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Dental Caries Etiology: A Modified Six-Factor Model and Venn Diagram

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Abstract: Dietary fermentable carbohydrates or sucrose may be metabolized by cariogenic bacteria at a specific tooth surface site, which may result in an episode of the site having a pH of 5.5 or less, that may cause temporary net demineralization. Numerous demineralization episodes at a specific tooth surface may result in a carious lesion if saliva does not remineralize the surface after each episode of net demineralization. However, this general model of carious lesion causation does not explain why carious lesions occur at specific tooth surface sites. This article reviews dental caries etiology, and suggests a modified model of dental caries etiology, proposing that dental caries results from five known factors, namely a combination of a tooth surface site, frequent exposure to fermentable carbohydrates at that tooth site, cariogenic bacteria fermenting the carbohydrates, poor oral hygiene or dietary habits, inadequate natural anti-caries cleansing and re-mineralization processes, and a sixth factor, consisting of a factor that facilitates the retention of plaque or fermentable carbohydrates at a specific tooth surface site. This article describes forty different examples of facilitators of carbohydrate and plaque retention at tooth surface sites that help facilitate net demineralization at specific tooth surfaces, when combined with other caries-inducing factors. This caries etiology model is used to modify the conventional 3-factor Venn diagram illustrating caries etiology, resulting in a more comprehensive 6-factor Venn diagram.

Keywords: Dental Caries, Etiology, Causality, Risk Factors, Diet, Cariogenic, Tooth Demineralization

Introduction

Dietary habits and oral hygiene habits influence the rate of formation of a carious lesion. Good oral hygiene habits, such as tooth brushing, flossing, or rinsing with water after every meal or snack, reduce the rate of carious lesion formation by reducing the total amount of time per day that sugary foods or drinks, or fermentable simple carbohydrates (Kashket *et al*., 1994) or cariogenic bacterial biofilm, are in contact with carious tooth surfaces. Routine rinsing with topical fluoride dentifrices (Newbrun, 1989; Shahid, 2017) increases tooth surface resistance to dissolution by acid produced by carious bacteria. Salivary phosphates, buffering agents, pellicle proteins, enzymes and calcium ions help protect and remineralize tooth surfaces damaged by acid attack from cariogenic bacteria. The avoidance of frequent consumption of sticky sugary foods such as sticky dry simple carbohydrates such as white flour pretzels or crackers, prevents carbohydrate retention at carious tooth surfaces (Bibby *et al*., 1951) fissures or pits, where such retention increases the total amount of time per day that fermentable carbohydrates are in contact with, and providing nutrition to, cariogenic bacteria on or inside teeth surfaces. Avoiding consumption of sugary drinks prevents smooth surface tooth demineralization or caries (Bibby *et al*., 1951; Nakayama and Mori, 2015; Almasi *et al*., 2016; Tahmassebi *et al.,* 2006; Armfield *et al*., 2013). Consuming sugary foods or simple carbohydrates with meals (Gustafsson *et al*., 1953) also helps reduce the total time per day that fermentable carbohydrates are in contact with carious tooth surfaces.

The cause of carious lesions is net demineralization at the lesion site. An episode of demineralization may

occur when cariogenic bacteria at a tooth surface ferment sucrose, obtained from the oral environment, to produce acid. If the resulting acid produces a pH at the caries site that is less than 5.5, an episode of demineralization occurs. (Newbrun, 1989) If saliva only partially remineralizes the site before the next episode of demineralization occurs, then the episode causes net demineralization at that site. With each episode of net demineralization, the carious lesion expands. The lesion will expand at a faster rate if demineralization episodes occur frequently per unit of time at that site, or if the total amount of demineralization exceeds the total amount of remineralization, over the multiple episodes of demineralization and remineralization at that site.

