Role of Fluoride in Dental Caries and Risk Management

Educational Objectives

Upon completion of reading these articles, the clinician will be able to:

1. Know the new concept of dental caries its etiology and risk factors
2. Know how Fluoride control dental caries and its mechanism of action
3. Know the effectiveness and limitations of fluoride in the control of dental caries
4. New concept how to improve dental caries prevention and risk control
Abstract

Dental caries is the single most common, chronic oral disease of childhood (Boyce et al., 2010). It is progressive and cumulative, and becomes more complex with time. Contemporary caries management philosophy has changed from the traditional surgical approach to a physician model that emphasizes prevention. Among various strategies for caries management, fluoride therapy has been highly promoted. Dental professionals need to identify and assess the caries risk level of patients and optimize the use of fluorides as part of dental caries management. Since multiple sources of fluoride exposure exist, a coordinated approach to fluoride delivery and safety is essential.

Introduction

Dental caries is a multifactorial oral condition with a complex etiology (Bowen, 2002; Cummins, 2006). It is a paradigm shift, to consider dental caries as a complex disease caused by an imbalance in physiological equilibrium between tooth mineral and biofilm physiology (Kidd and Fejerskov, 2004).

The interplay between the dental biofilm, constituents of the diet, and host tissues, as well as genetic and environmental factors, have each increasingly been recognized for their roles in the pathogenesis of dental caries. The etiology of dental caries was traditionally described in the Keyes diagram (Keyes, 1960) with its circles intercepting each other, which represent the host, agent, and environment as primary or essential factors (Figure 1).

![Diagram of the three critical factors in dental caries. Adapted from Keyes, Arch Oral Biol 1960;1:304-320.](image)
In 1978, Newbrum added a fourth factor, time, and it must be considered in any discussion of the etiology of dental caries. In other words, caries requires a susceptible host, a cariogenic oral flora, and a suitable substrate that must be present for a sufficient length of time (Figure 2).

![Diagram of the fourth circles that represent the factors involved in the carious process.](image)

Figure 2. Diagram of the fourth circles that represent the factors involved in the carious process. Taken from Newbrum E. Cariology, 1st edition Baltimore: Williams y Wilkins; 1978.

Adding a new factor increases the complexity of the disease, indicating that the start does not depend exclusively on the so-called primary etiologic factors. Therefore, the intervention of other etiological factors, known as modulators, decisively contributes and influences the onset and progression of the carious lesion.

More recently, modifications have been introduced (ten Cate, 2009), that include factors, such as life style and behavioral parameters. In the schematic illustration below (Figure 3), the complex interplay between saliva, dietary habits, and the many biological determinants determine biofilm composition and metabolism. At both, individual and population level, many of these variables (oral hygiene, diet, etc.) will be highly influenced by behavioral and socio-economic conditions prevailing.
Dental caries has changed its distribution in many populations (Beltran-Aguilar et al., 1999; Marthaler et al., 1996; Clenton-Jones, 2001; Whelton, 2004) and has gone from a disease that affects the majority to a disease with a displaced distribution at the individual level, in countries where oral health has improved, as well as in societies with different economic development levels. This means that a divergent pattern has developed; containing individuals with high caries susceptibility, frequently associated with low income level groups and less prosperous societies. In addition to the changes in caries patterns and a change in caries levels, there has also been a change in the relative contribution of pits and fissure and smooth surfaces to the overall caries experience (Whelton 2004), where the pit and fissures of the teeth are the sites that contribute in most cases to the caries experience (Acevedo et al., 2005; Acevedo et al., 2013).

