentin's hydroxyapatite crystals are smaller, and enamel can be repaired and regenerated.
- C. There are no significant differences.
- D. Enamel contains tubules that connect it to pulp, dentin is harder, and enamel has more inorganic matter.

5. Which of the following is true about pulp?

- A. It is formed by epithelial cells called ameloblasts.
- B. One of its main roles is the creation of dentin.
- C. It is devoid of blood vessels and nerves.
- D. It is directly connected to enamel via tubules.

6. What is the main role of cementum?

- A. To protect against tooth sensitivity.
- B. To create dentin.
- C. To anchor teeth to the body walls of tooth sockets.
- D. None of the above.

7. Which of the following are major salivary glands?

- A. Parotid
- B. Submandibular
- C. Wharton's ducts
- D. A and B

8. Which of the following traits differentiates the sublingual salivary glands from the parotid and submandibular glands?

- A. The sublingual glands secrete mostly mucous and have excretory ducts.
- B. The sublingual glands secrete mostly serous fluid and have excretory ducts.
- C. The sublingual glands produce thicker saliva and have intercalated ducts.
- D. There is no difference between sublingual glands and parotid and submandibular glands.

9. Salivary glands are innervated by the parasympathetic and the sympathetic branches of the autonomic nervous system. Which of the following pairings is correct for nervous control and secretion?

- A. Parasympathetic stimulation favors mucoid secretion.
- B. Parasympathetic stimulation favors serous secretion.
- C. Direct sympathetic stimulation favors serous secretion.
- D. Indirect sympathetic stimulation favors serous secretion.

10. Which of the following represents the physical role of saliva?

- A. Cleanses the oral cavity.
- B. Dilutes and removes organic acids from dental plaque.
- C. Aids in digestion.
- D. A and B

11. In humans, major and minor salivary glands secrete approximately what volume of saliva each day?

- A. 5 liters
- B. 2 liters
- C. 1 liters
- D. 0.01 liters

12. Which of the following acts as pH neutralizing agents in saliva?

- A. Sodium bicarbonate, phosphates, and sialin
- B. Statherins, phosphates, and calcium
- C. Sodium bicarbonate, hydroxyapatite, sialin
- D. All of the above.

13. Which of the following is an antibacterial property of saliva?

- A. Mucins that trap, aggregate, and clear bacteria.
- B. Lysozyme that deprives bacteria of iron.
- C. Lactoferrin that activates bacterial clumping.
- D. Salivary peroxide that triggers hydrogen peroxides.

14. The increase of which of the following results from an exposure to cariogenic bacteria?

- A. Plasma cells
- B. Antigens
- C. Immunoglobulin
- D. Opsonizes

15. Which of the following correctly describes secretory immunoglobin A (slgA)?

- A. slgA is produced in salivary ducts and primarily inhibits adherence of bacteria.
- B. slgA is produced in gingival fluid and triggers phagocytosis.
- C. slgA is produced only in gingival fluid and inhibits adherence of bacteria.
- D. slgA is made of three molecules of light and heavy chains.