This general model of caries formation is evidence-based and essentially describes five factors that affect the rate of formation of a carious lesion (cariogenic bacteria, a tooth surface, oral hygiene and food consumption habits, fermentable carbohydrates, and anti-caries factors such as salivary remineralization or fluoride usage). However, this model does not explain why a specific carious lesion forms at a specific tooth surface site. This article presents a theory that a sixth factor is also necessary for a carious lesion to develop at a specific tooth surface site, consisting of a factor that facilitates the retention of plaque or fermentable carbohydrates at a specific tooth surface site. This factor can consist of a static morphological characteristic on a tooth surface that facilitates the continuous retention of plaque or fermentable carbohydrates at a specific tooth surface site; or a dynamic intra-oral process that over time causes morphological changes to a tooth surface, such as to facilitate retention of plaque or fermentable carbohydrates at a specific tooth surface site; or a characteristic of an artificial dental prosthesis or restoration that facilitates the continuous retention of plaque or fermentable carbohydrates at a specific tooth surface site. This article describes forty different examples of facilitators of plaque retention or fermentable carbohydrate retention at specific tooth surface sites. By combining an awareness of the conventional 5-factor caries formation model with the ability to identify specific factors for retaining plaque or fermentable carbohydrates at specific tooth surface sites, a dentist can explain how a diagnosed carious lesion could have formed at the specific tooth surface site of its formation. This article presents a modification of the conventional, incomplete 3-factor Venn diagram of carious lesion formation (tooth surface + cariogenic bacteria + fermentable carbohydrates = caries), and replaces the conventional Venn diagram with a more comprehensive 6-factor Venn diagram model of carious lesion formation (Fig. 1).

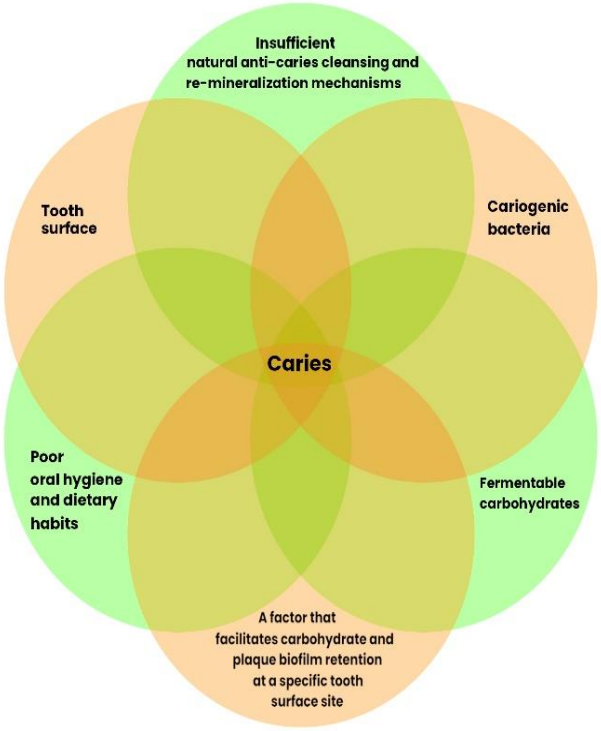

Fig. 1: A modified 6-factor Venn diagram illustrating caries etiology. "Time" is a tacit, underlying carrier facilitating factor that is not included in this diagram

Examples of Facilitators of Site-specific Retention of Cariogenic Plaque and Fermentable Carbohydrates

Plaque and Carbohydrate Retention Facilitated by Static Morphological Features on a Tooth Surface

Small pits and fissures on posterior teeth occlusal surfaces can retain microscopic amounts of cariogenic plaque and fermentable carbohydrates. Tooth brush bristles or salivary cleansing mechanisms may not be able to penetrate pits and fissures to remove the fermentable carbohydrates, resulting in prolonged retention of cariogenic plaque and fermentable carbohydrates at these sites, and facilitating net demineralization at these sites (Carvalho *et al*., 2016; Ismail *et al.,* 2007).

Fissures or pits on maxillary anterior teeth's lingual surfaces can also retain microscopic amounts of fermentable carbohydrates that toothbrushing cannot dislodge (Ismail *et al.,* 2007).

Fermentable carbohydrates may become trapped between inter-proximal spaces of anterior or posterior teeth due to the morphology and tightness of inter-proximal contacts (Ismail *et al.,* 2007; Dablanca-Blanco *et al*., 2017; Li *et al*., 2016). Saliva may cleanse carbohydrates within the embrasure space, but may not cleanse carbohydrates

trapped within the contact point. Consequently, fermentable carbohydrates linger at the contact point, especially if the patient does not floss.

Posterior teeth buccal pits can retain tiny amounts of cariogenic plaque or fermentable carbohydrates that cannot easily be removed by toothbrush bristles (Ismail *et al.,* 2007).