**Caries risk factors and concepts to prevent and treat dental caries**

Risk is the probability that an event will occur within a period of time. The concept of assessing and managing risk in dental caries is a relatively recent concept which must be considered a necessary component in the clinical decision-making process. It is important for the clinicians to understand that caries risk factors, which are biological, environmental, or behavioral in origin, drive the changes in dental caries patterns at the population level and also they are of critical significance in the dynamic process of demineralization and remineralization of tooth structures.
Dental caries is a very complex process and many risk factors are associated with the onset of the disease. It has been widely accepted that the following risk factors are involved in a greater or lesser extent with dental caries disease: saliva (salivary flow rate, saliva buffer capacity, high saliva viscosity, low lysozyme, lactoferrin or lactoperoxidase levels, low arginine and urea levels, low fluoride, calcium and phosphate levels); dental plaque (increased intracellular and extracellular polysaccharide formation, acid-producing bacteria, imbalance between acid and base producing bacteria); tooth physical characteristics, (deep pit and fissures morphology, malformed teeth with retentive areas, enamel defects); dietary habits (frequent high sucrose food ingestion, chronic medication intake, adherent high sucrose food ingestion); previous caries experience, (family history of high caries rate, history of early childhood caries); systemic disease (xerostomia related autoimmune disease, syndrome); and socioeconomic factors (low income with limited access to dental care providers, young parents with low education level, behavior and attitudes toward health care). The interaction of many components in the oral cavity leads to whether a patient remains caries free or is at high risk of caries development (Hicks et al., 2003; Kidd, 1998; Reich et al., 1999; Anderson, 2002; Featherstone, 2006; Fontana and Zero, 2006). A summary of the high risk factors for caries development is presented in Table 1.

### Salivary Factors

<table>
<thead>
<tr>
<th></th>
<th>Low Risk</th>
<th>Intermediate Risk</th>
<th>High Risk</th>
</tr>
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<tbody>
<tr>
<td>Flow rate</td>
<td>&gt;1mL/minute</td>
<td>0.7 to 1 mL/minute</td>
<td>&lt;0.7mL/minute</td>
</tr>
<tr>
<td>Buffer Capacity</td>
<td>pH 5-7</td>
<td>pH 4-5</td>
<td>pH &lt;4</td>
</tr>
<tr>
<td>Mutans Streptococci</td>
<td>&lt;105 cfu/mL</td>
<td>105-106 cfu/mL</td>
<td>&gt;106 cfu/mL</td>
</tr>
<tr>
<td>Lactobacilli</td>
<td>&lt;104 cfu/mL</td>
<td>104-105 cfu/mL</td>
<td>&gt;105 cfu/mL</td>
</tr>
</tbody>
</table>

- High Acidity (low pH)
- Low Lysozyme, Lactoferrin or Lactoperoxidase Levels
- Low Arginine, Histidine–Rich Protein and Urea Levels
- High Viscosity
- Low Fluoride, Calcium and Phosphate Levels
- Low Immunoglobulin Levels
- Dental Plaque
- Acidogenic Bacteria (Mutans streptococci and Lactobacilli) with High Acid Production
- Increased Intracellular and Extracellular Polysaccharide Formation
- High Plaque and Gingival Indices

### Tooth Physical Characteristics

- Immature Enamel lacking Post–Eruption Maturation and Exposure to Fluoride
- Surface Enamel with Low Fluoride or High Carbonate, Magnesium and Organic Content
- Increased Acid Solubility
- Malocclusion with Plaque Retentive Areas
- Deep Pit and Fissure Morphology
- Malformed Teeth with Retentive Areas
- Enamel Defects (hypoplasia and/or hypo calcification
However, as each of these factors is beyond the scope of this paper, they will not be discussed further. Considering the current understanding of the caries disease process, the definition of dental caries risk factors, originally developed by Reich et al., (1999), has become controversial in the last decades. Risk factors such as: development of new caries lesion, the presence of active lesions, and the placement of restorations due to active disease have been considered as risk factors to dental caries development or progression, instead of factors that indicate the presence of the disease in a moderate to high caries risk population (Fontana and Zero, 2006; Featherstone et al., 2007).

