Caries Process and Prevention Strategies: The Environment

The following is a transcription that has been taken verbatim from the presenter's audio. No Edits have been made.

Video Transcript

Hello and welcome to dentalcare.com's cariology course. Today's section is called The Environment. This is part four of a 10 part series entitled Caries Process and Prevention Strategies. It has been established that the oral environment is one of the primary factors in the caries process. Only when acidity increases in the oral environment does demineralization of enamel and, subsequently, caries occur. In this section the role of fermentable carbohydrates is discussed, paying particular attention into how caries can be influenced by the cariogenic potential of ingested sugars and starches, the physical traits of ingested carbohydrates, such as their adhesiveness, and the frequency of intake and exposure to sugars. The Stephan curve, which illustrates the dental pH changes over time in response to a carbohydrate challenge, is also introduced with a discussion of how factors such as the type of carbohydrate, the buffering capacity of saliva, and the type and quantity of bacteria present in plague effect the dental pH responses.

First, we wanted to go over a couple of clinical significant snapshots, questions that you may come across in your practice. The first, which environmental factors can easily be modified to aid in the prevention of dental caries? Well, if a patient or other family member have signs of caries it's critically important to look for environmental factors that may be contributing to increased risk of developing the disease. While modifying environmental factors is always a challenge, success is more likely if the family's unit environment is investigated as well as that of the specific at risk individual family member. Changing the environmental factors of only one family member is unlikely to succeed.

Although some factors are easier to modify than others there are some things that can be done without too much effort that may have a significant impact toward the prevention of caries. These include A) alteration in the consumption of sugars as fermentable carbohydrates to minimize the potential for demineralization. B) controlling the dental plague biofilm to reduce the quantity of cariogenic bacteria present. Although there are a few studies showing direct correlations between oral hygiene in the prevention of dental caries, likely due to the fact that it is impossible to remove all of the acidogenic or cariogenic bacteria present in the mouth. It makes sense to try to minimize the overall challenge as much as possible. And C) the encouragement of remineralization processes to maximize the potential for mineral repair.

Another question that may come up is, how can I work with my patient in modifying these environmental factors to reduce the risk of developing dental caries? After making an overall assessment of the caries status of each patient, positive environmental changes could be suggested that will help reduce the risk of caries by minimizing negative factors and encouraging positive ones. For example A) ingestion of fermentable carbohydrates, especially the mono- and disaccharides of glucose and sucrose, is the most important causative factor to change. Eating and drinking habits should be investigated, ideally through a three or four day diet diary that lists all eating and drinking occasions to access the amount and frequency of exposure. Another, sugars as part of a meal should be reduced whenever possible and increased consumption of fruits and vegetables can be suggested as alternatives to sugar containing foods. The use of sugar substitute should be encouraged. Between meal episodes should be minimized. And if obesity is present in the family, referral to a dietician or nutritionist may be very beneficial for all.

B) Control the dental plaque biofilm to regularly remove as many acidogenic, cariogenic bacteria from the mouth as possible. Encourage frequent and thorough oral hygiene practices, including twice daily brushing with fluoride containing toothpaste, regular flossing, and the use of fluoride containing or antibacterial mouth rinses wherever warranted.

And C) modify the oral environment to enhance the potential for remineralization, encourage the use of sugar free chewing gums to stimulate the production of saliva. Saliva is the key remineralizing agent. It's super saturated with respect to calcium and phosphate and it has very good buffering capacity. Increasing the flow of saliva helps to decrease clearance time, reduce the length of exposure to cariogenic substances in the oral cavity.

Upon completion of this course the dental professional should be able to identify the role of the environment in dental caries ideology, to understand and discuss the Stephan curve, to be able to explain the impact of various diets on the incidence of caries, to be able to describe the concept of frequency versus amount of cariogenic carbohydrates, to be familiar with the complex chemical structure of sugars, and to be able to relate the cause and effect of diet and dental caries to the patients.

