Caries Process, Prevention, and Management: The Diet

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Acknowledgement

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Conflict of Interest Disclosure Statement

- Dr. Marshall 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.

Course Contents

Overview

This course is part 4 of a 10-part series entitled *Caries Process, Prevention, and Management*. Dietary carbohydrates are an essential and modifiable component of the caries process. In this course, the caries process is defined, and the science supporting the role of fermentable carbohydrates in the caries process is discussed. How dietary behaviors influence the length of carbohydrate exposure to increase or decrease caries risk is also described. Chairside dietary screening for caries, counseling strategies, and referrals for in-depth dietary counseling are presented.

Learning Objectives

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

- Describe the caries process.
- Identify the role of fermentable carbohydrates in the caries process.
- Describe how dietary behaviors impact caries risk.
- Identify fermentable carbohydrates within food groups and their relative cariogenic potential.
- Screen patients for diet-related caries risk and provide appropriate counselling.

Introduction

Dental caries was identified by the National Institute of Health's 2021 Oral Health in America report as the most prevalent chronic disease in children and adults.[1] The Oral Health Surveillance report (2017-2020) reported that 11% of children aged 2-5 years, 17% of children aged 6-9 years, 10% of adolescents 12-19 years, and 21% of adults aged 20-64 years had untreated caries.[2] Individuals of minority racial or ethnic backgrounds and/or living in poverty have higher rates of caries experience (untreated, filled, missing teeth) across the lifespan.[2] Untreated caries can lead to pain, tooth loss, compromised dietary intake, missed school and work, and lower quality of life. Understanding the role of fermentable carbohydrates in the caries process enables the oral health care practitioner (OHCP) to screen patient's diets for diet-related caries risk and provide appropriate counseling or referrals.

The Dental Caries Process

The classic Keyes model suggests that dental caries result from bacterial fermentation of carbohydrates at the tooth surface (Figure 1). Acid produced as a byproduct of fermentation erodes the enamel in a site-specific manner. The removal of the outer mineral component of the enamel is termed demineralization. Between periods of acid production leading to demineralization, minerals including fluoride from saliva, the diet, and/or water are added back to the enamel. The uptake of minerals into the enamel tissue is called remineralization. The development of a carious lesion reflects an imbalance between demineralization and remineralization when mineral removal outpaces mineral uptake. A cavity is formed when sufficient enamel is removed to create a hole.

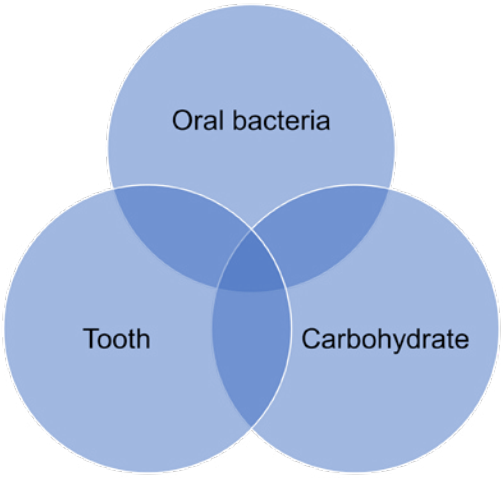

Figure 1. Classic Keyes model.

The oral microbiome refers to the diverse community of microorganisms that live within the oral cavity. The presence of caries reflects a loss of healthy bacteria and an increase in cariogenic bacteria. *S. mutans* were initially thought responsible for the caries process. However, our current understanding suggests that *S. mutans* is one of several bacteria responsible for the initiation of caries, and a host of bacteria and fungi support the progression of the caries process.[3] The bacterial composition of the oral microbiome changes throughout the caries process. Oral hygiene behaviors including brushing and flossing disrupt the oral microbiome, and fluoride can both interfere with bacterial growth and remineralize the tooth surface.

The tooth is composed of several layers – the inner pulp, the dentin, and the outer enamel surface. Although the enamel is relatively inert once it's developed, the enamel is susceptible to insults during development. The insults can increase susceptibility to bacterial colonization and/or bacterial acid attack. Enamel hypoplasia and hypomineralization are enamel defects associated with vitamin A, vitamin D, calcium, and phosphorous deficiencies and/or other environmental exposures during tooth development.[4] Lower maternal serum concentrations of vitamin D have been associated with early childhood caries in their infants at one year of age (Figure 2).[5] Lower overall diet quality and/or malnutrition have been also associated with an increased risk of severe early childhood caries.[6-8]

Caries is a diet-dependent disease; without fermentable carbohydrates, caries do not occur. While sugars are the primary substrate for oral bacteria, starches are also fermentable with modified starches more easily fermented. The nature of carbohydrate might influence the length of time the carbohydrate spends in the mouth. For example, baked starches with sugars are considered more cariogenic than either starches or sugars as they are more retentive. The form (i.e., liquid vs. solid) or texture (i.e., processed snack foods) of the carbohydrate might also influence the time spent in the

