Caries Process and Prevention Strategies: The Agent

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Video Transcript

Hello, and welcome to dentalcare.com's Cariology course. It focuses, this time, on the agent. This is part two of a 10 part series entitled Carrie's Process and Prevention Strategies. This course describes the etiology and pathways of progression of dental caries including an in depth review of the role of dental plaque and oral bacteria.

Commonly termed tooth decay, caries is a multi-factorial disease that can be difficult to manage. The processes associated with caries result in the localized destruction of tooth tissues, occurring over time, when certain oral bacteria ferment dietary carbohydrates, generating acid that cause damage to the teeth. The primary oral bacteria responsible for caries is streptococcus mutans.

Dental caries is the most prevalent disease in man, affecting most of the dentate population at some time in the lives. In the U.S., caries is the most common chronic disease of childhood. Forty-two percent of children, ages two to 11, have had caries in their primary teeth. Twenty-three percent of children in this age group have had untreated caries. Among dentate adults, ages 20 to 64, 91 percent have or have had caries in their permanent teeth.

Many oral bacteria live in aggregate in dental plaque, forms on the outer surface of the teeth. In a healthy mouth, the plaque bacteria are harmless. When the environment becomes acidic however, the population changes to bacteria that thrive in acidity in our link to caries. The caries process, as well as multiple factors that influence caries development in plaque is a microbial bio filled ecosystem are discussed.

First, we'd like to go over a couple of clinical significant snap shots. These are questions that have come up with regard to the agent itself. First, simply put, what causes dental decay? How can I explain it to my patients? Well, dental decay is caused when bacteria that accumulate on the surfaces of the teeth feed on sugars in the diet and convert the sugars into acids and then dissolve the hard tooth material. This results in the loss of minerals. Which then, in turn, results in cavities.

Nearly every mouth contains the bacteria that cause decay. The mouth can withstand several attacks each day from the bacteria that turn sugar into acids. During times between means, with sugar, it's possible for the tooth to repair itself, replacing the minerals that have been dissolved by the acids.

Fluoride in toothpaste, helps make teeth more resistance to the acid attack. It's important to clean teeth well to remove as much bacteria as possible. But those at high risk of developing caries, the finish meals with items that are rich in calcium such as, yogurt milk or cheese. Once sufficient mineral has been lost, the tooth forms a cavity that only be repaired by the dentist by placing the filling. The key process starts with the appearance of a white spot on the surface of the teeth.

Another question, of all the factors listed, what are the most important to control in order to present the onset of dental caries? The

frequency of intake of sugar should be reduced as much as possible. And ideally, limited to meal time so that acids in dental plaque are only produced three to four times a day. And, and that there is plenty of time between meals for saliva to act by replacing any minerals that have been resolved by the acid production during the meal times.

The presence of fluoride makes enamel more resistance to acid dissolution and encourages the process of re-mineralization. The removal of plaque biofilm is important too. Although, it's impossible to remove all the decay causing bacteria from the mouth. Therefore, the next most important action, after reducing the frequency or sugars in the diet, is to brush at least twice a day with a fluoride toothpaste. It strengthens the enamel against acid attack, encourages mineralization and removes the plaque biofilm.

Upon successful completion of this course, it is expected that you, the dental professional, will be able to first, define dental caries, discuss the overall history of caries, identify the combination of factors associated with caries and how some factors influence that process. To be able to define dental plaque as a microbial biofilm and describe how it develops and matures. To fully understand the microbial diversity of plaque and recognize it as an ecosystem, to be able to discuss the ecological plaque hypothesis. To be able to name the bacteria most commonly associated with caries and to discuss how the activity and the oral environment is a major determinate of plaque ecology. Finally, how bacteria convert dietary carbohydrates into acids.

Dental caries is a biofilm disease that results in the targeted destruction of tooth tissue by bacterial acids such as lactate acid. Lactic acids is produced in the mouth by oral bacteria primarily streptococcus mutans. Also commonly known as S mutans. S mutans ingest and ferment or breakdown the dietary carbohydrates, also known as sugars.

If the P.H. in the localized tooth environment becomes too acidic, defined as below P.H. 5.5, dissolution of the enamel or demineralization begins to occur. In its early stages, lost mineral could be replaced by way of the remineralization process. If not reversed in its early stages, demineralization can continue to eventual capitation.