If a buccal or lingual tooth surface is undercut relative to the coronal aspect of the tooth, due to the natural morphology of the tooth, or due to a heavy lingual inclination of the tooth, cariogenic plaque and fermentable carbohydrates may stagnate at the buccal or lingual surfaces.

A thin space between posterior buccal tooth surfaces and the buccal mucosa or ramus aspect of the mandible may facilitate carbohydrate retention at the buccal surfaces of posterior teeth facing the tight areas. Toothbrushes may be too wide to clean out these food particles, and a patient may not be able to see food trapped in these posterior areas.

Plaque and Carbohydrate Retention Facilitated by Static Morphological Features Involving Two or More Teeth

Tooth malposition may result in inter-proximal tooth surfaces that cannot be flossed due to tight contacts or due to a difficult angle of approach to bring floss to the area, causing fermentable carbohydrate retention at these inter-proximal contact areas (Fig. 2).

A tooth with extreme lingual or buccal displacement may form a 3-wall "chamber" with two neighboring teeth, where two walls of the chamber are made up of one side of each neighboring tooth, and a third wall is provided by the displaced tooth, forming a triangle-shaped chamber. Fermentable carbohydrates may collect within the chamber, which may be difficult to brush or floss away.

A tooth that is rotated 90°, such that the buccal and lingual surfaces of the tooth are in inter-proximal contact with the mesial and distal surfaces of neighboring teeth, may result in inter-proximal contact areas that are difficult to floss, facilitating plaque and carbohydrate retention.

Teeth that form inter-proximal contacts where the marginal ridges of the teeth are at different levels may collect fermentable carbohydrates inter-proximally more than would teeth with leveled opposing inter-proximal contacts (Li *et al*., 2016), facilitating inter-proximal carbohydrate retention.

A posterior tooth may be mesially inclined such that a convex aspect of the mesial surface of the tooth fits into a concave aspect of the distal surface of the neighboring tooth, resulting in an inter-proximal contact morphology that may retain fermentable carbohydrates and that may be difficult to floss. This morphology is sometimes observed at the contact points between mesially inclined mandibular third molars and the neighboring second molar. (Prajapati *et al*., 2017; Marques *et al*., 2017; Boffano *et al*., 2010).

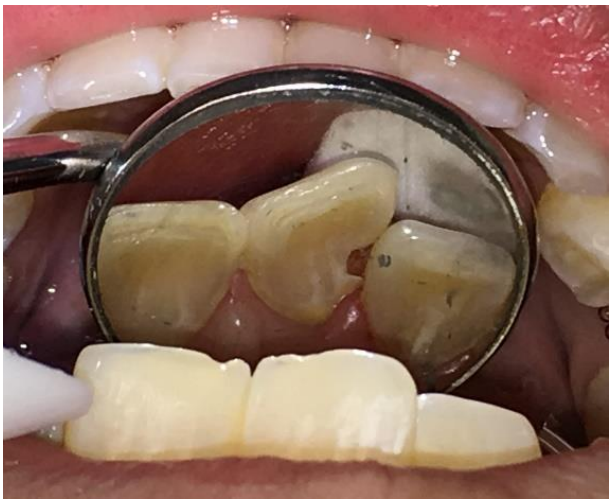

Fig. 2: A malposed right maxillary central incisor where the undercut cervical 1/3 was tightly wedged into the neighboring lateral incisor and difficult to floss. The contact area morphology facilitated plaque and carbohydrate retention, and decay

Plaque and Carbohydrate Retention Facilitated by Dynamic Intra-Oral Processes

A posterior tooth may have a pointy cusp that occludes directly over the inter-proximal contact point between two teeth in the opposing arch, which may often push fermentable carbohydrates into the inter-proximal contact area.

A patient may bite into a hard, tiny food particle, like a blueberry seed, which may create a microscopic pit on an occlusal surface or cusp tip, where fermentable carbohydrates could stagnate.

Periodontal disease may cause loss of attachment, recession, or bone loss, which may uncover concavities, undercuts or overhangs in tooth root surfaces that were previously covered by gingival attachment or bone. These revealed undercuts, concavities, or overhangs may retain fermentable carbohydrates (Merijohn *et al*., 2016; Do *et al*., 2017; Pasquinelli, 2018).