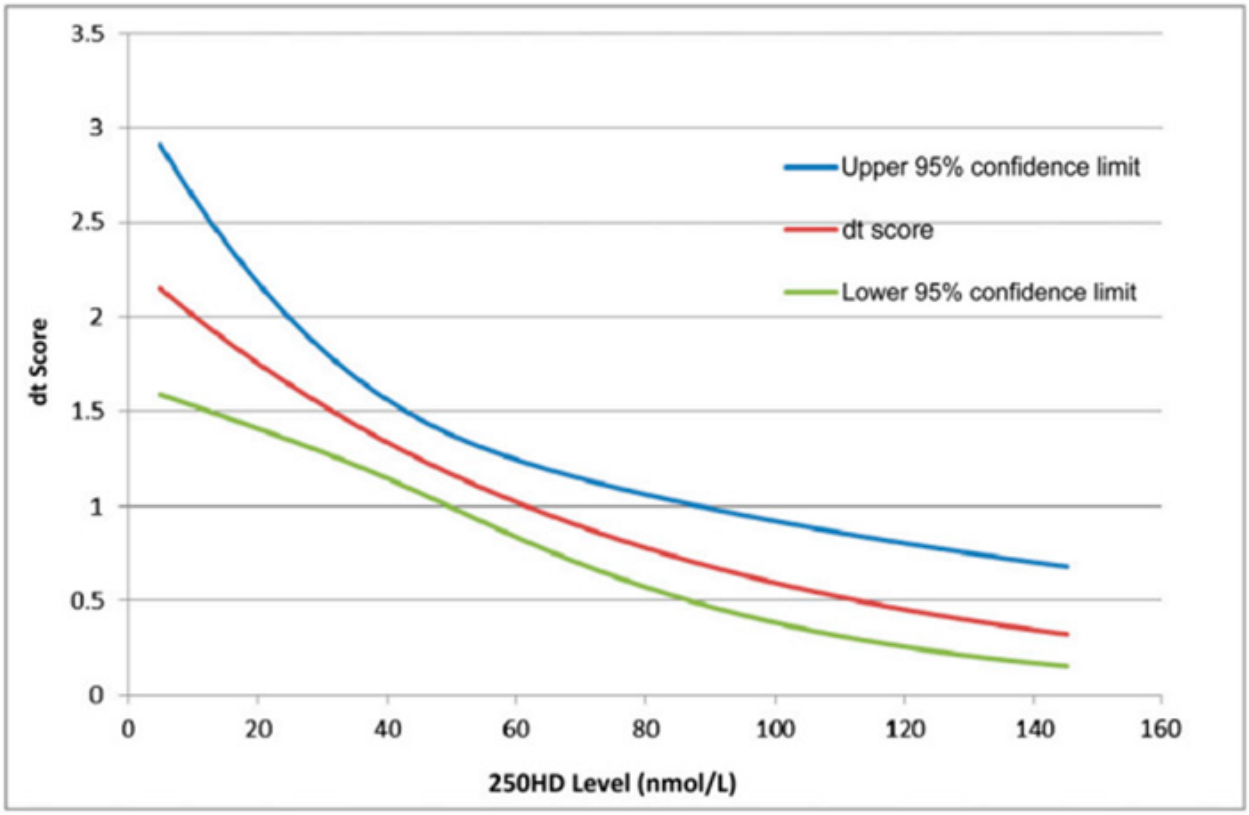

Figure 2. Prenatal vitamin D & ECC[5]

mouth, particularly if the composition is sticky. When considering caries risk, we often talk about the frequency or quantity of carbohydrate intake. However, the length of exposure to the carbohydrate is more relevant to caries risk. Exposure is a function of the frequency and duration of the eating or drinking event. For example, a 12 oz sugar sweetened beverage might be consumed in 20 minutes or over four hours. While it's the same quantity and the same frequency, the exposure times are very different. Individuals with oral motor skill deficiencies that result in pocketing of foods and/or reduced clearance might have extended exposure times which increases their caries risk.

Dietary Carbohydrates

Carbohydrates, which include sugars, starches, and fibers, are macronutrients (i.e., nutrients consumed in large quantities – fat, protein, carbohydrates, and water), provide 4kcal/gram, and are naturally present in fruits, vegetables, grains, and dairy foods. The term fermentable carbohydrates refer to sugars and starches that are metabolized by oral bacteria to produce acid. From a chemical perspective, fermentable sugars are categorized as mono- (i.e., fructose, glucose, and galactose) or di-saccharides (i.e., sucrose, maltose, and lactose). Starches are composed of long chains of glucose. The food industry hydrolyzes starches to produce short glucose chains for functional and/or sensory properties; the resulting modified starches are more cariogenic than the original starches. More complete hydrolyzation followed by isomerization results in high fructose corn syrup (HFCS) which is used as a sweetener in processed foods. Finally, the term 'added sugars' refers to sugars that are added to foods during production, by the home cook, or at the point of consumption. Similarly, 'free sugars' refers to sugars added to foods and beverages as well as those naturally present in honey and fruit juices. Both the United States Department of Agriculture's Dietary Guidelines for Americans and the World Health Organization recommend limiting added and free sugars, respectively, to less than 10% of total energy intake.[9,10]

Science Supporting the Role of Dietary Carbohydrates in the Caries Process

Stephan Curve

Acid produced by oral bacteria during fermentation of carbohydrates dissolves or demineralizes the enamel and/or dentin during the caries process. The extent to which acid is produced during fermentation of foods with different macronutrient compositions, meal patterns, or intake sequences can be identified and compared using plaque pH studies. Based on the assumption that acid produced by oral bacteria causes caries, the acid produced during fermentation can be quantified and the fall in plaque pH reflects the cariogenicity of the test food. The pH gradually returns to normal following removal of the acid through salivary clearance or buffering. The Stephan curve, the plot of the pH of plaque over time, allows the visualization of the food's cariogenic potential (Figure 3).[11] The critical pH is defined as the pH above which remineralization occurs, and below which demineralization occurs. Although individual critical pHs vary, the critical pH is generally defined as a pH of 5.5.

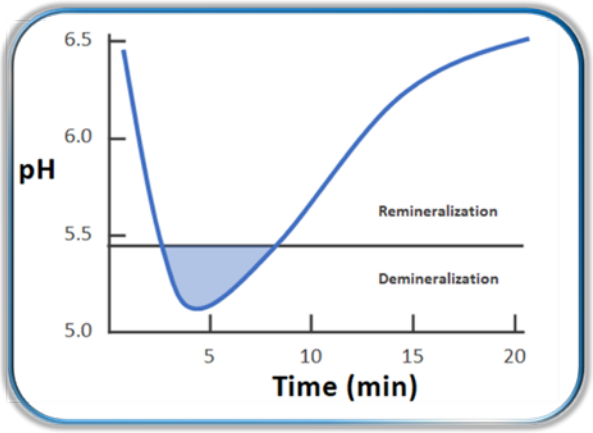

Figure 3. The Stephan Curve[11]

Plaque pH studies have been used to confirm that sugars are readily fermented by oral bacteria. Fibrous foods that require chewing increase the rate of acid removal and reduce caries risk. The impact of the carbohydrate form on the critical pH has also been determined. For example, Naval et al investigated the differences in plaque pH in response to different beverages following a sugary snack (Figure 4).12 First, they had subjects eat or drink a sugared cereal, a 10% sucrose solution (positive control), or a 10% sorbitol solution (negative control). The subject's plaque pH fell in response to the sugared cereal and sucrose challenges, but not the sorbitol solution. Navel et al then had the subjects drink whole milk (mixed protein, carbohydrate, fat), 100% apple juice (all sugar)

or tap water after eating the sugared cereal (Figure 5).12 The pH returned to normal faster after milk consumption, followed by water, and finally juice. Similar plaque pH studies have been used to confirm that oral bacteria are unable to metabolize saccharine, aspartame, acesulfame K, or sugar alcohols. As the low/no calorie sweeteners are not acidogenic, they are not cariogenic.