Although dental caries is as an infectious disease, one does not catch dental caries. Bacteria that causes caries, when they thrive under specific conditions, populate the oral cavity of all humans. First, entering the body when a baby passes through the birth canal. Caries is better referenced as a shift in oral microflora to caries causing bacterial types in response to an acidic P. H. caused by the metabolism of sugars.

Our knowledge of caries has evolved over many, many centuries. Around 2500 B.C., the ancient Chinese believed cavities were caused by tooth worms, as depicted here. By 350 B.C. Aristotle and others acknowledged that certain foods, such as sweets and figs, were likely the cause of decay. In 1819, Levi Parmly hinted that the real cause of decay was bacteria growing on food particles lodged around and between the teeth. In 1881, Myles and Underwood stated that acid and germs were necessary for cake decay to occur. In 1889, W. D. Miller formulated the concept of caries is a local phenomenon related to carbohydrate retention and acidic bacteria.

During the early to mid 1900s, research uncovered important new findings. In 1938, H. Trendley Dean linked fluoride to caries reduction and later studies high sugar consumptions were linked to caries. But only when oral bacteria was present. Findings from the first half of the 1900s led to a greater understanding of fluoride's effect on enamel development and advances in the delivery of beneficial amounts of fluoride to reduce caries.

In 1955, the Proctor and Gamble company introduced Crest, the first fluoride toothpaste clinically proven to be an effective in preventing cavities. Held as a major breakthrough in dentistry, it received an endorsement from the American Dental Association as an effective decay preventive dentifrice. It can be a significant value.

The American Dental Association has long emphasized the benefit of fluoride. In its statement commemorating the 60th anniversary of community water fluoridation, the ADA noted, “Studies conducted through the past 60 years, have consistently indicated that fluoridation of community water supplies is safe and effective in preventing dental decay in both children and adults. It is the most efficient way to prevent one of the most common childhood diseases, tooth decay. Five times as common as asthma and seven times as common as hay fever in five to 17 year olds.”

Subsurface mineral loss and progression to dental caries is defined as a multi-factorial disease. Characterized as the interaction of four key factors: the host, the substrate or food and drink, oral bacteria and the amount of time the bacteria lasts. It’s remaining contact with the hydroxyapatite mineral fatigue. We will discuss each of these in detail and how they interact to cause subsurface mineral leading to, uh, caries lesion progression.

First, let’s discuss the host. The host includes two tooth morphology, saliva production and the immune response. Oral bacteria includes the proliferation of acidogenic or acid producing bacteria. The substrate refers to the intake of formidable carbohydrates or sugars. And finally, the time of acid interaction... This is described in detail by use of the Stephan Curve and description of the de-mineralization of enamel.

As noted, caries is the result of four key factors and multiple sub-factors. The combination of factors is the primary reason that caries can be such a difficult problem to control. Understanding the impact of the various factors, will help support intervention and prevention strategies... sub-factors that influence caries development are, for example, age such as children’s deciduous teeth are more susceptible to demineralization of the enamel than adults.

If fluoride has been used on a regular basis, tooth morphology, which various within the mouth and from person to person. Root surface exposure due to gum recession, and saliva flow rate and buffer capacity. Caries risk is also higher if the diet is low in nutrients, such as magnesium and vitamin D that are necessary for healthy teeth development as well as when teeth have numerous and exaggerated grooves and pit and fissure-aries.

Probably the most important of the sub-factors associated with the host is saliva. Physically, saliva helps reduce the potential for caries. Specifically, when the salivary flow rate is high or around two mills per minute. The saliva can help to clear oral debris and bacterialasses. Chemically, saliva is buffer capacity and the ability to neutralize acids helps limit demineralization damage. These are prone to less caries or less prone to caries activity in situations where tooth enamel has been strengthened by fluoride or when the diet includes sufficient quantities of tooth strengthening nutrients, such as calcium and phosphate.

