

Dental Caries: A pH-mediated disease

Learning Outcomes:

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

- Discuss the history of dental caries research.
- Compare and contrast the evolution of the plaque hypotheses theories.
- Explain the role of saliva and salivary glands in the dental caries process.
- Differentiate between salivary resting pH, stimulated pH and buffering capacity.
- Formulate appropriate patient recommendations to prevent, treat, and maintain oral health related to dental caries.

Introduction

Despite advancements in oral disease science, dental caries continues to be a worldwide health concern, affecting humans of all ages, especially children where caries disease is on the rise.¹ According to a recent national study, dental caries continues to affect a large number of Americans in all age groups, with tooth decay in primary teeth increasing among children aged 2-5 years.² This National Health and Nutrition Examination Survey (1999-2004) reported that 42% of children, aged 2-11, have had carious lesions in their primary teeth and 21% of children, aged 6-11, have had carious lesions in their permanent dentition. Approximately 59% of adolescents, aged 12-19, have experienced dental caries and by adulthood, aged 20-75+, well over 92% of those surveyed have experienced dental caries in their permanent dentition. These statistics demonstrate that although it is not always obvious in an exam (a snapshot in time), the war on tooth decay is being lost as we age. Current science demands that dental caries be treated as a disease, rather than with tooth repair alone. While it may be true tooth decay is not as rampant as it once was, the fact the disease still exists demands our attention as health professionals.

Dental caries is an infectious bacterial biofilm disease which is expressed in a predominantly pathologic oral environment.³⁻⁴ Although acid generating bacteria are the etiologic agents, dental caries has been thought of as multifactorial since it is influenced by dietary and host factors as well.⁵ In addition, the role of saliva as a defense system against dental caries is well documented. These defense systems include clearance, buffering, antimicrobial agents, and calcium and phosphate delivery for remineralization to name a few.⁶ The caries process is dependent upon the interaction of protective and pathologic factors in saliva and plaque biofilm as well as the balance between the cariogenic and noncariogenic microbial populations that reside in saliva. A brief historical review of important concepts and scientific developments regarding dental caries will enhance the understanding of the dental caries process.

Dental Caries

The word *caries* derives from the Latin for *rot* or *rotten*. The earliest theory was the “tooth worm theory” proposed by the ancient Chinese in 2500 BC, where it posited a toothworm as the cause of this rotteness. In 350 BC Aristotle observed figs and sweets caused tooth decay and by the 12th century, caries was described as the condition of having holes in the teeth—or cavities. Treatment of cavities was based on extraction of the teeth or the use of home remedies such as plugging the hole with tobacco ash and other questionable materials.⁷ In 1728, Pierre Fauchard, a French military surgeon, wrote the first text on dental diseases and treatment entitled, “Le Chirurgien Dentiste.” Fauchard dispelled the toothworm theory and asserted that cavities were caused by enamel erosion, advocating excavation of the cavity using dental instruments and filling the area with gold foil, lead or tin.⁸⁻⁹ Dentistry emerged as a separate discipline from medicine in the mid 19th century and theories regarding the etiology of dental caries developed. In 1881, Drs. Miles and Underwood proposed the development of dental caries was dependent upon microorganisms entering the dentinal tubules and destroying the organic components of dentin, leaving the inorganic dentin to be washed away by the fluids in the mouth.¹⁰⁻¹¹

In 1881, Dr. Willoughby D. Miller published his chemoparasitic theory of dental caries.¹² According to this theory, microorganisms metabolize fermentable carbohydrates and acids are produced. These acids then breakdown or demineralize tooth enamel, the first step in dental caries. Although his theory is still relevant today, Dr. Miller failed to identify plaque biofilm as the source of the bacteria and subsequent bacterial acids. It was not until 1954 that researchers were able to conclusively prove that bacteria, not fermentable carbohydrates, were indeed the culprit in the acid production and subsequent demineralization of the tooth structure.¹³ Orland and colleagues were able to demonstrate that germ-free rats, when fed a cariogenic diet, did not develop dental caries. After cariogenic bacteria were introduced into the rat’s environment along with a cariogenic diet, carious lesions did develop.

The association of a given bacterial species with disease historically has been through the application of Koch’s Postulates. These criteria were formulated by Robert Koch in 1884 and refined and republished by Koch in 1890.¹⁴ The criteria are as follows:

1. A specific organism can always be found in association with a given disease.
2. The organism can be isolated and grown in pure culture in the laboratory.



Ivory carving, Southern France, 18th Century

Continued on Page 10

3. The pure culture will produce the disease when inoculated into a susceptible healthy animal.
4. It is possible to recover the organism in pure culture from the experimentally infected animal.

Applying Koch's Postulates, the transmissibility of the dental caries infection was first shown by Fitzgerald and Keyes in 1960 when they caged caries-inactive hamsters with caries-active hamsters and the result was dental caries in all caged animals.¹⁵ Interestingly, neither Robert Fitzgerald or Paul Keyes identified the cariogenic bacteria that was transmitted as *Mutans streptococci* (MS) or *Lactobacilli* (LB) species. It was not until 1968 that scientists cogently argued the caries conducive streptococci of Fitzgerald and Keyes were, indeed, the *Streptococcus mutans*.¹⁶ In addition, cross sectional studies have shown individuals with high rates of dental caries tend to harbor higher levels of MS and LB than those individuals that are caries free.¹⁷⁻¹⁸ Recently, there have been many other bacteria identified as being associated with dental caries which have demonstrated acid generating capabilities.⁴ Over the years, several plaque hypotheses have been proposed to explain the etiology of dental caries. A review of the different plaque hypotheses, as it relates to dental caries, will summarize current theory.