Hopewood House Study

The Hopewood House was a home in rural South Whales, Australia that provided care for economically deprived children from birth through 12 years of age in the 1950s.13 While living in Hopewood House, the children consumed a lacto-ovo vegetarian diet with emphasis on whole grains and raw vegetables. Sugars and refined starches were quite limited.

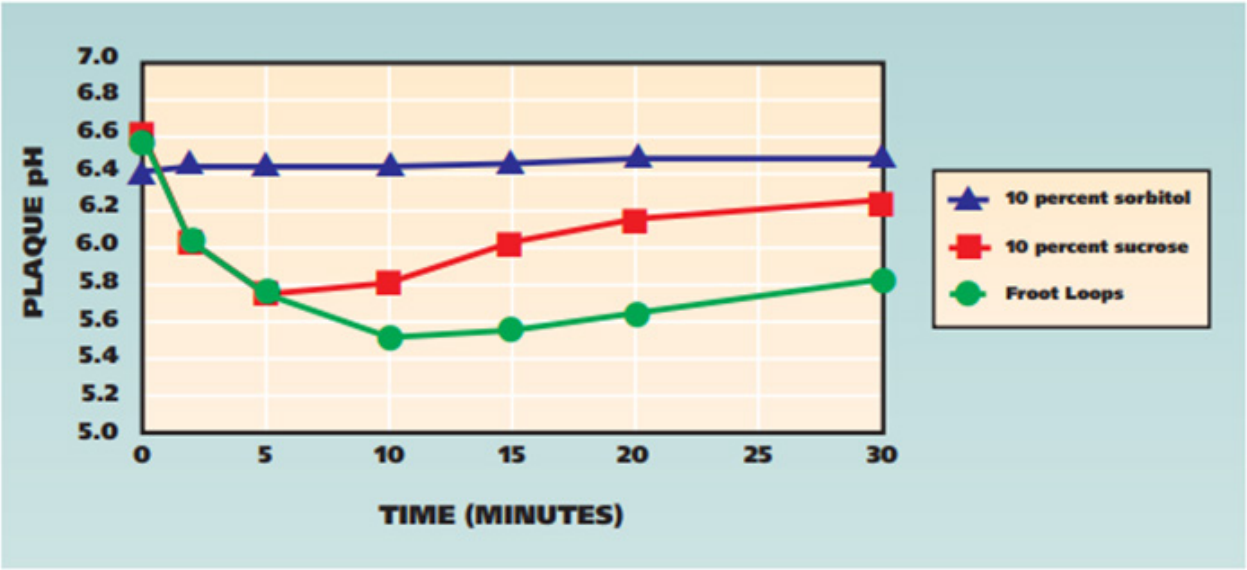

Figure 4. In vivo mean plaque pH after participants rinsed with sucrose or sorbital or consumed Froot Loops (Kellog's, Battle Creek, Mich.).[12]

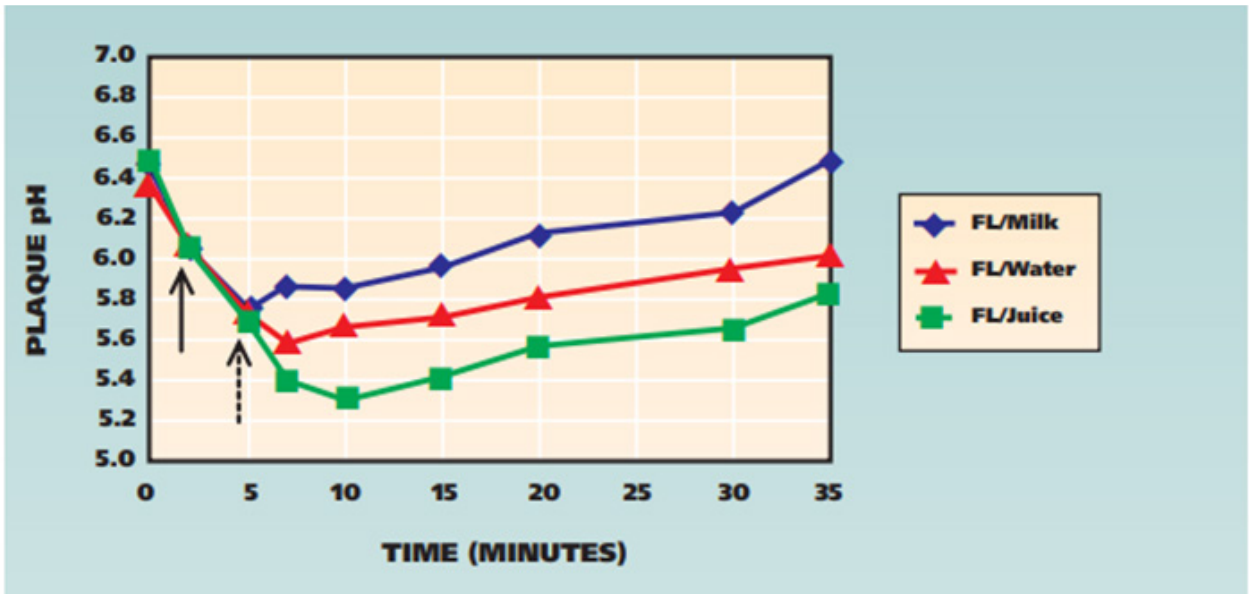

Figure 5. In vivo mean plaque pH after participants consumed Froot Loops (FL) (Kellog's Battle Creek, Mich.) followed by FL and whole milk (FL/milk), FL and 100% apple juice (FL/juice) or FL and tap water (FL/water) (n=20). The solid arrow indicates two minutes after complete consumption of FL. The dotted arrow indicates the time of beverage consumption.[12]

While in residence at Hopewood House, the caries prevalence of children was much lower than that observed in children attending state schools consuming a diet containing sugars and refined starches (Figure 6). At 12 years, the children moved out of Hopewood House and adapted the diet of the local community at which time their caries incidence paralleled that of their peers attending the state schools. This study suggests that teeth do not acquire permanent protection against dental caries.