In 1947 a series of human experiments were begun on patients in Vipeholm Mental Hospital in Sweden. Unknown to the Swedish government but sanctioned by the dental community and the confectionary industry, a group of mental patients were fed copious amounts of sweet foods, like chocolates and caramels, in a full scale experiment designed to bring about tooth decay. The experiments provided extensive knowledge about dental health and resulted in the breakthrough findings that the intake to... of sugar was linked to dental caries, that certain physical qualities of sugars, such as their stickiness, influenced caries risk, and that the frequency with which sugary foods are consumed also affects caries development.

While scientifically speaking the experiment was a success with more having been learned about dental health and caries than from any previous study, the study would never have taken place today. It violates the principles of medical ethics. Many subjects ended up with their teeth completely ruined to provide fodder for subsequent studies that continued to increase dental knowledge and to provide much of the information that follows about the oral environment factors that play a role in the dental caries process.

The presence of fermentable carbohydrates changes the oral environment. Three main factors play a role in the dental caries process. The cariogenic potential of fermentable carbohydrates, the physical traits of fermentable carbohydrates, and the frequency of intake of and exposure to these fermentable carbohydrates. Simple sugars like sucrose, fructose, lactose, galactose, and glucose foster colonization and growth of bacteria linked to caries, particularly streptococcus mutans. Studies indicate that subjects placed on high sucrose diets exhibit increased s. mutans count and the incidence of early carious lesions. Although starch is recognized to have a lower carcinogenicity than sucrose, frequent consumption of starch has been shown to produce a large number of carious lesions. This is because starch can be broken down into maltose by the enzymes in saliva called amylase. Maltose can then be further metabolized to acids which in turn leads to demineralizations.

Conversely, dietary restriction of fermentable carbohydrates and cooked starches reduce the level of cariogenic organisms in humans. A classic 15 year intervention study, the Hopewood House Study, conducted in Australia evaluated the clinical effects of a sucrose restricted diet among 81 children aged four to nine years. At the start of the study 78% of the children were caries-free and 53% continued to be caries-free at age 13. This was significantly higher than the proportion of caries-free 13 year olds within the general residential population, which is only the 0.4%. When the children from Hopewood House were relocated as they became older, they no longer adhered to their strict diet. The result was a steep increase in caries increment similar to that found in other children, indicating that teeth do not acquire any permanent resistance to dental caries.

Physical traits include the adhesiveness, or stickiness, and clearance time of dietary carbohydrates, as well as the frequency of exposure to them. In general those dietary carbohydrates that are sticky confer the highest potential for caries. This was first demonstrated in the Vipeholm study, which had study subjects consume sucrose in toffee, chocolate, caramel, bread, or in liquid form. It was shown that the intake of sticky foods, like toffees and caramels, produced higher caries rates among monitor subjects than those who consumed rapidly swallowed sugars. The increase in caries activity disappeared when sugar rich foods where reduced or removed from the diet. However, the cariogenicity of liquid sugars should not be discounted given the high incidence of caries associated with soft drink consumption and with the occurrence of early childhood or baby bottle caries.

It's also important to consider the clearance rate of dietary carbohydrates in the caries process. Different foods are cleared from the oral cavity at different rates. For example, sticky retentive foods, such as toffees, or foods that can compact in the pit and fissures of the teeth, such as biscuits and cakes, have increased clearance times. In general, refined carbohydrates that are retained for long periods tend to be the most cariogenic. Also, bacterial acid production can persist after the carbohydrate has cleared from the oral cavity.