Plaque Hypotheses Theories

The *Non-Specific Plaque Hypothesis* purports the caries disease is an outcome of the overall activity of the total plaque microflora and not a specific organism.¹⁹ In this, everybody is treated alike since everybody has plaque and since plaque is forming all the time. Successful treatment to remove plaque biofilm would have to be universally and continuously applied to everyone.²⁰

In contrast, the *Specific Plaque Hypothesis* proposes that among the diverse collection of bacteria encompassing the plaque microflora, only a



Streptococcus mutans

few species of bacteria are involved in the disease.^{5,17,21} The plaque per se is not pathogenic, but the presence of pathogenic species, such as MS and LB within the plaque causes dental caries. MS is a group of bacteria, rather than one species and is composed of several streptococcus species. However, only two of these, *S.*

mutans and *Streptococcus sobrinus* are found in humans. These MS species all share the same phenotypic characteristics, but are not identical.²² MS is part of the normal oral flora, however, under certain conditions, it will become dominant to cause dental caries.²³ LB constitute an acidogenic and aciduric group of microorganisms associated with dental caries, but they are not considered essential in the formation of the carious lesion. LB are found in the deep parts of the carious lesion and are now considered secondary and are more involved

in the progression of the already established lesion.²⁴⁻²⁷ In this hypothesis model it makes sense to target the causative agents such as MS and LB by culturing (or some other metric to identify them) and applying antibacterial therapy to reduce or eliminate them. In theory, the search for a cure by way of a specific dental vaccine aimed at targeting specific etiologic agents could be possible.

Ecological Plaque Hypothesis differs from the specific plaque hypothesis in that pathogens (MS and LB) can be present, but in too low numbers to cause disease. Disease will result only when there is a shift in the homeostatic balance of the resident microflora due to a change in local environmental conditions (such as pH) which favor the growth of pathogens.²⁸ Research has demonstrated it is the pH, not the sugar, which causes the pathologic shifts of biofilms.²⁹ Disease prevention should focus on maintenance of a normal, health-associated ecosystem and not solely on the inhibition of pathogens. Interventions which can shift the environmental factors that drive selection and enrichment for the bacteria are key prevention under this model (Figure 1).

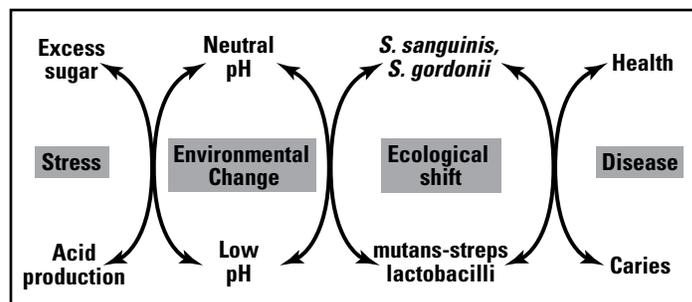


Figure 1: Ecological Plaque Hypothesis. Adapted with permission from Marsh (1994)

Extended Ecological Plaque Hypothesis goes one step further and purports that in the presence of low pH, the non-MS bacteria and the normally non-pathogenic *Actinomyces spp.* bacteria, can adapt to produce acid which then destabilizes the homeostatic biofilm causing a shift to a more overall acidogenic plaque biofilm. MS and LB can then predominate at a lower pH.⁴

Caries Transmission

Much has been written on the transmissibility of the dental caries infection. Humans are not born with cariogenic bacteria, but it has been shown that transmission of MS is from caregivers, usually mothers, by mouth-to-mouth transmission via kissing or by sharing a spoon during feeding.^{3,30} Studies support that primary colonizers, the first bacteria to adhere to the teeth, determine the eventual pathogenicity of the plaque biofilm.³¹ Once the species of bacteria has established itself, other bacteria introduced at a later date have more difficulty in colonizing. Contemporary studies have shown a distinct difference between the microflora of healthy, caries-free individuals compared to those with the caries disease.³²⁻³³ It is generally accepted that children with the highest number of MS in primary teeth will

experience a higher caries rate in their permanent teeth.³⁴ Although *S. mutans* has been implicated as the major etiologic agent of dental caries, new bacterial species are being identified. These bacteria profiles, many which have not yet been cultivated, change with the progression of the disease and differ between primary and secondary teeth.²⁷ A factor that has been recently explored related to dental caries is the pH of human plaque biofilm. Low pH of the oral cavity allows for an environment where cariogenic bacteria can flourish.