Vipeholm Study

How exposure to retentive and nonretentive sugars both at and between meals influences caries risk was explored at a hospital setting in Vipeholm, Sweden in the 1940s.[14] The hospital was divided into 12 separate wards preventing sharing of the different diets between subjects. Subjects within a ward were provided various carbohydrate containing foods (i.e., breads, toffee, caramel, sucrose); access was provided at meals or both at and between meals (Figure 7). Caries incidence was higher for sweets proved both at and between meals than when provided at meals only.

Sugars Intake

In 2014, Moynihan and Kelly published a systematic review sponsored by the World Health Organization (WHO) identifying the effect of restricting sugars intake on caries in adults and children.[15] The authors sought to identify all randomized clinical trials and observational studies published since 1950 exploring effects of individual sugars (i.e., sucrose) and/or sugar categories (i.e., added sugars, total sugars) on dental caries. Specifically, they were interested in the effect of lowering sugars intakes as well as the effect of sugars intakes below 10% of energy intakes in both adults and children. They reported that dental caries are lower when free-sugars intakes are less than 10% of energy intake across the lifespan. Following Moynihan and Kelly's publication, the WHO recommended limiting free sugars intakes to less than 10% of energy intakes to reduce risk of caries.[10]

Sugar-sweetened Beverages (SSB)

OHCP have long used the terms 'nursing bottle caries' and 'Mt Dew mouth' to refer to dental caries specifically associated with extensive

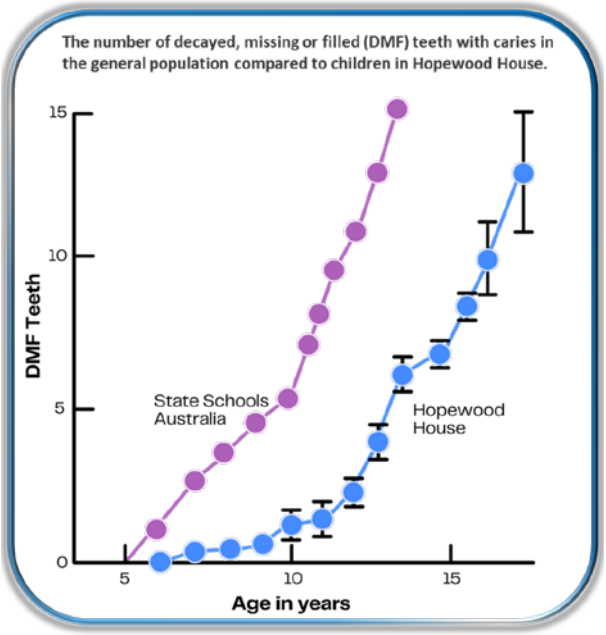

Figure 6. Adapted from: Harris R. The biology of the children of Hopewood House[13]

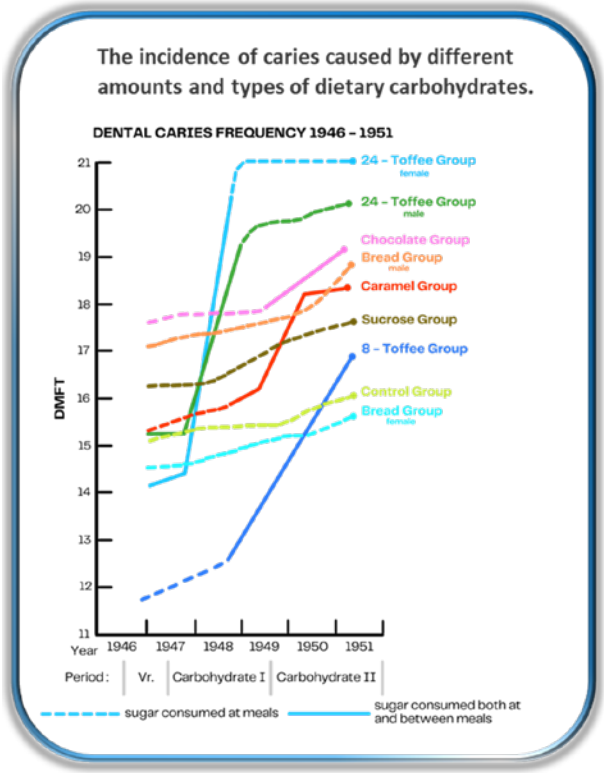

Figure 7. Adapted from: Gustafsson BE, et al. The Vipeholm dental caries study; the effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years.[14]

bottle use by infants and SSB intakes in older children and adults. In both disease processes, the anterior maxillary teeth experience significant decay often requiring extraction. In 2003 Marshall et al investigated associations among age 1-5 year beverage intakes, dairy food intakes, nutrient intakes, and overall diet quality and 5 year caries experience in participants of the Iowa Fluoride Study.[16] They reported that high intakes sugar-sweetened soda-pop intake and beverages made from powders were associated with increased caries.[16] They subsequently explored the meal or snack timing of individual beverages and reported that daily intakes of sugar-sweetened soda pop, but not 100% juice or milk/formula were associated with dental caries at 5 years of age.[17] More recently, they explored the effect of age 1-17 year beverage intakes on caries at age 17 while controlling for fluoride intake, toothbrushing, sex, and socioeconomic status. They reported that each additional 8 oz of daily SSB increased the expected decayed, filled, surface attack rate (i.e., caries experience adjusted for tooth surfaces; DFSAR) by 42%, while each additional 8 oz of daily juice or water/sugar free beverage decreased expected DFSAR by 53% and 29%, respectively (Figure 8).[18] It is unclear why juice which contains sugars is not associated with increased caries in this study; however, multiple other investigators have reported similar findings.[19,20]

Table 5. Effect of beverage intakes on expected DFSAR[a] at age 17 years, adjusted for other beverage intakes, fluoride intake, toothbrushing frequency, sex, and SES[b] in Iowa Fluoride Study participants (AIC[c] = 1399.1)