The link between the frequency of carbohydrate an... intake and caries incidence was also

investigated in the Vipeholm study. When study subjects ingested 300 grams of sugar with meals a significantly lower caries rate was observed, 0.43 new carious lesions per year, compared to subjects ingesting the same amount of sugar as snacks between meals, where they saw 4.02 new carious lesions on an annual basis. Increased snacking increases the risk of caries because increasing the frequency of sugar intake extends the duration of acid production and exposure thereby tipping the scale toward the development of caries. This can be demonstrated simply by measuring plague pH which would be the immediate environment of the tooth throughout the day. In the example below it can be clearly seen that in graph A, the one on the top, uh, increasing the frequency of eating and drinking increases the episodes when the pH of plague falls below 5.5, the critical pH. In B, the one on the bottom, restricting between meal snack and drinking non sugary drinks reduces the time that the plaque falls below pH 5.5.

An interesting observation is that it can be less beneficial to eat one sweet than it is to eat five sweets in immediate succession. With five in succession, the levels of sucrose may be toxic to bacteria and there may be a greater salivary stimulatory effect. Furthermore, if five sweets are spread out throughout the day, oral pH would be depressed for more episodes. The message for patients of consume all sweets in one episode and preferably following a meal rather than spreading them out throughout the day.

Acidogenic bacteria in dental plaque rapidly metabolize fermentable carbohydrates producing acidic end products. In the mouth these changes over time in response to a challenge, usually a cariogenic food, are known as the Stephan responses, or Stephan curves. The pH of dental plaque under resting conditions, in other words when no food or drink has been consumed, is fairly constant. Differences do exist, however, between the individuals and in different sites within an individual. The response after exposure of dental plaque to a fermentable carbohydrate is that pH decreases rapidly reaching a minimum in approximately five to ten minutes, as shown in this graph. This is followed by a gradual recovery to it's starting value, usually over 30 to 60 minutes, although this can be longer in some individuals.

The following is a short video by Dr. Edward Lowe on the significance of the Stephan curve as it relates to the initiation and progression of caries.

The significance of a Stephan curve, 'kay, shows that, 'kay, the p- plague pH, 'kay, would drop a lot, 'kay, quickly, 'kay, after consumption of a fermentable carbohydrates, 'kay, and then, kep... will drop below what we call critical pH, 'kay, and the which, 'kay, demineralization, uh, of the tooth enamel, 'kay, mineralize, 'kay, can o- occur, 'kay, and there actually it takes quite a long time, 'kay, for the plaque pH to slowly, 'kay, climb up, 'kay, and then, 'kay, return back to, uh, the resting level. That means that there were a period of time, 'kay, where demineralization, 'kay, will occur, 'kay. And then the more frequent the, uh, consumption of the, uh, carbohydrate intake, it means that the longer, 'kay the tank it will be, 'kay, for demineralization to occur. In terms of refinishing, uh, the most important part is that, 'kay, we need to, 'kay, make sure that the, uh, that our patients do not consume, 'kay, fermentable carbohydrate, 'kay, at a high frequency, 'kay, and then we try to promote, 'kay, uh, remineralization, 'kay, in that, 'kay, will, um, make sure that the starches, uh, for build a critical pH for long time.

The following will discuss some of the mechanisms underlying each stage of the Stephan curve. Resting plaque pH: this describes plaque that has not been exposed to fermentable carbohydrates for approximately two hours and generally has a pH between six and seven. The resting plaque pH value for an individual tends to be stable and they remain so for long periods. One example of an exception is if antibiotics have been taken which may alter the oral flora. There are... they are relatively high concentrations of less acidic acetate compared with more acidic lactate at resting plaque, with the amino acids glutamate and proline being predominant. Ammonia, a pH neutralizer, is also present. These metabolic products are present in plaque in much higher concentrations then in saliva, partly because they're constantly produced from the intraand extracellular metabolism of bacterial carbohydrate stores, as well as from the breakdown of salivary gland proteins.