The risk factors that are amenable to modification and can be addressed by the development of new and improved oral care products are the two key biological factors: susceptible host tissue and cariogenic bacteria in the dental biofilm; an inadequate fluoride exposure contributes to increased caries susceptibility of the host tissue. Therefore, it seems to be, that maintaining an adequate concentration of fluoride in the oral fluids might contribute to a minimization of the risk of developing dental caries lesions. Nonetheless, all factors should be managed as a whole, in

### Table 1. High Risk Factors for Caries Development
Taken from Hicks et al., 2004; J Clin Pediatr Dent 28(3): 203–214.

<table>
<thead>
<tr>
<th>Dietary Habits</th>
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<tbody>
<tr>
<td>Frequent High Sucrose Food Ingestion</td>
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<tr>
<td>Adherent High Sucrose Food Ingestion</td>
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<tr>
<td>Chronic Medication Intake with High Sucrose/Sweetener Content</td>
</tr>
<tr>
<td>Trace Elements in Diet: High Selenium and Low Fluoride, Strontium, Molybdenum, Aluminum, Lithium and Boron</td>
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<tr>
<th>Previous Caries Experience</th>
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<tr>
<td>High Caries Experience in Primary Dentition and/or Permanent</td>
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<tr>
<td>History of Early Childhood Caries</td>
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<tr>
<td>Smooth Surface Caries, Especially with Anterior Teeth</td>
</tr>
<tr>
<td>Family History of High Caries Rate</td>
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<tr>
<td>Systemic Disease</td>
</tr>
<tr>
<td>Xerostomia: Autoimmune Disease (Systemic Lupus Erythematosus, Sjogren’s Syndrome, Diabetes Mellitus, Head and Neck Radiation, HIV Infection)</td>
</tr>
<tr>
<td>High Carbohydrate Dietary Requirements (Cystic Fibrosis, Phenylketonuria)</td>
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<tr>
<td>Chronic Illness Requiring Medications with High Sucrose Content</td>
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<tr>
<td>Xerostomic–Inducing Medications</td>
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<th>Socioeconomic Factors</th>
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<tr>
<td>Low Income with Limited Access to Dental Care Provider</td>
</tr>
<tr>
<td>Young Parents with Low Education Level</td>
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<td>Behavior and Attitudes toward Health Care</td>
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order to control the onset and progression of dental caries lesions, since individual risk factors studied separately from the pool of risk factors tend to be poor predictors of dental caries onset.

Historically, application of caries research to prevention and treatment has been focused on the host tissue. Attempts to reduce caries risk have focused on reducing the tooth susceptibility to acid attack by rendering less vulnerable surfaces to the demineralization process (Arends and Christoffersen, 1982; ten Cate and Arends, 1977; Hoppenbrouwers et al., 1987). Likewise, enhancing the uptake of calcium and phosphate ions into demineralized tissue can be increased with fluoride exposure, which would favor some remineralization of existing caries lesions. However, the dental professional must emphasize and accomplish a balance between factors that induce or protect against the development of caries lesions (Figure 4), and be able to recommend specific preventive and treatment steps to reduce pathological factors and increase protective factors to help develop and maintain a prosper caries balance (Featherstone, 2006).

![Figure 4. Conceptual illustration of the caries balance concept. If the pathological factors outweigh the protective factors, caries progresses. Adapted from Featherstone, Pediatric Dentistry 2006;28:128-132](image)

**How Fluoride Control Caries**

The concept that fluoride controls caries lesion development, in the past, was considered to be essentially systemic and pre-eruptive in action, changing enamel structure, but by the end of the 1980s it has been well established that this effect was primarily a topical effect (ten Cate, 1990; Featherstone et al., 1990; Featherstone, 1999) interfering with the caries process. Hence, fluoride must be available in small concentrations (ppm) in the right place (biofilm fluid, saliva) and at the right time (sugar exposure) to interfere with demineralization and remineralization
events. Figures 5a and 5b clearly describe how fluoride affects these two processes (Cury and Tenuta 2008). Enamel is dissolve by the low pH reached in dental plaque, due to acid production every time sugar is ingested (Figure 5a). When the pH decreases to below 5.5, under saturation with calcium and phosphate with respect to hydroxyapatite (HA) is reached in the biofilm fluid, resulting in mineral dissolution (Figures 5a). However, if the pH is higher than 4.5, and in the presence of fluoride, the biofilm fluid is supersaturated with calcium and phosphate with respect to fluorapatite (FA) and at the same time that hydroxyapatite (HA) is dissolved, fluorapatite (FA) can be readily formed (ten Cate et al., 2003). As a consequence, the net result is a decrease in enamel dissolution, since a certain amount of calcium (Ca) and phosphate (P) that was lost from the dissolution of the HA is recovered by enamel as FA. This indirect effect of fluoride reducing enamel demineralization, when the pH drops, is complemented by fluoride effect on remineralization when the pH rises (Figure 5b).