Variable of interest	Estimated multiplicative effect of exposure (95% CI[d])[e]	*P* value
Milk[f]	0.87 (0.69-1.11)	.254
Juice[fg]	0.47 (0.27-0.83)	.009
SSB[fh]	1.42 (1.05-1.92)	.025
Water/SFB[fi]	0.71 (0.54-0.93)	.014
Total fluoride, excluding SSB fluoride[j]	1.10 (1.01-1.20)	.029
Toothbrushing[k]	0.57 (0.38-0.86)	.008
Female indicator	1.55 (1.11-2.18)	.011
Baseline SES—low	1 (reference)	—
Baseline SES—middle	0.82 (0.53-1.27)	.366
Baseline SES—high	1.13 (0.73-1.76)	.582

[a]DFSAR = decayed and filled surfaces.
[b]SES = socioeconomic status.
[c]AIC = Akaike Information Criterion.
[d]CI = confidence interval.
[e]Estimated multiplicative effect of exposure of beverage on expected DFSAR from gender and socioeconomic adjusted generalized linear models based on the negative binomial distribution with a log link function.
[f]Effect for each additional 8 oz beverage/d.
[g]100% juice, including juice drinks before age 9 years.
[h]SSB = sugar-sweetened beverages, including liquid juice drinks after 9 years.
[i]Water/SFB = water and other sugar-free beverages.
[j]Effect for each additional 0.1 mg fluoride/d.
[k]Effect of each additional toothbrushing event/d.

Figure 8. Marshall's Age 17 Study[18]

Starches

Foods items composed primarily of starch include plain potatoes, rice, pasta, and some breads. Oral bacteria can metabolize starches to sugars in the mouth; however, the length of time required is extensive. Thus, pure starches are not considered cariogenic. Palmer et al reported that intakes of 'low cariogenic' foods including rice, pasta, cooked vegetables, milk, fresh fruit, and unsweetened yogurt were similar between children with and without early childhood caries.[21] Marshall et al similarly reported that primarily starch foods consumed at snacks did not increase caries risk while those consumed at meals were associated with a lower caries risk in young children.[17] On the other hand, Palmer reported that processed starches (i.e., breads, chips, crackers and sweetened cereals) were associated with an increased caries risk.[21]

Frequency of Intake

Evaluation of Stephan curves tracking individual or multiple eating events suggests that the plaque pH requires about 30-60 minutes to return to normal levels following an eating event. Thus, prolonged snacking and/or multiple eating events are thought to increase caries risk. Palmer reported that children with early childhood caries consumed foods and beverages more frequently than children without caries (Figure 9).[21] Marshall reported that increased snacking at ages 1, 2, 3, and 4 years of age were associated with increased caries risk at 5 years of age.[17] Secular dietary patterns have devolved in recent times with many individuals having unstructured eating patterns. Unstructured eating has been associated with an increased caries risk.[22]

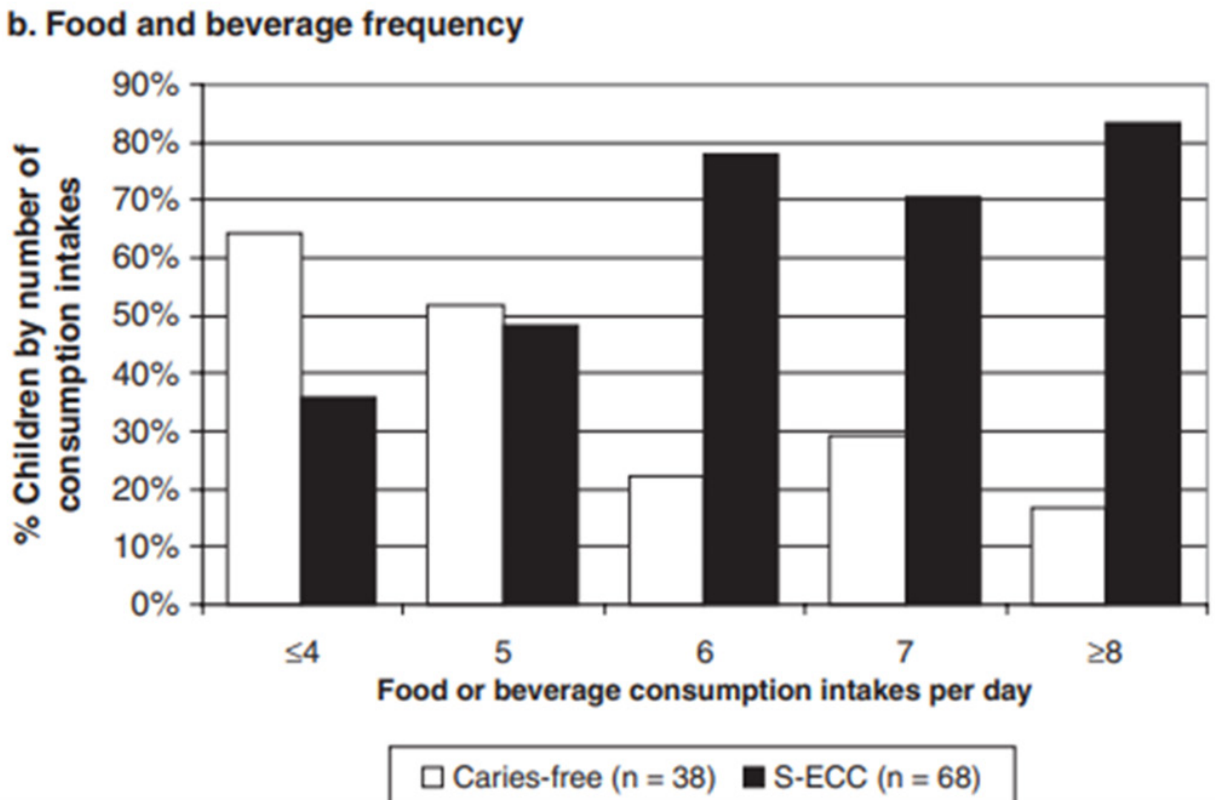

Figure 9. Palmer's Frequency[21]

Food Composition

We do not typically eat macronutrients in isolation; rather, we eat foods composed of macronutrients. Translation of caries science to clinical practice requires a thorough understanding of caries risk associated with macronutrients and macronutrient composition of foods (Figure 10). Proteins and fats are not subject to fermentation by oral bacteria, and therefore, are not cariogenic. Proteins and fats accompanied by calcium might have beneficial effects as the calcium could remineralize enamel or dentin. The likelihood of oral bacteria fermentation determines the individual carbohydrate risk. Complex carbohydrates are of limited caries risk, processed starches are of significant caries risk, and sugars and baked starches with sugars are of high caries risk. It is worth noting that the carbohydrate's origin is not relevant to caries risk; oral bacteria do not distinguish natural or organic sugars. Thus, organic honey and/or raw sugar are highly cariogenic.