Bruxism may transmit forces to the gingival aspect of a tooth, which may result in loss of attachment and recession, and expose concavities in tooth root surfaces that previously were covered by the attachment, which may then collect fermentable carbohydrates.

Bruxism may gradually wear away enamel at the incisal aspects of anterior teeth, causing the incisal surface of the worn anterior tooth to develop a central ovoid dentinal area bordered by enamel. This creates a tiny bowl shape on the incisal surface that may collect fermentable carbohydrates, facilitating demineralization episodes in the central dentinal areas.

Bruxism may transmit heavy forces to the buccal and

facial aspects of teeth at the gingival area, causing abfraction lesions that may collect fermentable carbohydrates, facilitating demineralization episodes (Fig. 3).

Occlusal stress may cause micro-fractures to occur at the palatal cervical areas of maxillary anterior teeth, resulting in retention of fermentable carbohydrates in the micro-fractures, facilitating demineralization episodes.

A posterior tooth may have a pointy cusp that protrudes beyond the imaginary occlusal plane, and which occludes into the opposing posterior tooth, such as to put strong point forces on the occlusal surface of the opposing tooth. These forces may mechanically damage the occlusal surface of the opposing tooth at the point of occlusion, creating a pit that collects fermentable carbohydrates, resulting in demineralization episodes.

If a maxillary molar has a pointy palatal cusp that extends below the maxillary occlusal plane, that pointy cusp may transmit large occlusal forces into the central fossa of the opposing mandibular molar. These forces may then transmit to the mesial and distal marginal ridges of the mandibular molar, causing microscopic marginal ridge craze lines or fractures (Mamoun and Napoletano, 2015). These fractures cause tiny crevices that collect fermentable carbohydrates, resulting in demineralization episodes that weaken the marginal ridges. Further weakening by caries accelerates the rate at which further mechanical force fractures these areas, facilitating inter-proximal caries.

The pulp chamber of an endodontically treated tooth may have been filled with excess amounts of flexible gutta-percha, instead of mostly hard core material, resulting in excess flexure of the remaining tooth structure. Eventually, micro-cracks may develop in the remaining tooth structure, which retain fermentable carbohydrates, leading to demineralization episodes.

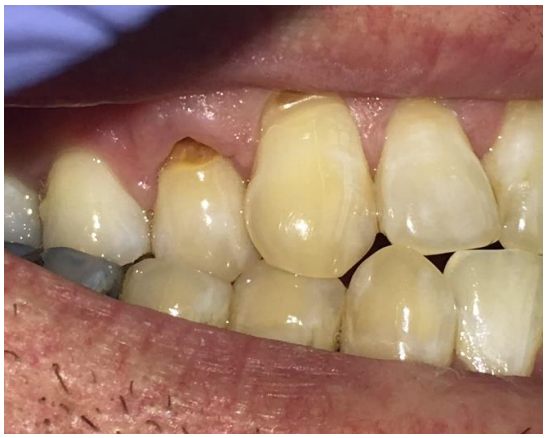

Fig. 3: A mandibular premolar is in cross-bite with a right maxillary first premolar in maximum inter-cuspation, causing cycles of bending forces on the maxillary premolar, which facilitated buccal abfraction lesion formation, which facilitated plaque and carbohydrate retention

Radiation treatment for head and neck cancer may damage the parotid gland and other salivary glands, resulting in chronic xerostomia. This may result in unusual caries at surfaces such as smooth buccal and lingual surfaces, cusp tips (Levi and Lalla, 2018; Brennan *et al.,* 2017; Palmier *et al*., 2017), or incisal line angles that normally would not become carious with normal salivary secretion rates. (Palmier *et al*., 2017; Ritter *et al.,* 2013; Boushell *et al.,* 2019; St Germain and Rusz, 1996; Croll and Swift, 2014).

Some medications, or drugs such as heroin or methamphetamine, may induce xerostomia, leading to multiple multi-surface carious lesions. Heroin or Methamphetamine users may develop behavioral changes such as developing poorer oral hygiene habits or consuming more sugary drinks, compared to non-users (Brown *et al*., 2012; Saini *et al*., 2005; Stanciu *et al.,* 2017; De Campaigno *et al.,* 2017; Shetty *et al.,* 2016).