On the other hand, after exposure to sugars has ceased, acids in the biofilm are cleared by saliva and converted to salts. As results, the pH increases, and at pH 5.5 or above, the biofilm fluid is supersaturated with respect to HA and FA (Figure 5b). Consequently, small amounts of Ca and P lost by enamel during the pH drop can be more efficiently recovered, if fluoride is still present in the oral environment after the cariogenic challenge (Cury and Tenuta, 2008).

This physicochemical effect of fluoride, reducing demineralization and enhancing remineralization of dental enamel could be supplemented by some antibacterial effect, if an appropriate concentration of fluoride can be maintained in the oral environment by the current methods of fluoride use.

It is important to highlight, that fluoride does not have a direct effect on the etiological factors responsible for dental caries by reducing tooth demineralization and enhancing remineralization. Consequently, if the current methods of fluoride use were able to interfere with the equilibrium between acid/base producing bacteria in the dental biofilm or its metabolism, when dietary sugars are consumed, the disease could be controlled more efficiently. Nonetheless, the
The objective is the maintenance of a constant low level of fluoride in the oral environment, which could be achieved by fluoride administration, either systemic or topical. (Cury and Tenuta 2008). Among these different methods of fluoride delivery (Ellwood et al., 2008), drinking water and dentifrices present unique properties to control the caries process, since the oral environment is exposed daily to low concentrations of fluoride.

What do we understand by remineralization?

Remineralization is the natural repair process of carious lesions, and even though it is a process that has been known for hundreds of years, it has only been in the last decades, when the importance of remineralization therapy has been accepted. Remineralization occurs when calcium and phosphates of salivary origin present in the fluid around enamel crystals (enamel fluid) is taken up by the crystals that had previously lost these ions. However, net remineralization produced by saliva is small and is a slow process, with a tendency for the mineral gain to be in the surface layer of the lesion due to the low ion concentration gradient from saliva into the lesion (Silverstone, 1972). More recently, Cochrane et al., (2010) defined remineralization as process whereby calcium and phosphate ions are supplied from an external source to the tooth to promote ion deposition into crystal voids in demineralized enamel to produce net mineral gain. The term void is used to define any accessible space in a crystal caused by ion loss from the demineralization process. This definition of remineralization therefore includes any crystal repair to bring about net mineral gain to an enamel subsurface lesion, but does not extend to precipitation of solid phases onto enamel surfaces.

Fluoride is the cornerstone of the non-invasive management of non-cavitated caries lesions, but its ability to promote net remineralization is limited by the availability of calcium and phosphate ions (Reynolds et al., 2008). Fluoride ions can drive the remineralization of non-cavitated caries lesions if adequate salivary or plaque calcium and phosphate ions are available when the fluoride is applied. It is known that the mineral formed during remineralization is more resistant to acids, when it is compared to the original enamel, especially if fluorides are present to join the surfaces of the new crystals. Fluorides, in a variety of presentations, such as dentifrices, varnishes, mouthrinses, gels and sealants had been proven to be relatively effective as remineralizing agents. In this review, scientific evidence for clinical effectiveness of the different fluoride presentations will be limited to dentifrice and varnish.

Clinical effectiveness of fluorides

In past decades a number of studies have reported a reduction in dental caries prevalence, possibly due to the preventive effects of fluorides (Hugoson et al., 2005; Divaris et al., 2012; ten Cate, 2004). Fluorides have their largest effects on reducing demineralization, promoting remineralization of non-cavitated lesions, and can affect other biological activities of cariogenic microorganisms.