Most foods are composed of mixed macronutrients. The presence of fat and/or protein within a food or consuming foods high in fats/proteins as part of mixed meals can reduce the caries risk associated with carbohydrates. When evaluating the caries risk of food groups, it is important to consider the presence of carbohydrate as well as the degree of carbohydrate processing (Figure 11). In doing so, one can quickly identify the overall caries risk of a food. For example, fresh fruit contains several sugars; given the presence of fiber and water within the fruit, the caries risk associated with fresh fruits is minimal. Removal of either the water (dried fruit) which concentrates the sugars or fibrous material (juice) which produces a sugared beverage increases the caries risk compared to the original fruit.

Dietary Screening for Caries Risk

The objective of dietary counseling in oral health is to reduce caries risk, support soft tissue health (i.e., periodontal tissue), reduce

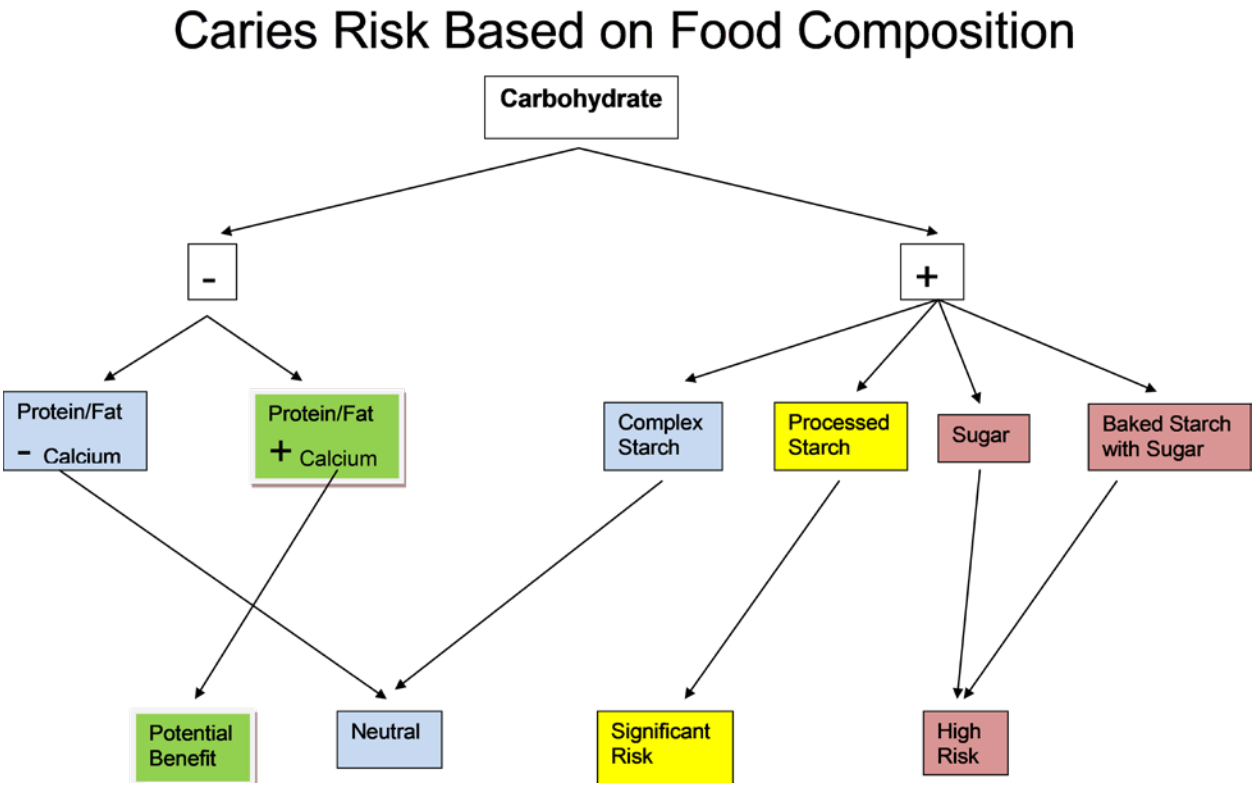

Figure 10. Caries Risk Based on Food Composition

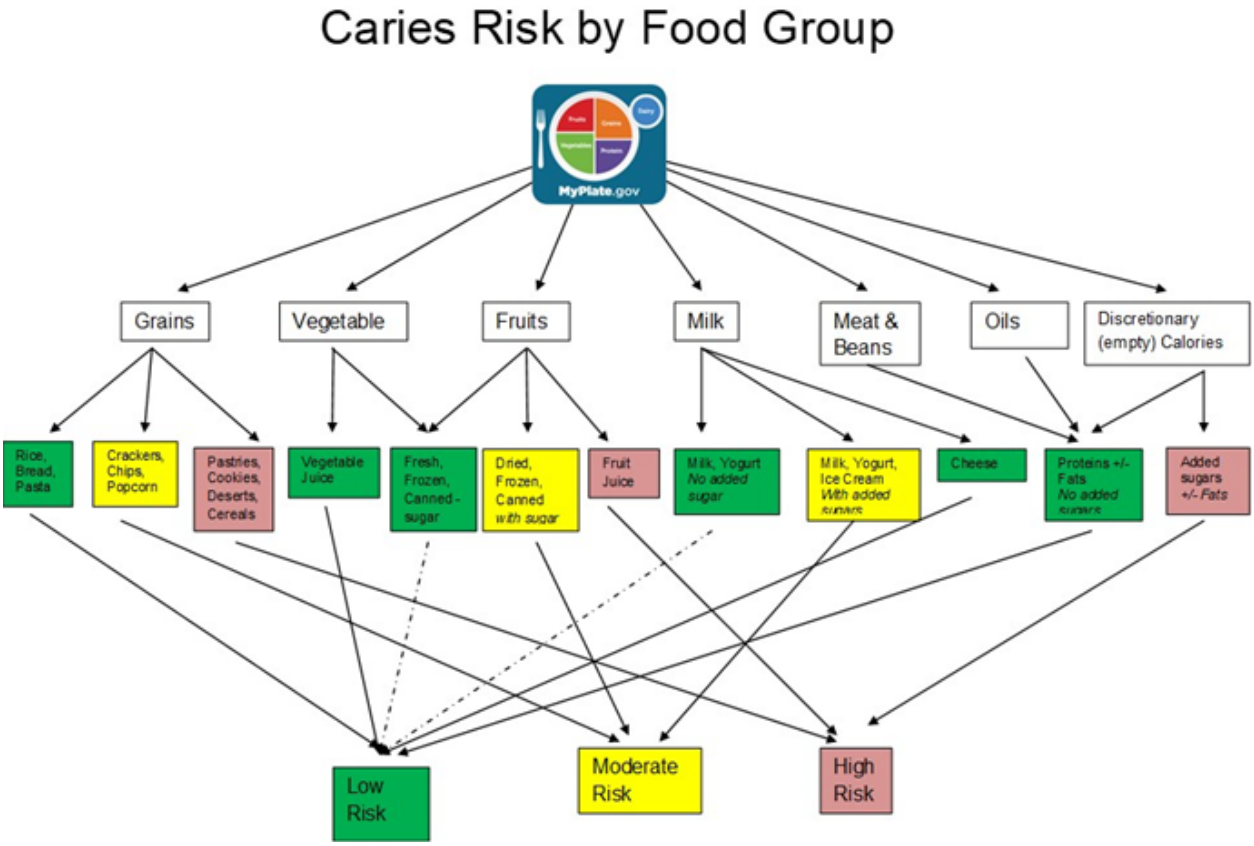

Figure 11. Caries Risk by Food Group

oral cancer risk, and support systemic health.[23] Extensive dietary counseling is neither practical from a time perspective nor within an OHCP's scope of practice. However, dietary screening to identify diet-related caries risks and providing guidance to reduce oral disease should be standard practice for all clinic patients. For practical purposes, dietary guidance to reduce caries risk is consistent with dietary guidance to support soft tissue health and reduce systemic disease.[24] When patients present with complex dietary needs, referrals to other health care providers including registered dietitians is appropriate.

The objective of dietary screening is to identify healthy patients who are at risk of oral disease due to their marginal dietary habits. For patients presenting with oral disease, similar screening questions will identify dietary factors contributing to the disease process. Additional discussion to understand dietary behaviors is appropriate to provide dietary recommendations that are doable for individual patients. Dietary recommendations should provide guidance for the patient to achieve 'better' dietary behaviors and is a negotiation process with the patient. After all, the patient must be motivated and able to execute the recommendations in their daily life.

Screening components: A dietary screen is designed to identify specific high-risk behaviors and enable anticipatory guidance.[22] Contemporary caries research suggest that both high SSB intakes and frequent carbohydrate exposures are significant risk factors for caries. For patients with high SSB intakes, dietary recommendations to lower SSB intakes inherently reduce energy intake. Anticipatory guidance is necessary to provide the patient with strategies to avoid caffeine withdrawal if the SSB contained caffeine as well as provide the patient with better food/beverage choices to replace lost energy. Recommendations for better food choices will complement soft tissue and systemic health. To make such recommendations, the clinician must understand the patient's food preferences. Finally, as a health care professional screening for weight changes consistent with undiagnosed disease is appropriate. With these considerations in mind, the following screening questions are appropriate:

- Meal structure
  - How many meals and snacks do you eat daily?
    - o The desired outcome is 3 meals and up to 3 snacks per day.
    - o Greater eating frequency is associated with caries risk.[17,21]
  - How would you describe your eating behaviors? Structured – as in eating at regular times on most days? Or unstructured – eating throughout the day with no regular pattern?
    - o The desired outcome is structured eating events.
    - o Unstructured eating events are associated with caries risk.[22]
- Sugar-sweetened beverages
  - How much sugar-sweetened beverages do you drink per day? This includes soda-pop, juice drinks, energy drinks, sweetened coffee or tea, sports drinks, etc.
    - o Consistent with WHO and USDGA recommendations, 8 oz is the recommended upper limit of SSB intake.
    - o Greater than 20 oz/day is associated with caries risk.[22]
  - How long does it take you to drink your sugar-sweetened beverages?
    - o Consumption limited to less than 30 minutes/day is recommended with consumption preferable at meals and snacks.
    - o Greater than 30 minutes/day is associated with caries risk.[22]
- Sugared candy
  - How often do you eat sugared candy or medicated lozenges?
    - o The desired outcome is no more than once a day.
- Compliance with MyPlate (Figure 12)[25]
  - How many food groups (i.e., fruits, vegetables, proteins, grains, dairy) do you eat per day?
    - o The desired outcome is an intake of all food groups consistent with age and sex MyPlate recommendations.[25]
    - o Recommendations to encourage intakes consistent with MyPlate are appropriate to replace energy from SSBs, support soft tissue health, and support systemic health.
- Unintentional weight gain or loss
  - Have you gained or lost more than 10 pounds during the past six months?

- The desired response is no unintended weight change.
- An unintentional weight gain or loss of 10 pounds or more over a six-month period is not normal and may be consistent with undiagnosed systemic disease. A referral to the patient's primary medical provider is appropriate.

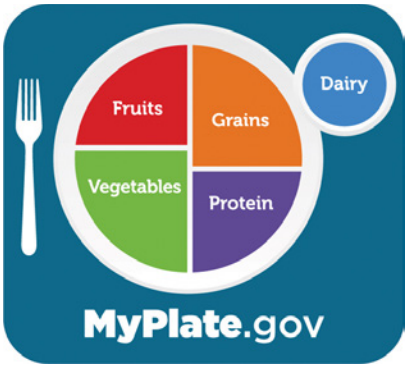

Figure 12. Compliance with MyPlate[25]

Most of us do not consume perfect diets, and many patients have extensive dietary needs. Rather than overwhelming patients with multiple recommendations, the OHCP should engage the patient in the conversation. Clearly communicate the rationale for the desired change to ensure patient understanding. With that knowledge, what is the patient's priority? What might they be willing to change? What might be the ripple effects of choosing to implement a recommendation? Providing patients with strategies to address the ripple effects will support success in making the desired changes and facilitate improved dietary habits.