Sub-factors that influence caries development are oral clearance, if food is retained or not in the mouth after eating, oral hygiene, if after eating, food is actively removed with a sharp instrument, such as a tooth pick, eating frequency, food detergency or if consumed food can clean the teeth, consumption of carbohydrates and if the cariogenicity of consumed carbohydrates. For example, sucrose is more cariogenic then group glucose or fructose.

When food is retained in the mouth and not actively removed after eating, is consumed more frequently and or more sugars, sucrose containing foods and sticky foods like toffee, marshmallows, raisins and the like, are consumed there is a higher risk of caries. Where remaining food particles are actively removed after eating, food is consumed less frequently, fewer sugars, sucrose containing foods and sticky foods are eaten and then completely swallowed and or more tooth cleaning foods like apples are eaten, the likely hood of caries is lower.

The development of caries depends on the microbial load, which means how much bacteria is present. Plaque composition,

since some types of plaque microbs are more cariogenic than others. Plaque [inaudible 00:12:39], acidjeda-denicity or how much acid can be produced by the plaque that is present. Plaque aciduricity or how well plaque can survive in acidic conditions. Oral hygiene, as this determines how often the microbial load is reduced by brushing or prophylaxis and if fluoride is present in the plaque.

Likelihood of caries is higher when the microbial load is high, which is indicated by excessive plaque. When more caries linked bacteria are present, when plaque produces more acid in the presence of excess sugars in the diet. When more plaque can survive in acidic conditions and or when plaque is not regularly moved by brushing, the odds of caries will develop are lower when the microbial load is low as indicated by little plaque, plaque [inaudible 00:13:29] cariogenic bacteria or that can withstand highly acidic conditions. Plaque acid production is low and or plaque is regularly removed by brushing or flossing.

There are two concepts that are important to understand with regard to oral bacteria. These are sugar metabolism and acid production. Dietary sugars, starches and formidable carbohydrates, usually [inaudible 00:13:53], usually collectively referred to as sugars, are present in the diet and in direct contact with plaque during eating and for some time afterwards.

The breakdown of sugars is an important step that influences the plaque environment. Enzymes and bacteria and saliva breakdown sugars polysaccharides and disaccharides to monosaccharides. S mutans use the enzyme glucan-glucansucrase, also known as glucasyltransferase to convert sucrose into a sticky, extra cellular, dextran polysaccharide that allows them to cohere forming plaque.

There are five main mechanisms by which oral streptococci hydrolyze or breakdown sucrose. First, extra cellular invertase clears the energy rich alpha one to two glycosidic bond between glucose and fructose glyotease. Second, the bacteria cell transports sucrose across the cell membrane and clears the glycosidic bond using tricellular invertase. Third, extra cellular glucosyltransferase py-polymerize the glucose molecule while liberating fructose so it can enter the bacterial cell.

Fourth, extra cellular fructosyltransferase is polymerize the fructose. While the glucose molecule is liberated and freed to enter the cell. And fifth, solidary-alma-laze clears the polysaccharides. And now with regard to acid production, oral bacteria convert glucose, fructose and sucrose into energy, an acid [inaudible 00:15:33] process called glycolysis. Which is the main energy generating pathway in all bacteria, including S mutans. The bacteria most closely associated with caries along with lactobacilli.

The monosaccharides, glucose, glactose and fructose give andoglycolisis pathways at points shown in the diagram. Additional intermediate steps occur in the production of energy. S mutans is capable of metabolizing pyruvate or pyruvic acid further to generate yet more energy and more acid by products. In the presence of excess sugars, they favor what is called the lactate dehydrogenous pathway to produce lactic acid.

While between meals, they utilized sir energy reserves and produce formic, antacedic acid instead. These are not associated with caries... While the shift in microfluoric can assure... can occur over a fairly short period, a significant amount of time is needed for demineralization to lead to the development of white spot and or caries lesions. The critical P.H. for demineralization is approximately 5.5. With the extent of time the environment remains in the de-mineralization phase determining how much damage occurs.

Acid production does not instantly trigger tooth decay. In an early stages, remineralization can restore enamel, keeping the effects of dental caries at bay. The Stephan Curve describes the phenomena of P.H. change of dental plaques over tine when challenged with cariogenic foods. In other words, carbohydrates. Dental plaque P.H. is consistent under what is called resting conditions. When no food or drink is present and is generally around P.H.s six to seven.