After exposure of dental plague to fermentable carbohydrates, the pH decreases rapidly. This is due primarily to the production of lactic acid in plague, with acidic and propionic acids being simultaneously lost from the plaque. The rate at which the pH decreases is due in part to the microbial composition of dental plaque. In general, if more acidogenic aciduric bacteria is present in plaque, the pH would lower more rapidly. The rate of pH decrease is also dependent on the speed with which plague bacteria are able to metabolize the dietary carbohydrate. While sucrose would be metabolized quickly prompting more rapid decrease, larger molecules like starch would diffuse into plague more slowly because it would need to be broken down before it can be assimilated by the plaque microbes. Another factor that affects the rate of pH decrease is the buffering capacity of unstimulated saliva. The rate at which plague pH decreases is also influenced by the density of plague. Less dense plaque can be penetrated more easily by buffering saliva and oxygen, causing slowly pH decreases than very dense plaque which cannot be accessed via saliva and oxygen.

The gradual recovery of the plaque pH is influenced by various factors. These include the buffer capacity of saliva, whether fermentable carbohydrate remains in the mouth, the pH value which may be unfavorable to bacterial enzyme systems, and the diffusion of acids from plague into saliva or teeth. It's also influenced by base production in plaque. Ammonia from the deamination of amino acids and the breakdown of urea in saliva are examples of reactions that contribute to the pH rise. These bases are important to neutralize acid when carbohydrate intake is moderate. The rise in pH may also be assisted by the removal of acids by bacteria such as those from the genus Veillonella, that use lactate as a substrate metabolizing it to less acidic products, as shown in the... in the chart here.

The fact that saliva is sop beneficial in terms of buffering and neutralizing acidic plaque pH values has stimulated much interest in agents that increase salivary flow rates. Chewing gum or unflavored materials such as paraffin wax, after consuming fermentable carbohydrates leads to an increase in saliva... salivary flow with a concurrent rapid rise in plague pH. This rise has been shown to be closely associated with a rise in bicarbonate buffer capacity, as well as an increase in supply of nitrogenous substrates which are metabolized to basic or less acid end products. The chewing of cheeses rich in nitrogenous compounds gives rise to similar pH increases found with paraffin wax despite the cheese itself being acidic. This is probably due to the breakdown of casein and other cheese proteins, as well as the fact that cheese is a strong sialagogue, or an agent that increases the flow of saliva. Cheese has the added advantage of rising the plaque concentrations of calcium and phosphate and therefore increasing the chance of remineralizing the teeth.

The critical pH is the pH at which saliva and plague fluids cease to be saturated with calcium and phosphate, thereby permitting the hydroxyapatite in dental enamel to dissolve. It's the highest pH at which there is a net loss of enamel from the teeth which, is generally accepted to be about 5.5 for enamel. The solubility of acid varies with pH but is also complicated by the fact that teeth are bathed in saliva which is constantly replenished and supersaturated with apatite whose concentration varies. By increasing the concentration of calcium and or phosphate it's possible to reduce the effect of critical pH so the teeth may be able to withstand lower pH values before they begin to demineralize.

Here are a couple of figures that depict the processes related to the initiation and progression of decay through the production of bacterial acids that then work to remove subsurface mineral from the tooth structure. The first one just shows the carious lesion initiation and progression, the fermentation that produces acids leading to the demineralization process, and the bottom one shows a bit more in terms of the initiation of... and progression of the entire demineralization process as a mineral is removed from the tooth structure.

So in conclusion, understanding how fermentable carbohydrates influence the oral environment and in turn caries risk, is key to helping the dental professional teach the patient about effective caries prevention. To that end, it's important to understand the factors that affect foods cariogenicity and the Stephan responses of plaque pH to challenges by fermentable carbohydrates.

Let's conclude this section by discussing how this information can help you in your practice. First, fully understanding the environment will help you clearly identify evidence-based and scientifically supported interventions to reduce subsurface mineral loss, and making decisions regarding your patients at-home care and reduction of caries risk. Second, information on the environment, 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 recommendation instills a strong sense of trust and confidence in patients and can be far more powerful than simply instructing patients to brush more often. Thank you very much.