Many patients face economic barriers that limit their ability to partake in desired oral hygiene behaviors and select healthier diets. OHCP can screen for both food and water insecurity and make appropriate referrals for patients facing such barriers. Food insecurity is defined as having limited or uncertain access to food due to economic or social circumstances.[26] OHCP are in position to screen for food insecurity, and refer patients with food insecurity to their local food banks and social services (Figure 13).[27] Water insecurity is defined as having uncertain access to potable water, and has been associated with increased intake of SSBs.[28] Although screening tools for water insecurity have not been validated, asking patients if they have running water and if they are comfortable drinking their tap water is appropriate. For patients with limited access and/or who are uncomfortable drinking their tap water, providing guidance to resources for safe drinking water is appropriate.

Summary

Dietary carbohydrates, particularly added sugars, are an essential part of the caries process. Acid produced during bacterial fermentation of these carbohydrates erodes the enamel producing a site-specific lesion. Dietary behaviors that increase the frequency and/or length of eating increase caries risk. Recommendations to consume structured meals and limit intake of added sugars including SSB are indicated to reduce caries risk.

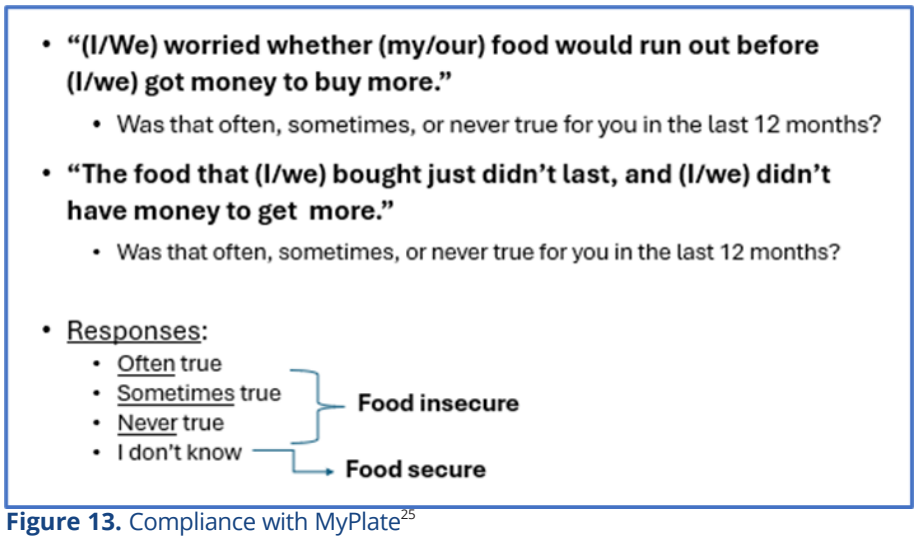

Figure 13. Compliance with MyPlate[25]

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/ce711/start-test*

1. Which of the following statements is true about caries risk?

A. Both starches and sugars can be metabolized by oral bacteria to increase caries risk.
B. Starches and sucrose are equally cariogenic.
C. Increased frequency of sugars intake reduces caries risk.
D. Structured meal patterns increase caries risk.
E. Calcium increases sugar's cariogenicitiy.

2. What is one main finding of the Australian Hopewood House study?

A. As part of a lacto-ovo vegetarian diet, added sugars do not increase caries risk.
B. Limiting dietary sugars and refined starches reduces caries risk.
C. Restricting sugars intake during childhood provides permanent protection against caries.
D. Caries risk of children living in Hopewood House did not increase when they moved into the local community.

3. Deficiencies of vitamins A and D during tooth development are associated with enamel hypoplasia and hypomineralization, both of which increase caries risk.

A. True
B. False

4. What is one main finding of the Swedish Vipeholm study?

A. Liquid foods cause little to no caries.
B. When study subjects ingested sugar with meals, a lower caries rate was observed than when study subjects ingested the same amount of sugar as snacks between meals.
C. Retentive foods are not significantly cariogenic.
D. Only teeth with pits and fissures are prone to caries.

5. Which of the following statements is true?

A. Hydrolyzed starches are short glucose chains that are not cariogenic.
B. High fructose corn syrup (HFCS) is produced by starch hydrolysis and isomerization and is cariogenic.
C. The term 'added' sugars refers to only those sugars added by the home chef.
D. The World Health Organization recommends limiting free sugars intakes to less than 25% of energy intake.
E. Sugar-sweetened beverages (SSBs) are generally high in starch and sugar.

6. Foods containing which of the following macronutrient compositions are most likely to increase caries risk?

A. Protein and/or fat without calcium.
B. Protein and/or fat with calcium.
C. Complex starches
D. Processed starches
E. Baked starches with sugar

7. The objective of dietary screening for caries risk is to:

A. Identify food choices and dietary behaviors that increase caries risk.
B. Refer patients to other healthcare providers.
C. Identify oral hygiene behaviors.
D. Monitor diet-related systemic conditions.
E. Identify preferred foods and beverages.

8. Which of the following is NOT a recommended dietary habit to reduce caries risk?

A. Consume 3 meals /day
B. Consume 0-3 snacks/day
C. Consume foods and beverages at structured eating events
D. Consume more than 20 oz SSB /day
E. Consume sugared candy less than once/day

9. Food insecurity is a barrier to consuming a healthy diet. Food insecurity is defined as:

A. A lack of access to running tap water due to economic circumstances.
B. Limited or uncertain access to food due to economic or social circumstances.
C. Limited or uncertain access to potable water due to corrosive pipes.
D. Limited full scale grocery stores within walking distance.
E. Limited food preparation skills.

10. The critical pH is defined as

A. The lowest pH following consumption of a sugar or starch.
B. A pH of 7.2.
C. The pH below which demineralization occurs, and above which remineralization occurs.
D. Salivary clearance of acid produced during bacterial fermentation of carbohydrates.
E. The resting pH in the mouth.

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