Stephan Curve

In the early 20th century, Dr. Robert Stephan, an officer in the US Public Health Service, suggested there was a continuous change in salivary pH following consumption of foods and beverages, especially with fermentable carbohydrates.³⁵⁻³⁶ The Stephan curve is a graphic representation to describe the rapid pH drop in plaque biofilm to a level that could cause demineralization of the dental enamel after consumption of sugar-containing foods and beverages. Stephan selected patients who were either caries-free or caries-inactive or who exhibited various degrees of caries activity.³⁶ Subjects were asked not to brush their teeth for three to four days prior to the measurement of the plaque biofilm pH on the labial surfaces of the anterior teeth. Prior to rinsing with 10mL of a 10% glucose solution for 10 seconds, pH readings were obtained. After rinsing with the glucose solution, pH readings were obtained at various time intervals until the pH returned to its original value. The initial drop in the pH was due to the speed at which the aciduric and acidogenic bacteria could metabolize sucrose. The low pH remained for some time, taking 30-60 minutes to return to its normal pH (in the region of 6.3-7.0). Differences were seen between the caries-free group and the caries-active group, with the later group having significantly lower plaque pH (Table I). This indicated the natural occurring saliva buffers were inadequate to neutralize the acids in the plaque biofilm and/or that acid was continually being produced in the plaque. This gradual return to a more neutral pH was the result of 1) acids diffusing out of the plaque and 2) bicarbonate ions in the saliva diffuse into the plaque biofilm and buffer the acids.

TABLE I – Stephan's pH Research

SUBJECTS CARIES STATUS	BASELINE SALIVA pH	BEFORE RINSE pH	AFTER RINSE pH
Caries Free	7.0	7.1	5.5
Caries Inactive	7.0	7.2	6.1
Slight Caries Active	6.7	6.8	4.9
Marked Caries Active	6.5	6.2	4.6
Extreme Caries Active	6.4	5.5	4.3

Adapted from Stephan (1944)

Demineralization

The saliva in contact with the teeth is supersaturated with calcium and phosphate, compared with the total levels of these minerals in enamel. The number of calcium and phosphate ions in plaque biofilm is greater than the number in the saliva.³⁷ As the pH drops from bacterial acid by-products, the level of supersaturation of the calcium and phosphate also drops and the risk of demineralization increases. While there is no exact pH at which demineralization begins, a general range of 5.5-5.0 is considered critical for tooth mineral to dissolve. As demineralization progresses, so does the carious lesion.

Salivary Glands and Saliva

The oral environment is controlled almost exclusively by the salivary glands. There are three primary glands that occur in pairs, located

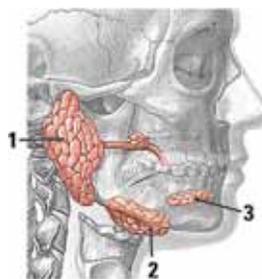


Figure 2: Salivary Glands
1. Parotid Gland
2. Submandibular gland
3. Sublingual gland

symmetrically on both sides of the head: Parotids, Submandibulars (sometimes referred to as Submaxillaries), and Sublinguals (Figure 2). The parotid glands are the largest of the glands and are located subcutaneously, below and in front of the ear. The saliva is carried into the oral cavity from the parotid via the Stensen's duct, opposite the maxillary second molar. Although the parotid glands are the largest, they only produce a quarter of the saliva volume.

The submandibular glands lie on the medial side (inside) of the mandible, in the submandibular fossa, below the mylohyoid ridge. Each submandibular gland has a duct that runs forward through the structures in the floor of the mouth and opens via the Wharton's ducts located at the lingual caruncles. The submandibular glands are the most active glands, contributing the most saliva volume. The sublingual glands are the smallest of the major glands and lie under the tongue in the floor of the mouth and contribute the least to the total saliva volume. Instead of having a single large duct, this gland has a row of 8-20 ducts called the ducts of Rivinus that lie beneath the mucosa membranes of the floor of the mouth. The largest of these ducts is called the sublingual duct of Bartholin and it joins the Wharton's duct to drain through the sublingual caruncle beneath the tongue.

Salivary secretions are classified as serous, mucous, or mixed. As the name implies, serous secretions contain more water than mucous. Each gland produces a different type of saliva (Table II). When salivary flow is unstimulated, such as in resting saliva (RS); the parotid, submandibular, sublingual and minor salivary glands contribute approximately 25%, 60%, 7%-8%, and 7%-8% respectively to the whole saliva volume.³⁸⁻³⁹ The flow rate of RS for all three glands is very low, approximately one-tenth of that during stimulated flow. The total amount of saliva secreted varies among individuals and environmental

Continued on Page 12

Gland Type	Saliva Type
Parotid	Serous
Submandibular	Mixed, more serous than mucous
Sublingual	Mixed, but mostly mucous
Most minor	Mucous

factors. Salivary flow is greater standing vs. sitting as well as during cool weather compared to hot weather. In addition, saliva is subject to a circadian rhythm, with the highest flow in mid-afternoon and the lowest

around 4:00 AM.⁴⁰ Approximately 0.5 – 1.7 liters of saliva is secreted into the oral cavity each day.⁴¹

Saliva

Saliva plays a critical role in the maintenance of optimal oral health and the creation of an appropriate ecologic balance. The function of saliva includes:⁴²

- Lubrication and protection of oral tissues
- Buffering action and clearance
- Maintenance of tooth integrity
- Antibacterial activity
- Taste and digestion

Except for during meal times and the occasional drink, saliva is the only fluid in the mouth. Consequently, the characteristics of the saliva have a direct impact on the oral environment. Cariogenic bacteria live in the mouth, and therefore saliva directly impacts their growth as well as their survival.