It is very well documented in clinical trials assessed by conventional visual-tactile detection methods (Jenkins, 1985), that fluoride products such as toothpastes, mouthrinses, and dental office topical applications can reduce dental caries between 30 and 70% compared to no
fluoride controls (Jackson et al., 2005; Marinho, 2009). Fluoride in the drinking water and other systemic ways of administration has also been shown to be effective in reducing dental caries in large populations (Yengopal et al., 2010; Rugg-Gunn and Do, 2012). Nonetheless, it has become obvious that even where the caries challenge is very high; it is not possible for fluoride to overcome this challenge completely (Fejerskov, 2004).

Systemic and topical vehicles such as (i) water and salt fluoridation and (ii) fluoridated dentifrice are recognized as the most effective methods to control dental caries (Ellwood et al., 2008; Yengopal et al., 2010). Fluoridated water had a relevant role in caries control until the 1980s-90s, when, in some countries, it was the only source of fluoride at the community level, and it is still considered important in many countries. Since the 1990s, however, the widespread use of fluoridated dentifrice has had a tremendous effect on caries decline at the population level (Rölla et al., 1991). The epidemiological changes in caries promoted by fluoride from drinking water or dentifrices have been observed both in developed (Brunelle and Carlos, 1990; Rölla et al., 1991; Rugg-Gunn and Do, 2012) and developing countries. Brazil is an example of a developing country in which the anticaries benefits of these two sources of fluoride delivery are clearly evident. Currently, 45% of the Brazilian population has access to water fluoridation, mostly in south and southeast regions, and all dentifrices are fluoridated (90% containing MFP) (Cury et al., 2004).

Fluoridated dentifrices

The concentration of fluoride in saliva increases every time teeth are brushed with a fluoride containing dentifrice. After 3 minutes, the fluoride concentration in saliva is more than 100 times higher than the baseline value, but after 2 hrs, it returns almost to baseline (Bruun et al., 1984; Duckworth and Morgan, 1991). However, during tooth brushing, fluoride is spread throughout the oral cavity and is stored in some compartments (Ekstrand and Oliveby, 1999), such as the enamel surface and any remaining dental biofilm. In this way, after tooth brushing, salivary clearance dilutes the residual fluoride in saliva, but the enamel surface and remaining biofilm are able to take up fluoride, as calcium-fluoride (CaF2)-like deposits, maintaining certain fluoride levels in the right place to control caries. These reservoirs can release fluoride to the fluid of the biofilm during pH-cycling due to sugar exposure (Rölla et al., 1991; ten Cate, 1997), thereby reducing enamel demineralization and enhancing its remineralization. If this were true, fluoride should be found in dental biofilm either soon after tooth brushing or preferably for longer periods. In an in situ study, 10 hrs after tooth brushing, the fluoride concentration in dental biofilms of a group of subjects using fluoride dentifrice was 30 times higher than that found in the control group (Paes Leme et al., 2004) (Table 2).
Table 2. Means ±SD (n=15) of Fluoride Concentration in Dental Biofilms and Enamel Demineralization (ΔZa), According to the Treatments

<table>
<thead>
<tr>
<th>Treatments</th>
<th>mg F/g Biofilm Wet Weight</th>
<th>Intermediate Risk</th>
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<tbody>
<tr>
<td>Non-F dentifrice (control)</td>
<td>1.5 ± 0.5</td>
<td>1,253.6 ± 697.2</td>
</tr>
<tr>
<td>APFb + non-F dentifrice</td>
<td>7.1 ± 12.0</td>
<td>971.4 ± 671.5</td>
</tr>
<tr>
<td>F dentifrice</td>
<td>46.6 ± 46.6</td>
<td>405.4 ± 216.4</td>
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a Area of mineral loss as measured by cross-sectional microhardness determination (Paes Leme et al., 2004).
b APF = acidulated phosphate fluoride. Taken from Cury and Tenuta, 2008

Even 14 days after APF fluoride application, which forms a high quantity of CaF2-like deposits, a high concentration of fluoride was found in the biofilm. Thus, the effect on reduction of enamel demineralization observed can be attributed to this residual fluoride maintained in the biofilm by these modes of fluoride use (Cury and Tenuta, 2008).