Upon exposure to formidable carbohydrates, P.H. decreases due to acid production by the bacteria reaching a minimum of about four and a half to five in approximately five to 20 minutes. The rate at which this occurs is dependent on the metabolic rate factors we discussed on the last slide. This is followed by a gradual recovery over 30 to 60 minutes to a resting P.H. influenced by the buffering capacity of the unstimulated saliva and the rate of stimulated saliva.

Demineralization or mineral loss takes place when the P.H. is below what is considered the critical P.H., which is the highest P.H. at which there is no la-loss of mineral of P.H. of around 5.5. The prevention of subsurface mineral loss in caries progression, hinges on the ability of at home oral care intervention to shift, to curve up, minimizing the time and exposure of enamel to dietary acids.

In summary, dental caries is a multifactorial disease. Bacterial fermentation of consumed acid, uh, of consumed sugars produces acid. The acid de-mineralize the tooth enamel. Over time, the dissolution of tooth structure leads to the development of carious lesions. And, the combination of factors and sub-factors makes dental caries very difficult to prevent.

Now, let's discuss dental plaque. There are four key aspects of plaque that are important to understand. First, is the biofilm. Then microbiology. Third is stages of development and four will be collagen health and disease. But first, the biofilm. Bacteria collect on the teeth and along the edges of the gums in a green colored mass called plaque. Importantly, the ba-, the bacterial deposits that form plaque on teeth, are considerably different from those on soft tissues.

Teeth have a non-shedding surface and this allows time for the development of a bacterial structure to form. The structure serves as a biofilm. Which is an aggregate of microorganisms in which cells adhere to each other and to an aqueous surface. Ninety percent of plaque volume is extra cellular polysaccharides, which is change of sugar units produced by the bacteria. The polysaccharides hold the bio film together and trigger changes that make it difficult to remove. When the cell becomes a component of a bio film, [inaudible 00:20:01] of experiences is a shift in gene expression. This makes it up to a thousand times more resistant to antibodies, antibiotics and them, and antimicrobials then its planktonic or [inaudible 00:20:15] counterparts.

Microbiology. All oral bacteria produced acidic byproducts when they metabolize sugars, which cause a drop in plaque P.H. Although there are many plaque bacteria, two specific types that are most associated with caries, streptococci, most notably S mutans and lactobacilli. Streptococci or Grand Positive Cocci, then form chains and make up 30 to 40 percent of the plaque.

With very efficient sugar transport and storage systems, they produce large amounts of lactic acid. When excess sugars are available, or formic anticidic acids, which are not associated with caries, when using their energy reserves. S mutans is a strain most strongly implicated in massive production in caries. It is an acidogenic or acid producing bacteria. It adheres to the tooth bio film by converting sucrose into an extremely adhesives substance, dextran polysaccharide or the enzyme dextran sucrase.

Interestingly, S mutans may not be present for caries to occur. Individuals without this strain can still get caries since they may have other oral bacteria that create acidimineralizing conditions. Lactobacilli or grandpa's [inaudible 00:21:40] rods make up only about one percent of plaque and are also aciduric, which means they can endure acidic environments.

While not likely to contribute to caries, they're frequently isolated from caries lesions due to their ability to thrive at this low P.H. So, while they are not associated with caries because they seem to be present when caries is occurring, it is not clear how much they are actively responsible for the caries themselves.

Stages of development. There are six stages of plaque bio film development. First is the formation of an acellular layer called the acquired pellicle. This layer of salivary

glycoproteins, phosphoprotein and lipids are fats. But no bacteria forms almost immediately on naked enamel surfaces.

Stage two is the initial attachment. Free floating early colonizers of the teeth, such as streptococcus sanguinous, also called S sangous, which are normal inhabitants of the mouth, form an initial attachment to the pellicle by weaken and reversely van der Waals forces. But these bacteria are not removed. They eventually anchor themselves with adhesive structures, such as pelli.

Stage three is referred to as irreversible attachment. Organisms that were unable to attach the pellicle begins to adhere to the first layer of colonizers, with irreversible attachment be a specific adhesive receptor interactions. The bacteria replicate and form micro colonies embedded in an extra cellular matrix.