Saliva contains electrolytes such as sodium, potassium, calcium, magnesium, bicarbonate, phosphate, as well as, immunoglobulins, proteins, enzymes, mucins, urea, and ammonia.⁴³ These components help to modulate: 1) the bacteria attachment in oral plaque biofilm; 2) the pH and buffering capacity of saliva; 3) antibacterial properties and; 4) tooth surface remineralization and demineralization. These various components give saliva its overall quality and character. With the recent emphasis on the extended ecological plaque hypothesis, most noteworthy are saliva's pH and buffering capacity. The pH can be either acidic or basic, and the buffering capacity stabilizes the salivary pH. In other words, as buffering capacity increases, the pH of the mouth fluctuates less. When the pH of the mouth decreases (or becomes acidic), cariogenic bacteria are likely to thrive.

The salivary glands all work together to create stimulated saliva (SS) when food is being thought of or when eating occurs. Because SS is a "cocktail" of secretions from numerous cells and glands, it should be a mineral-rich and highly buffered solution that stabilizes the pH of the biofilm once all food has been swallowed. Through Stephan's research in the 1940s, it has been learned the action of SS "rinsing the plaque" begins the complex process of changing plaque pH to a more basic,

non-cariogenic value. During periods of rest, when no eating, drinking, or thinking about such things occurs, the salivary glands produce unstimulated or resting saliva (RS) with 65% of RS produced by the submandibular glands. Since the submandibular glands produce less buffer, the pH of RS is usually lower than the SS. If the RS pH is too low (below 6.6), healthy biofilm can transform into cariogenic biofilm.

Patient Management

Caries treatment is typically focused on the traditional approach of removal of the carious lesion and restoration or the more modern approach of elimination of bacteria via oral hygiene and antibacterials. With the role of pH in plaque biofilm mentioned previously, perhaps a more effective way to treat caries disease is to alter the oral environment in such a way as to make the plaque biofilm behave in a healthier manner. In order for this approach to be successful, an understanding of the characteristics of a patient's saliva is required. Tests are currently available for the dental professional to determine

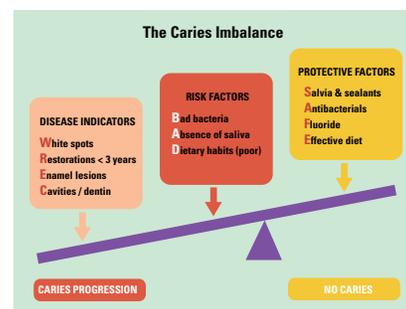


Figure 3: Caries Imbalance
Used with permission from the Journal of the California Dental Association (2007)

the viscosity, flow rate, resting pH, stimulated pH, and total buffering capacity of a patient's saliva. Armed with that data, a personalized treatment plan can be created and caries prevention becomes more successful. The focus is shifted from elimination of specific cariogenic microorganisms to the nurture/selection of a non-acidogenic biofilm through modification of pH of the environment.

Patient management should begin only after proper disease diagnosis, risk assessment, and prognosis has been determined. Once the decision is made that the patient is disease active and risk assessment is completed, an astute clinician can now evaluate the data and determine what pathological factors are out of balance and what protective interventions can improve the prognosis. This is done using the caries balance theory and caries risk assessment.⁴⁴⁻⁴⁷ The caries balance theory purports that dental caries disease depends upon the balance between demineralization and remineralization and is determined by the relative weight of the sums of pathological factors, protective factors and disease indicators (Figure 3).⁴⁷ Use of a risk assessment form can be supplemented with additional data collection based on how the patient presents. For example, a key part of the dental exam is analysis of the saliva and salivary glands. There are commercial tests available that can test pH, flow rate, and buffering capacity (Saliva Check Buffer, GC America). Once saliva problems are identified, the practitioner can recommend a course of preventive care.

Viscosity

Viscosity of the saliva relates to its thickness and is determined during the intra- and extra-oral examination. Here the clinician should assess the patency (unobstructed), consistency, and flow of the saliva. Saliva is 99% water and should look like water; not thick and stringy or frothy and bubbly. A quick and simple test to confirm function and duct patency is to “milk” one of the major glands. To milk the salivary duct, massage or squeeze the duct until saliva is expressed. At this time there is an opportunity to test the pH of the expressed saliva by using a simple piece of litmus paper. It is important to understand that just because saliva can be expressed from a gland and the mouth looks moist, does not rule out salivary gland hypofunction nor does it provide



Evert lower lip



Place filter or tissue paper



Beads of saliva will be seen through paper

any insight into the quality of the saliva. This must be measured quantitatively and qualitatively using saliva analysis.

The clinician can also evaluate the function of the minor salivary glands by everting the lower lip and drying it with a gauze pad. If the minor salivary glands of the lip express saliva in 60 seconds, the patient is considered to have adequate resting flow. Placing filter paper or tissue over the lip makes the beads of saliva easier to see. If the RS does not have the consistency of water (e.g. if it looks stringy or frothy) then it is possible the RS may be abnormal.