Although literature suggest that the incidence of coronal caries among adults is as high as that in children, about 1 new carious coronal tooth surface per year (Garcia, 1989; Griffin et al., 2005), with the exception of water fluoridation, virtually all primary preventive programs using dentifrices target children and youth (Association of State and Territorial Dental Directors, 2002). One possible reason for the lack of preventive programs for adults may be the lack of evidence on their effectiveness for this population. Curnow et al., (2002) designed an investigation in school children to determine the reduction in 2-year caries increment that can be achieved by daily supervised tooth brushing on school days with a toothpaste containing 1,000 ppm fluoride (as sodium monofluorophosphate) and 0.13% calcium glycerophosphate, combined with recommended daily home use, compared to a control group involving no intervention other than 6-monthly clinical examinations. The result at the end of the two year indicates significantly less caries developed in first permanent molars in the intervention group at both D1 and D3 levels. The mean DFS net caries increments (reversals included) and frequency distributions are presented by group in Table 3. Supervision of tooth brushing on schooldays and encouragement for regular use at home of this fluoridated toothpaste has led to a significant reduction in carious first permanent molar surfaces over a 2-year period in this high-caries-risk group of children.
Table 3. Twenty-four month DFS caries increments of first permanent molars: mean values and frequency distributions

Data from substantial clinical trials have suggested that for every 500-ppm fluoride increase in the toothpaste, a reduction of 6–8% in caries prevalence can be expected (Stephen et al., 1988; O’Mullane et al., 1997; Wong et al., 2011). This occurs in particular within the range in concentration from 1,000 to 2,500 ppm fluoride [Pessan et al., 2011]. Recently, Nordström and Birkhed (2010) carried out an investigation in order to evaluate the preventive effect on caries incidence and progression of a dentifrice containing 5,000 ppm F, compared with a standard dentifrice containing 1,450 ppm F (both as NaF), on caries-active adolescents. The design was a 2-year, single-blind randomized controlled trial and 211 adolescents of 279 (76%) completed the trial. The subjects were divided into two groups and were given one of the assigned F dentifrices for daily unsupervised toothbrushing: (1) Duraphat 5,000 ppm F and (2) Pepsodent Superfluor 1,450 ppm F, both as NaF. The outcome variables were caries incidence and progression of proximal and occlusal caries. The subjects were asked to fill in a questionnaire to evaluate their compliance and they were divided into two subgroups: subgroup A, excellent compliance, and subgroup B, poor compliance. The latter group (28%) comprised the subjects who did not brush twice a day or did not use the dentifrice regularly. Adolescents using 5,000 ppm F toothpaste had significantly lower progression of caries (A:p< 0.01, B:p<0.001). Subjects using 5,000 ppm F toothpaste had significantly lower caries incidence for compliance B compared to those using 1,450 ppm F toothpaste (p < 0.05). The same pattern was observed for the pooled group (A+B) (Figure 6). This may indicate that 5,000 ppm F toothpaste has a greater impact on individuals who do not use toothpaste regularly or do not brush twice a day. Thus, 5,000 ppm F toothpaste appears to be an important vehicle for caries prevention and treatment of adolescents with a high caries risk.
Another important issue to be discussed is the use of dentifrices with a high fluoride concentration is the prevalence of root caries in elderly people. Studies from Denmark indicate that: (1) the proportion of elderly people in the total population will increase dramatically during the next decades; (2) elderly people maintain more of their teeth than previously; (3) the dentition in those who still have their own teeth are heavily restored; (4) gingival recession is frequent in elderly people, and (5) many fragile elderly people use medicine and the standard of home-based oral hygiene is far from optimal (Petersen et al., 2004; Holm-Pedersen et al., 2005; Petersen and Yamamoto, 2005; Ekstrand et al., 2008). Similar oral health problems have been described in other industrial countries (Chalmers et al., 2002; Österberg et al., 2006). The effectiveness of tooth brushing with dentifrices containing 5000ppmF versus 1,450 ppm for controlling root caries in nursing home residents was evaluated by Ekstrand et al., 2013. The results of this 8-months randomized clinical trial allow the authors to conclude that 5,000 ppm F-toothpaste is significantly more effective for controlling root caries lesion progression and promoting remineralization compared to 1,450 ppm F-toothpaste. However, it is important to emphasize that the daily use of high fluoride concentration, such as dentifrices with 5000ppmF, must be done under professional prescription and supervision.
Fluoride Varnish