References

- 1. Jacobson A. Oral development and histology. 3rd edition. New York: Thieme Medical Publishers, Inc. 2002.
- 2. Fincham AG, Moradian-Oldak J, Simmer JP. The structural biology of the developing dental enamel matrix. J Struct Biol. 1999 Jun 30;126(3):270-299.
- 3. Robinson C, Brookes SJ, Shore RC, Kirkham J. The developing enamel matrix: nature and function. Eur J Oral Sci. 1998 Jan;106 Suppl 1:282-291.
- 4. Simmer JP, Hu JC. Dental enamel formation and its impact on clinical dentistry. J Dent Educ. 2001 Sep;65(9):896-905.
- 5. Lazarchik DA, Filler SJ. Effects of gastroesophageal reflux on the oral cavity. Am J Med. 1997 Nov 24;103(5A):107S-113S.
- 6. Cheng J, Malahias T, Brar P, Minaya MT, Green PH. The association between celiac disease, dental enamel defects, and aphthous ulcers in a United States cohort. J Clin Gastroenterol. 2010 Mar;44(3):191-194.
- 7. Arana-Chavez VE, Massa LF. Odontoblasts: the cells forming and maintaining dentine. Int J Biochem Cell Biol. 2004 Aug;36(8):1367-1373.
- 8. Gerli R, Secciani I, Sozio F, Rossi A, Weber E, Lorenzini G. Absence of lymphatic vessels in human dental pulp: a morphological study. Eur J Oral Sci. 2010 Apr;118(2):110-117.
- 9. Wiśniewska K, Rybak Z, Szymonowicz M, Kuropka P, Kaleta-Kuratewicz K, Dobrzyński M. Detection of lymphatic vessels in dental pulp. Biology (Basel). 2022 Apr 21;11(5):635.
- 10. Diekwisch TG. The developmental biology of cementum. Int J Dev Biol. 2001 Sep;45(5-6):695-706.
- 11. Song EC, Chung SH, Kim JH. Molecular mechanisms of saliva secretion and hyposecretion. Eur J Oral Sci. 2024 Apr;132(2):e12969.
- 12. du Toit DF, Nortjé C. Salivary glands: applied anatomy and clinical correlates. SADJ. 2004 Mar;59(2):65-6, 69-71, 73-74.
- 13. Proctor GB, Carpenter GH. Regulation of salivary gland function by autonomic nerves. Auton Neurosci. 2007 Apr 30;133(1):3-18.
- 14. Tenovuo J. Antimicrobial function of human saliva--how important is it for oral health? Acta Odontol Scand. 1998 Oct;56(5):250-256.
- 15. Fenoll-Palomares C, Muñoz Montagud JV, Sanchiz V, et al. Unstimulated salivary flow rate, pH and buffer capacity of saliva in healthy volunteers. Rev Esp Enferm Dig. 2004 Nov;96(11):773-783.
- 16. Gorr SU. Antimicrobial peptides of the oral cavity. Periodontol 2000. 2009;51:152-180.
- 17. Marcotte H, Lavoie MC. Oral microbial ecology and the role of salivary immunoglobulin A. Microbiol Mol Biol Rev. 1998 Mar;62(1):71-109.
- 18. Johansen FE, Braathen R, Brandtzaeg P. Role of J chain in secretory immunoglobulin formation. Scand J Immunol. 2000 Sep;52(3):240-248.
- 19. Tyler BM, Cole MF. Effect of IgA1 protease on the ability of secretory IgA1 antibodies to inhibit the adherence of Streptococcus mutans. Microbiol Immunol. 1998;42(7):503-508.
- 20. Hertel S, Hannig M, Hannig C, Sterzenbach T. Mucins 5B and 7 and secretory IgA in the oral acquired pellicle of children with caries and caries-free children. Arch Oral Biol. 2022 Feb;134:105314.
- 21. Reinhardt RA, McDonald TL, Bolton RW, et al. IgG subclasses in gingival crevicular fluid from active versus stable periodontal sites. J Periodontol. 1989 Jan;60(1):44-50.
- 22. Mousavizadeh A, Afroozi B, Hadinia F, Azarshab M, Hadinia A. The relationship between salivary and serum IgA and IgG levels and dental caries in adults. Clin Lab. 2021 Aug 1;67(8).

Additional Resources

No Additional Resources Available

About the Author



Amal A. K. Noureldin, BDS, MSD, MS, PhD

Dr. Noureldin BDS, MSD, MS, PhD, tenured Clinical Professor and Director of Cariology and Prevention in Department of Public Health Sciences School of Dentistry Texas A&M, specializes in three areas of dentistry, operative dentistry, preventive dentistry and cariology (caries management). She holds two master's degrees—one in Operative Dentistry from Cairo University (1998) and another in Biomaterials from Baylor College of Dentistry, Texas A&M University (2003). Additionally, she earned her PhD in Operative Dentistry in 2007 from Cairo University. This extensive education makes her an expert in the field of cariology.

Dr. Noureldin is a distinguished dentist, scholar, mentor, and educator, teaching at both the predoctoral and postdoctoral levels. Her research focuses on dental caries management and white spot lesion prevention and treatment, supported by industry contracts and intramural grants. She has published extensively in peer-reviewed journals. Dr. Noureldin serves on the Board of Directors for the American Academy of Cariology, is Vice President of Cariology Research Section of the International Association for Dental research (IADR) and is a member of the American Heart Association's Healthy Smiles, Healthy Hearts Scientific Advisory Group. She is also a national and international speaker. Her leadership and accomplishments have been recognized with the Distinguished Teaching Excellence Award (2016) and the Clinical Faculty Research Award (2019) from Texas A&M College of Dentistry.

Email: anoureldin@tamu.edu