Another helpful test is to measure the pH of RS by placing a piece of litmus paper under the upper lip adjacent to teeth #8 and #9. This may take a few minutes to perform, as there is very little flow in this area. Do not be surprised by a low pH in this area since the minor salivary glands

express saliva at a lower pH than the major glands. A low resting pH (less than 6.6) indicates lack of proper salivary quality and demineralization of tooth structure could occur. Since most people do not stimulate their saliva in-between meals, the resting pH becomes a valuable tool in correcting the intra-oral chemistry of the individual patient.

Resting pH

If the patient has abnormal looking saliva then a pH test is recommended. Resting pH of saliva is determined by having the patient spit once into a container without chewing on anything and then placing a piece of litmus paper into this unstimulated saliva and assessing the pH value. In contrast to SS, there is a need for a practical test to



Dip litmus paper in and out of saliva quickly

quantitatively assess resting saliva (RS). This is where more subjective appearance of the resting state can give clinicians clues on the state of resting saliva.

Flow Rate

Salivary flow rate is determined by measuring the amount of stimulated saliva (SS) produced in a given period of time. Usually, a patient is provided a piece of unflavored wax which he or she chews for five minutes. All saliva produced during this time is collected and measured. Dividing the amount of saliva produced by the time provides the stimulated flow rate. A patient can subjectively look or feel “xerostomic,” but until the flow rate is quantitatively measured, a conclusion that the patient has salivary gland hypofunction cannot be made. For a salivary gland hypofunction diagnosis, one would have to have less than 0.7 ml/min of flow. Since a sample of SS has been collected, it is an ideal time to further test for saliva buffering capacity and use the saliva sample to culture MS and LB.

Buffering Capacity

The ability of the saliva to minimize an acid challenge is the buffering capacity. A high saliva buffer capacity may result in an elevated surface pH of the enamel crystal, resulting in favorable conditions for mineral uptake.⁴⁸ Once the SS is obtained, it can be used to test for buffering capacity by dropping samples on special litmus paper and comparing results to obtain a buffering capacity score.



Use pipette to collect saliva sample and place on buffer test strip squares

Once all the salivary data is obtained, an assessment of the quality and functionality of the saliva can be determined. This has direct implications on the caries risk assessment of the patient and allows the clinician to recommend products which will alter the chemistry of the mouth toward remineralization and health. For each pathological factor there should be a counter acting protective intervention (Table III). For example, low stimulated salivary flow can be improved by chewing xylitol gum or mints to stimulate saliva. In the case of abnormal pH (SS or RS) the patient should be taught to use products to neutralize acid such as chewing sugar-free gum, using baking soda products or more convenient commercially available products designed to buffer acid such as CariFree Boost (Oral BioTech, Albany, OR), or Salese/Dentiva (Nuvora Inc., Santa Clara, CA). Patients with low buffering capacity should use calcium phosphate products such as MI Paste (GC America, Inc., Alsip, IL) and ACP Relief (Discus Dental, Culver City, CA). Any patient diagnosed with salivary dysfunction should be treated with acid neutralization strategies to improve their restorative prognosis. Non-functional glands (e.g. secondary to radiation therapy) are the most difficult to address as there are no products made which can replace all the valuable functions of normal healthy saliva. The use of systemic

Continued on Page 14

sialogogues (i.e. pilocarpine or cevimiline) may improve salivary flow.

Educating patients to alter behavior that reduces saliva, such as ingestion of potent diuretics (e.g. caffeine and alcohol) is also suggested. Inhalation of cigarette and/or marijuana smoke dry the mouth even further and could reduce the oxygen tension allowing for more bacterial anaerobes, which may create more acid.⁴⁹⁻⁵⁰

Patients should have adequate water intake to avoid dehydration. Dietary education should be provided to every patient including a discussion on what foods are acidic as well as carioprotective. Hard cheese has been shown to coat teeth with a lipid layer, protecting surfaces from acid attack.⁵¹ Increasing arginine-rich proteins in the diet as been shown to rapidly increase plaque pH and should be recommended to patients at risk for dental caries.⁵²⁻⁵³ Ammonia production from arginine and urea metabolism has been identified as a mechanism by which oral bacteria are protected against acid killing, as well as maintain a relatively neutral environmental pH that may suppress the emergence of a cariogenic microflora.⁵⁴⁻⁵⁶ Arginine-rich proteins include

foods such as a variety of nuts (peanuts, almonds, walnuts, cashews, pistachios), seeds (sunflower, pumpkin, squash), coconut, kidney beans, soy beans, watermelon and tuna. BasicMints™ (OrtekTherapeutics, Inc.) contain arginine bicarbonate/calcium carbonate, and have been shown to raise pH levels and inhibit caries onset and progression.⁵³

Conclusion

Dental caries is an infectious and communicable disease. Multiple factors, such as the interaction of bacteria, diet, and host response, all influence dental caries initiation and progression. Saliva plays an important role in optimal oral health and new research suggests that salivary pH is even more critical to the development and progression of dental caries than once thought. Science suggests it is pH, rather than sugar, which is the selective factor for cariogenic plaque biofilms. Low salivary pH promotes the growth of aciduric bacteria which then allows the acidogenic bacteria to proliferate creating an inhospitable environment for the protective oral bacteria. This allows for a shift in the environmental balance to favor cariogenic bacteria, which further lowers the salivary pH and the cycle continues. Simple chemistry dictates at what pH enamel and cementum/dentin will demineralize. By controlling pH it is possible to alter the plaque biofilm, remineralize existing lesions, and perhaps prevent the disease altogether. The dental hygienist is well qualified to administer salivary tests which can determine the patient's caries infection risk, as well as the prevention and/or treatment measures to modify it.