Frequent consumption of acidic, sugary drinks, such as soda (Bibby *et al*., 1951; Nakayama and Mori, 2015; Almasi *et al*., 2016; Tahmassebi *et al.,* 2006; Armfield *et al.,* 2013) may demineralize tooth surfaces at a rate that is too high for saliva to remineralize the lesions, resulting in smooth surface decay. Normally, shallow smooth surface undercuts would not retain plaque or simple carbohydrates, but may do so if a patient combines poor oral hygiene with frequent consumption of acidic, sugary drinks or simple carbohydrates.

If a child often falls asleep while drinking from a baby bottle filled with a sugary drink like apple juice or with other fermentable carbohydrates, then over time this habit may result in severe decay of pediatric maxillary anterior teeth that may turn the teeth into carious root tips (Anil and Anand, 2017; Winter and Hamilton, 1966; Avila *et al*., 2015). Mandibular anterior teeth would generally be unharmed due to the protection of these teeth by saliva from sub-lingual salivary glands, or because the bottle directs the liquid flow towards the maxillary anterior teeth and away from the mandibular anterior teeth.

Plaque or Carbohydrate Retention Facilitated by Artificial Dental Prostheses or Restorations

A removable partial denture prosthesis may collect fermentable carbohydrates and cariogenic plaque on tooth surfaces that are undercut relative to various parts of the prosthesis, such as clasp areas (Preshaw *et al*., 2011).

Fermentable carbohydrates may collect in a space between a removable prosthesis guide plane and the tooth surface that neighbors the guide plane.

There may be a gap between a removable prosthesis's major framework and a tooth, where cariogenic plaque and fermentable carbohydrates may stagnate.

Recurrent decay (Mjör and Toffenetti, 2000) is a carious lesion where at least one surface of the carious

lesion is contiguous with a pre-existing restoration. A carious lesion may have formed before or after direct restoration placement, which initially was not contiguous with the restoration, but later expanded to become contiguous with the restoration.

A carious lesion may have been left in the tooth at the time the direct restoration was originally placed, and the carious lesion at that time was contiguous with the newly placed restoration, and eventually expanded to become a larger lesion. Caries left at external margins of newly placed restorations seem more likely to expand compared to caries completely sealed by the restoration (Schwendicke *et al*., 2016; Maltz *et al.,* 2012).

A dentist may have completely removed a carious lesion before placing a direct restoration, but the restoration had a structurally weak aspect that eventually fractured, creating a crevice within the restoration that collected cariogenic plaque and fermentable carbohydrates (Hodges and Mangum, 1995; Ferracane, 2017). An example of this is a Class II restoration, where the isthmus aspect of the restoration at the proximal box area was thin in cross-section and fractured from occlusal forces. Another example is where a mandibular anterior tooth with a pointy cusp would occlude into a weak aspect of a class III restoration of an opposing maxillary anterior tooth, eventually fracturing the restoration.

An overhang on a direct restoration may retain cariogenic plaque and fermentable carbohydrates under the overhang. Examples include caries underneath class III or class II restoration overhangs, or class V buccal or lingual overhangs.

A crown may have been placed on an abutment with minimal coronal tooth structure, resulting in excess forces being transmitted to the remaining coronal tooth structure that was retaining the indirect restoration. This may result in micro-fractures forming in that tooth structure, creating crevices that retain fermentable carbohydrates.

Decay may have been left in an abutment when the crown or fixed partial denture was placed, and the decay expanded underneath the fixed prosthesis. Decay sealed by the fixed prosthesis may stop developing, but decay at the margin of the crown may retain fermentable carbohydrates and be more likely to expand. (Memarpour *et al*., 2016) Decay left at inter-proximal surfaces facing pontics of fixed partial dentures may be difficult to clean.

A fixed prosthesis may have a marginal gap or a marginal overhang (Fig. 4) that retains fermentable carbohydrates (Ericson *et al*., 1990; Mounajjed *et al*., 2018; Yüksel and Zaimoğlu, 2011; Alomari *et al*., 2009). A fixed partial denture may collect fermentable carbohydrates at an open contact between the fixed prosthesis and a neighboring tooth (Nagarsekar *et al.,* 2016).

The abutments of a fixed partial denture may not originally have been shaped such as to provide a path of placement for the fixed partial denture such that there are no abutment undercuts relative to the path of placement (Nagarsekar *et al.,* 2016; Mamoun, 2012). This may result in a space between the intaglio surface of the fixed partial denture and the abutment surface that collects fermentable carbohydrates.