Another important vehicle to deliver fluoride to the tooth and oral fluid, are the fluoride varnish, which had been extensively proved to be effective controlling dental caries in high risk population. Several clinical studies on children and adolescent have demonstrated the clinical effectiveness of fluoride varnish. Weintraub et al., (2006) determined the efficacy of fluoride varnish (5% NaF, Duraphat®, Colgate) added to caregiver counseling to prevent early childhood caries. A two-year randomized, dental-examiner masked clinical trial was conducted. Initially, 376 caries-free children, from low-income Chinese or Hispanic San Francisco families, were enrolled (mean age ± standard deviation, 1.8±0.6yrs). All families received counseling, and children were randomized to the following groups: no fluoride varnish, fluoride varnish once/year, or fluoride varnish twice/year. An unexpected protocol deviation resulted in some children receiving less active fluoride varnish than assigned. Analyses of the results showed a significant fluoride varnish protective effect in caries incidence (p<0.01). Caries incidence was higher for counseling only vs. counseling + fluoride varnish assigned once/year (OR = 2.20, 95% CI 1.19-4.08) and twice/year (OR = 3.77, 95% CI 1.88-7.58), indicating that fluoride varnish added to caregiver counseling is efficacious in reducing early childhood caries incidence.

A recent systematic review reported by Marinho et al., (2013) dealt with the effect of fluoride varnish on caries prevention and caries risk management in children and adolescents. Later, after reviewing twenty-two trials with 12,455 participants, the pooled D(M)FS prevented fraction estimate comparing fluoride varnish with placebo or no treatment was 43 to 57% (P< 0.0001). In relation to the pooled d(e/m)fs prevented fraction estimate was 37% to 51% (P< 0.0001) for the 10 trials that contributed data for the primary tooth surfaces meta-analysis. No significant association between estimates of D(M)FS or d(e/m)fs prevented fractions and caries severity, background exposure to fluorides, application features such as prior prophylaxis, concentration of fluoride and frequency of application were found. The analyses allow the authors to suggest a substantial caries-inhibiting effect of fluoride varnish in both permanent and primary dentition.

Improvement in Dental Caries Prevention and Risk control

Theoretically, caries risk can be controlled with an appropriate diet and oral hygiene, which includes the frequent removal of dental plaque and an appropriate exposure to fluoride from different sources. Even if fluorides can counteract the mineral loss in dental tissue produced by a highly cariogenic diet, a limit exists to its efficacy (Ripa, 1991; Sales-Peres and Bastos, 2002). This statement was confirmed by Duggal et. al., (2001) in an in situ study, where the subjects were asked to consume a sugar in solution either once, 3, 5, 7 or 10 times daily and brush their teeth with a toothpaste containing 1450 ppm of fluoride. The study concluded that the subjects showed an evident demineralization only with the 7 and 10 times a day regimes. These results were subsequently confirmed by Ccahuana-Vásquez et al., (2007), who observed changes in the matrix of the biofilm after frequent rinses with sucrose. Also, these changes could not be prevented with the presence of fluorides. This permits us to confirm that even with the use of fluorides, dental caries can still develop due to frequent intake of sugar. Therefore, it is important to state that, even if caries lesion is controlled by fluorides, the disease has not been eliminated (DePaola et. al., 1993; Cummins, 2013).
While fluoride products have dramatically reduced dental caries, the fact that caries remains a prevalent oral health and public health problem calls for new strategies to supplement existing measures to reduce caries risk and improve dental health in individuals and in populations on a global basis (Pitts and Stamm, 2004; NIHCDP, 2001).