Table III – Patient Management

Diagnosis	Diagnostic Criteria	Possible Treatments
Hyposalivation Xerostomia	Oral tissues appear dry Patient complains of “dry mouth” Known xerostomic medications taken Salivary flow rate <1ml/min Unresponsive minor labial glands	Increase hydration Saliva substitutes/stimulants Calcium phosphate therapy Saliva promoting lozenges Xylitol mints/gum Sialogogues MD consult to change xerostomic medications
Low Resting pH	Unstimulated pH < 6.8	Increase oral hygiene (power brush) Baking soda dentifrice Neutralizing gum or lozenges Baking soda rinses Xylitol mints/gum Cheese & nut snacks Increase Arginine intake (alter diet) Re-test at one month
Low Buffering Capacity	Buffering capacity score <10	Calcium phosphate therapy (at least t.i.d.) Increase Arginine intake (alter diet) Sialogogues
Poor Risk Assessment Score	CRA form indicates medium – high risk	Alter specific areas
Incipient lesions (ICDAS Criteria)	Rough white spots Radiographic enamel lesions	Tray delivered calcium phosphate therapy
Frank Cavitation (ICDAS Criteria)	DIAGNOdent readings >25 Radiographic D1 (1/3 into dentin) lesions	Strongly consider glass ionomer restoratives Culture one month post treatment
Orthodontic treatment in progress	Patient in brackets Patient has permanent retainer	Power brush Calcium phosphate therapy at bedtime

About the Authors

Michelle Hurlbutt, RDH, MS, is an Assistant Professor at Loma Linda University, Department of Dental Hygiene and Interim Director of the online BSDH Degree Completion Program. She teaches a variety of courses, including etiology and management of dental caries.

Dr. Brian Novy, DDS, is an Assistant Professor of restorative dentistry at Loma Linda University, where he teaches a variety of courses including caries management. He maintains a private practice in Valencia, California and acts as clinical director for three non-profit dental clinics in southern California. Dr. Novy is co-chair of the Western CAMBRA Coalition.

Dr. Douglas A. Young, DDS, MS, MBA, is an Associate Professor at University of the Pacific. Dr. Young speaks around the world on minimally invasive dentistry, lasers, and cariology. He has been published in several peer-reviewed dental journals and textbooks. Dr. Young is co-chair of the Western CAMBRA Coalition.