A cantilever fixed partial denture may put excess leverage forces on abutments that are distant from the cantilever abutment when occlusal forces are put on the cantilever abutment (Anderson, 2005). This may result in micro-fractures of abutment teeth at areas where cantilever leverage stresses are highest, which can retain cariogenic plaque and fermentable carbohydrates. Numerous episodes of micro-cracking and carious breakdown may accelerate the rate of lesion expansion (Fig. 5).

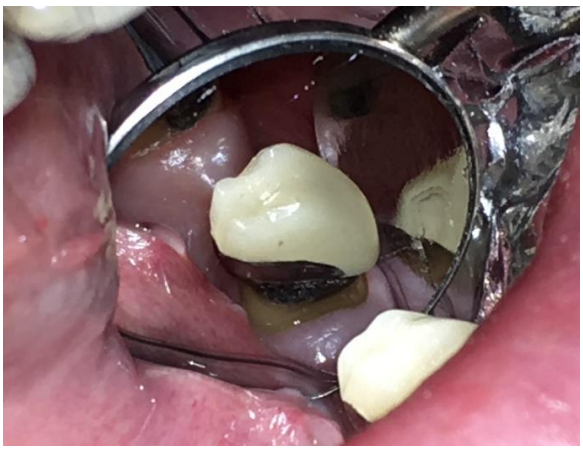

Fig. 4: A premolar crown with a marginal overhang that retains cariogenic plaque and fermentable carbohydrates. Bone loss, from periodontitis around the overhang, may have exposed root concavities, further facilitating plaque retention and caries

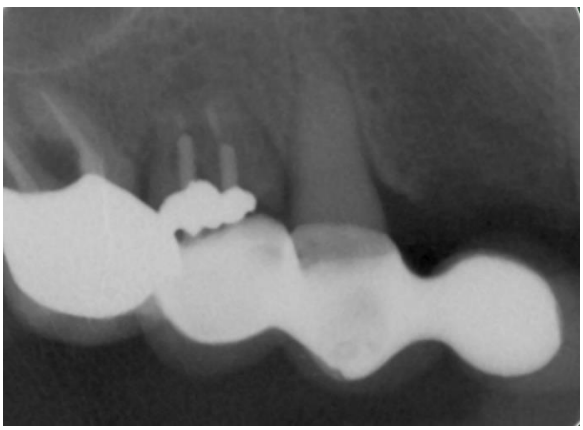

Fig. 5: In this radiograph of a cantilevered fixed partial denture, mesial marginal decay is observed on the right second premolar, at an area of high bending stress, since this area is distal from the cantilever fulcrum. Continuous micro-cracking stresses at the mesial surface of the abutment facilitated plaque and carbohydrate retention, and decay

An implant crown may have an open contact between the crown and the neighboring tooth (Varthis *et al*., 2016; Jeong and Chang, 2015). facilitating carbohydrate retention.

Orthodontic brackets or bands may trap cariogenic plaque or fermentable carbohydrates in contact with various tooth surfaces. (Arash *et al*., 2016; Kachuie and Khoroushi, 2017; Lipták *et al*., 2018).

Discussion

This proposed six-factor model of caries etiology aligns with current models by including the five basic factors known to cause carious lesions. This model deviates from current models by suggesting that a sixth factor, which facilitates simple carbohydrate retention or plaque retention at a specific tooth surface site, must be present alongside the other five factors before a carious lesion can occur at a specific tooth surface site. The inclusion of this sixth factor lacks quantitative validation and may be a product of selection bias. Future research to assess the validation of this model may involve mapping specific plaque and carbohydrate retentive factors, as described in this model, to a large sample of carious lesions.

Conclusion

This article proposes a caries etiology model suggesting that dental caries is caused by five basic known factors plus a sixth factor: A factor that facilitates simple carbohydrate retention or plaque retention at specific tooth surface sites. By identifying a specific intra-oral facilitator of simple carbohydrate or plaque retention at a specific tooth surface site, and understanding the five other demineralization-facilitating factors, a dentist may explain why any specific observed intra-oral carious lesion formed. Eliminating factors that facilitate plaque and carbohydrate retention at specific tooth surface sites, combined with standard preventive care, may improve lesion control and prevention.

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Ethics

The author states that this study complies with and respects scientific ethics.

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