Step four is called Early Maturation or Maturation I. Following from the initial steps, early colonizers become established leading to increased dental plaque complexity. Allogenic factors such as oxygen consumption within plaque create anerobic zones. Food chains become established and an increased range of receptor site become available for bacterial attachments. Cell division and recruitment of new bacteria also allows the bacteria population to increase.

Stage five is also called Late Maturation or Maturation II. In this stage, microbial diversity continues to increase while the rates of cell division decrease. The heterogeneous nature of plaque becomes apparent as a mosaic of microenvironments develop, particularly areas of different P.H., oxygen concentrations and secondary metabolite accumulations around, within the micro colonies. The plaque microbial ecology reaches a pseudo-steady-state climax community where there is constant turnover cells. But the overall composition remains roughly the same. At this point, a thick three dimensional layer of dental plaque is formed.

Finally, stage six or the Dispersion Stage. Enzymes that degrade the biofilm, such as dispersion B allow some bacteria to detach themselves from the biofilm, sometimes in response to deleterious environmental conditions in order to spread and colonize new surfaces in the oral cavity.

Next we will talk about ecology and health and disease. Mature dental plaque is composed of a highly complex microbial community, with a population varying from person to person and between different sites in the mouth. Classical microbiological techniques have estimated that plaque contains an estimated 800 distinct oral species, with a healthy individual possessing 50 to 100 different species at any one time.

Pyrosent... Pyrosequencing, a powerful molecular technique which analyzes ribosomal RNA, estimation of at least 19,000 phylotypes, assuming a six percent difference in RNA sequence, constitutes a new species. These populations of bacteria form their own microbial ecosystem and plaque. Just like any other ecosystem, the plaque microbial ecosystem, can both influence its environment and be influenced by the oral environment.

Production of acids by sugar fermentation lowers the plaque P.H. causing a shift in plaque ecology. Which acid sensitive bacteria, such as S sanguinous are less able to survive. While aciduric bacteria, strep mutants and lactobacilli, will thrive. The end result is disruption in the natural balance between dental plaque and the tooth surface. More acid production and more de-mineralization.

On the other hand, were P.H. remains neutral, acid sensitive bacteria, like the S sanguinous, can survive, keeping acid production low and increasing the possibility of remineralization. The concept of the oral environment being able to cause a shift in dental plaque ecology that can either lead to good health or disease, such as caries and gingivitis, is referred to as the ecological plaque hypothesis.

What drives the shift in plaque ecology is not simply the present of sugars. But rather the acid formed by their fermentation that can cause P.H. to drop from a neutral of seven to a P.H. of lower than 5.5. At a P.H. of 5.5, the plaque community remains stable. But as the

P.H. drops toward 4.5, S mutans and lactobacilli increase. The drop in the P.H. to below 4.5 is considered an environmental catastrophe for plaque microflora, like S sanguinous that normally inhabit a healthy mouth. At this P.H., acid sensitive speties can be inhibited or killed while the acid tolerant species will proliferate.

Conclusions. Dental caries is a multifactorial infectious disease affecting a significant percentage of the population. It's more accurate to consider caries is caused, not by an infectious agent, that by a shift in the oral microflora to caries causing types, in response to acidity, resulting from the [inaudible 00:27:57] metabolism of sugars. The development of caries is dependent on the interaction of four primary factors. These are our hosts or tooth surface, a sub-straight food, the presence of oral bacteria and time. Caries will not develop if any of these four primary factors are not present.

Understanding the etiology and pathways of progression of dental caries will enable the dental professional to strive toward early intervention and hopefully prevention... Let's conclude this section by discussing how this information can help you and your practice. First, fully understanding information regarding the agent and the appropriate prevention strategies involved in the caries process will help you identify evidence based and scientifically supported interventions to reduce sub-surface mineral loss and make decisions regarding your patient's at home care and reduction of caries risk.

Second, information about the agent and appropriate prevention strategies, when communicated at the level of the patient, can be a powerful tool in driving compliance and overall adherence to your at home oral care recommendations. Describing how caries develop and making the connection to your specific recommendations instills a strong sense of trust and confidence in patients and can be far more powerful then simply instructing patients to brush more often. Thanks very much.