References

- Mouradian WE, Wehr E, Crall JJ. Disparities in children's oral health and access to dental care. *JAMA*. 2000; 284:2625-31.
- Dye, B.A., Tan, S., Smith, V., Lewis, B.G., Barker, L.K., Thornton-Evans, G., et al. Trends in oral health status: United States, 1988-1994 and 1999-2004. *Vit Health Stat*. 2007; 11(248):1-92.
- Berkowitz RJ. Acquisition and transmission of mutans streptococci. *J Calif Dent Assoc*. 2003; 31(2):135-38.
- Takahashi N, Nyvad B. Caries ecology revisited: microbial dynamics and the caries process. *Caries Res*. 2008; 42:409-18.
- Loesche WJ. Role of *Streptococcus mutans* in human dental decay. *Microbiol Rev*. 1986 Dec; 50(4):353-80.
- Van Nieuw Amerongen A, Bolscher JG, Veerman EC. Salivary proteins: protective and diagnostic value in cariology. *Caries Res*. 2004 May-Jun; 38(3):247-53.
- Anderson T. Dental treatment in medieval England. *Brit Dent J*. 2004; 197:419-24.
- Spielman AI. The birth of the most important 19th century dental text: Pierre Fauchard's *Le Chirurgien Dentiste*. *J Dent Res*. 2007; 86(10):922-26.
- Ismail AI, Hasson H, Sohn W. Dental caries in the second millennium. *J Dent Educ*. 2001 Oct; 65(10):953-9.
- Barrett W. The etiology of dental caries. *Am Dent Assoc Trans*. 1885; 24:91-114.
- Searle F. The septic theory of dental caries. *Am J Dent Sci*. 1884; 18:204-12.
- Miller W. Dental caries. *Am J Dent Sci*. 1883; 17:77-130.
- Orland FJ, Blayney JR, Harrison RW, Reyniers JA, Trexler PC, Wagner M, Gorodon HA, Luckey TD. Use of the germfree animal technic in the study of experimental dental caries. I. Basic observations on rats reared free of all microorganisms. *J Dent Res*. 1954 Apr; 33(2):147-74.
- Fredericks DN, Relman DA. Sequence-based identification of microbial pathogens: a reconsideration of Koch's Postulates. *Clin Microbiol Rev*. 1996; 9(10):18-33.
- Fitzgerald RJ, Keyes P H. Demonstration of the etiologic role of streptococci in experimental caries in the hamster. *JADA*. 1960; 61:9-19.
- Edwardsson S. Characteristics of caries-inducing human streptococci resembling *Streptococcus mutans*. *Arch Oral Biol*. 1968; 13:637-46.
- Loesche WJ, Rowan J, Straffon LH, Loos PJ. Association of *Streptococcus mutans* with human dental decay. *Infect Immun*. 1975 Jun; 11(6):1252-60.
- Alaluusua S, Kleemol-Kujala E, Nyström M, Evalahti M, Gronroos, L. Caries in the primary teeth and salivary *Streptococcus mutans* and lactobacillus levels as indicators of caries in permanent teeth. *Ped Dent*. 1987; 9:126-30.
- Loesche WJ. Chemotherapy of dental plaque infections. *Oral Sci Res*. 1976; 9:65-107.
- Loesche WJ. Proceedings of the first North American Congress on anaerobic bacteria and anaerobic infections. *Clin Infect Dis*. 1993; 16(S4): S203-10.
- Theilade E. The non-specific theory in microbial etiology of inflammatory periodontal diseases. *J Clin Periodontol*. 1986; 13:905-11.
- Coykendall AL. Classification and identification of the viridans streptococci. *Clin Microbiol Rev*. 1989; 2:315-28.
- Marsh PD. Are dental diseases examples of ecological catastrophes? *Microbiology*. 2003 Feb; 149(Pt 2):279-94.
- van Houte J. Bacterial specificity in the etiology of dental caries. *Int Dent J*. 1980; 30(4):305-26.
- Kingman A, Little W, Gomez I, Heifetz SB, Driscoll WS, Sheats R, Supan P. Salivary levels of *Streptococcus mutans* and lactobacilli and dental caries experiences in an US adolescent population. *Com Dent Oral Epidemiol*. 1988; 16:98-103.
- Corby PM, Lyons-Weiler J, Bretz WA, Hart TC, Aas JA, Boumenna T, Goss J, Corby AL, Junior HM, Weyant RJ, Paster BJ. Microbial risk indicators of early childhood caries. *J Clin Microbiol*. 2005; 43(11):5753-59.
- Aas JA, Griffen AL, Dardis SR, Lee AM, Olsen I, Dewhirst FE, Leys EJ, Paster BJ. Bacteria of dental caries in primary and permanent teeth in children and young adults. *J Clin Microbiol*. 2008; 46(4):1407-17.
- Marsh PD. Microbial ecology of dental plaque and its significance in health and disease. *Adv Dent Res*. 1994; 8:263-71.
- Bradshaw DJ, Marsh PD. Analysis of pH-driven disruption of oral microbial communities in vitro. *Caries Res*. 1998; 32(6):456-62.
- Alaluusua S. Transmission of mutans streptococci. *Proc Finn Dent Soc*. 1991; 87(4):443-47.
- Loesche WJ, Eklund S, Earnest R, Burt B. Longitudinal investigation of bacteriology of human fissure decay: epidemiological studies in molars shortly after eruption. *Infect Immun*. 1984; 46:765-72.
- Aas JA, Pastor BJ, Stokes LN, Olsen I, Dewhirst FE. Defining the normal bacterial flora of the oral cavity. *J Clin Microbiol*. 2005; 43:5721-32.
- Corby PM, Lyons-Weiler J, Bretz WC, Hart TC, Aas JA, Boumenna T, Goss J, Corby AL, Junior AH, Weyant RJ, Paster BJ. Microbial risk indicators in early childhood caries. *J Clin Microbiol*. 2005; 43:5753-59.
- Berkowitz RJ. Mutans streptococci: acquisition and transmission. *Pediatr Dent*. 2006; 28(2):106-109; discussion 192-98.
- Stephan RM. Intra-oral hydrogen ion concentrations associated with dental caries activity. *J Dent Res*. 1944; 23:257-66.
- Stephan RM. Changes in hydrogen ion concentration on tooth surfaces and in carious lesions. *J Am Dent Assoc*. 1940; 27:718-23.
- Lagerlöf F, Oliveby A. Caries protective factors in saliva. *Adv Dent Res*. 1994; 8:229-38.
- Dawes C. Salivary flow patterns and the health of hard and soft oral tissues. *JADA*. 2008; 139:185-245.
- Spielman AI. Interaction of saliva and taste. *J Dent Res*. 1990; 69(3):838-43.
- McKinney BE. Host defense mechanisms in the oral cavity. In Harris NO, Garcia-Gody F, Nathe CN, eds. *Primary Preventive Dentistry (7th ed.)*. Upper Saddle River, NJ: Pearson; 2009:115-19.
- Mese H, Matsuo R. Salivary secretion, taste and hyposalivation. *J Oral Rehabil*. 2007; 34(10):711-23.
- Mandel ID. The function of saliva. *J Dent Res*. 1987; 66:623-27.
- Humphrey SJ, Williamson RT. A review of saliva: Normal composition, flow, and function. *J Prosthet Dent*. 2001; 85(2):162-69.
- Featherstone JDB. The science and practice of caries prevention. *JADA*. 2000; 131(7):887-99.
- Featherstone JD. The caries balance: contributing factors and early detection. *J Cal Dent Assoc*. 2003 Feb; 31(2):129-33.
- Featherstone JD. The caries balance: the basis for caries management by risk assessment. *Oral Health Prev Dent*. 2004; 2 Suppl 1:259-64.
- Featherstone JDB, Domejean-Orliaguet S, Jensen L, Wolff M, Young DA. Caries risk assessment in practice for age 6 through adult. *J Cal Dent Assoc*. 2007 Oct; 35(10):703-13.
- Aiuchi H, Kitasako Y, Fukuda Y, Nakashima S, Burrow MF, Tagami J. Relationship between quantitative assessments of salivary buffering capacity and ion activity product for hydroxyapatite in relation to cariogenic potential. *Aus Dent J*. 2008; 53(2):167-71.
- Cho CM, Hirsch R, Johnstone S. General and oral health implications of cannabis use. *Aus Dent J*. 2005; 50(2):70-4.
- Avsar A, Darka O, Topalolu B, Bek Y. Association of passive smoking with caries and related salivary biomarkers in young children. *Arch Oral Biol*. 2008 Oct; 53(10):969-74.
- Gedalia I, Ben-Mosheh S, Biton J, Kogan D. Dental caries protection with hard cheese consumption. *Am J Dent*. 1994; 7:331-332.
- Bowen WH. Food components and caries. *Adv Dent Res*. 1994 Jul; 8(2):215-20.
- Acevedo AM, Montero M, Rojas-Sanchez F, Machado C, Rivera LE, Wolff M, Kleinberg I. Clinical evaluation of the ability of CaviStat in a mint confection to inhibit the development of dental caries in children. *J Clin Dent*. 2008; 19(1):1-8.
- Burne RA, Marquis RE. Alkali production by oral bacteria and protection against dental caries. *FEMS Microbiol Lett*. 2000; 193:1-6.
- Casiano-Colon A, Marquis RE. Role of the arginine deiminase system in protecting oral bacteria and an enzymatic basis for acid tolerance. *Appl Environ Microbiol*. 1988; 54:1318-1324.
- Marquis RE, Bender GR, Murray DR, Wong A. Arginine deiminase system and bacterial adaptation to acid environments. *Appl Environ Microbiol*. 1987; 53: 198-200.