It is important to highlight, that topical fluoride products do not target dental plaque, which is arguably the primary modifiable pathological factor in dental caries. Specifically, they do not reduce pathological factors either by reducing dental plaque levels, by promoting microbial homeostasis in sites with dental plaque, by preserving the dynamic balance in favor of non-pathogenic organisms, or by preventing environmental perturbations that lead to an overgrowth of acid-producing bacteria. Because of their primary mode of action, topical fluoride products help to control, but they cannot completely prevent, dental caries (Fejerskov, 2004).

Any new anti-caries strategy should recognize and complement the effects of fluoride. As fluoride’s benefits are focused on the host tissue as a means of damage control after the caries process has been initiated and is in progress, combining fluoride with an agent that targets plaque pathogenicity and prevents the caries process would have potential to deliver a step-change improvement in caries prevention (Cummins, 2013).

**Conclusion**

This review provides an overview of the most current concept of dental caries including its etiology, risk factors and concept to prevent and treat dental caries. In addition, the role of fluoride in caries risk management and its efficacy in reducing and preventing caries is emphasized, proving its successes in the control of dental caries in high caries risk populations.

Therefore, with the enormous scientific evidence on the effectiveness of fluoride as anti-caries agent, clinicians must emphasize on its use, in order to promote remineralization and reversion of the existing carious lesion at the earliest stage possible, but also, target dental plaque, which is arguably the primary modifiable pathological factor in dental caries.
References


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Figure 1. Diagram of the three critical factors in dental caries.

Figure 2. Diagram of the fourth circles that represent the factors involved in the carious process.

Figure 3. Modified Keyes Diagram. Determining factors in the development of dental caries. The need for antibacterial approaches to improve caries control.

Figure 4. Conceptual illustration of the caries balance concept. If the pathological factors outweigh the protective factors, caries progresses.

Figure 5a. Enamel demineralization in the presence of fluoride in dental biofilm.

Figure 5b. Enamel remineralization in the presence of fluoride in the dental biofilm.

Figure 6. Total caries incidence and progression using 5.000 ppm dentifrice in adolescents.

Table 1. High Risk Factors for Caries Development

Table 2. Means ±SD (n=15) of Fluoride Concentration in Dental Biofilms and Enamel Demineralization (ΔZa), According to the Treatments

Table 3. Twenty-four month DFS caries increments of first permanent molars: mean values and frequency distributions
Author Bio

Dr. Ana María Acevedo received her DDS from the Facultad de Odontología, Universidad Central de Venezuela in 1972. In 1978, received an MSc in Biochemistry at the Instituto de Investigaciones Científicas (IVIC), Venezuela; in 1982, received the PhD at the Faculty of Science, University of Bristol, England and finally in 1992 did post-doctoral training at the School of Dental Medicine, Department of Oral Biology and Pathology, State University of New York at Stony Brook, NY, USA.

Teaching: Professor at the Instituto de Investigaciones Odontológicas, Facultad de Odontología, Universidad Central de Venezuela, Caracas, Venezuela; Visiting Professor Facultad de Odontología, Universidad de Carabobo, Valencia, Venezuela; Visiting Professor at the Department of Oral Biology and Pathology, School of Dental Medicine, State University of NY at Stony Brook NY, USA.

Research: My research skills are in Cariology and Salivary Research and in a team with Dr. Israel Kleinberg and Robi Chatterjee, we developed a formulation constitute by Arginine Bicarbonate/Calcium Carbonate highly effective in the control of Hypersensitivity and Dental Caries. Over 60 papers published in national e international journals and more than 300 abstracts presented in national and international meetings.

Author Contact Information

Ana María Acevedo DDS, MSc, PhD

e-mail: aacevedo1947@yahoo.com

Phone: USA:1-631-5654493 (Mobil)
Venezuela: 58-414-3388437 (Mobil)

Office USA: 1-631-6328949
Office Venezuela: 58-212-6053796