Home Study Correspondence Course

“Dental Caries: A pH-mediated disease”

2 CE Units – Member \$25, Potential member \$35

Circle the correct answer for questions 1-10

1. Dental caries in on the rise in primary dentition, among children aged 2-5 years.
 - a. True
 - b. False
2. The first researcher to publish a theory about the involvement of microorganisms in the dental caries process was:
 - a. Pierre Fauchard
 - b. Paul Keyes
 - c. Robert Koch
 - d. Willoughby Miller
3. Which plaque hypothesis purports that dental caries is caused by only a few species of bacteria?
 - a. Non-specific plaque hypothesis
 - b. Specific plaque hypothesis
 - c. Ecological plaque hypothesis
 - d. Extended ecological plaque hypothesis
4. The Ecological Plaque Hypothesis suggests that:
 - a. it is the pH, not the sugar, that causes the pathologic shifts of plaque biofilms.
 - b. the caries disease is an outcome of the overall activity of the total plaque microflora and not a specific organism.
 - c. the primary colonizers, the first bacteria to adhere to the teeth, determine the eventual pathogenicity of the plaque biofilm.
 - d. the presence of pathogenic species, such as *Mutans Streptococci* and *Lactobacilli* (MS and LB) within the plaque biofilm causes dental caries.
5. The Stephan’s curve describes the change in dental plaque pH in response to:
 - a. the type of bacteria present in the plaque biofilm.
 - b. the number of bacteria present in the plaque biofilm.
 - c. supersaturation of the calcium or phosphate ions.
 - d. consumption of a beverage or food.
6. Humans are not born with cariogenic bacteria. Transmission of cariogenic bacteria occurs during the birth process.
 - a. Both statements are true.
 - b. Both statements are false.
 - c. The first statement is true, the second statement is false.
 - d. The first statement is false, the second statement is true.
7. This gland produces the most watery type of saliva.
 - a. Parotid gland
 - b. Stenson’s gland
 - c. Sublingual gland
 - d. Submandibular gland
8. Resting saliva is:
 - a. a sterile salivary secretion.
 - b. produced after mastication.
 - c. produced primarily from the parotid gland.
 - d. unstimulated saliva.
9. Salivary pH can contribute to all of the following EXCEPT:
 - a. if the plaque biofilm will be aciduric.
 - b. if the plaque biofilm will be acidogenic.
 - c. if the mineral will demineralize.
 - d. the rate of stimulated salivary flow.
10. Why does raising the salivary pH favor less dental caries?
 - a. Raising the pH will create an environment with more calcium and phosphate ions.
 - b. Raising the pH will not allow the acid loving bacteria to exist.
 - c. Raising the pH will increase saliva production.
 - d. Raising the pH will ensure remineralization of the tooth surface.

The following information is needed to process your CE certificate. Please allow 4 - 6 weeks to receive your certificate. Please print clearly:

ADHA Membership ID#: _____ Expiration: _____

Name: _____ License #: _____

Mailing Address: _____

Phone: _____ Fax: _____ Email: _____

Signature: _____

**Please mail photocopy of completed Post-test and completed information with your check payable to CDHA:
130 N. Brand Blvd, Suite 301, Glendale, CA 91203**