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Fluorides and Other Preventive Strategies for Tooth Decay

Jeremy A. Horst, DDS, PhD[1], **Jason M. Tanzer, DMD, PhD, DHC**[2], and **Peter M. Milgrom, DDS**[3]

[1]Postdoctoral Fellow, Department of Biochemistry and Biophysics, University of California San Francisco, San Francisco, CA USA

[2]Professor Emeritus, Department of Oral and Maxillofacial Diagnostic Sciences, University of Connecticut Health, Farmington, CT USA

[3]Professor, Department of Oral Health Sciences, University of Washington, Seattle, WA USA

Synopsis

We focus on scalable public health interventions that prevent and delay development of caries and enhance resistance to dental caries lesions. These interventions should occur throughout the life cycle, and need to be age-appropriate. Mitigating disease transmission and enhancing resistance are achieved through use of various fluorides, sugar substitutes, mechanical barriers such as pit-and-fissure sealants, and antimicrobials. A key aspect is counseling and other behavioral interventions that are designed to promote use of disease transmission-inhibiting and tooth resistance-enhancing agents. Advocacy for public water fluoridation and sugar taxes is an appropriate dental public health activity.

Keywords

Fluorides; topical; Public Health Dentistry; Dental Caries; Silver Diamine Fluoride; Pit and Fissure Sealants

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This chapter focuses on strategies to reduce the burden of dental caries across the population, using fluorides and some other dental caries preventive agents. It is imperative to be purposeful about the goals of employing the various interventions, and particularly that agents should be targeted by patterns of disease susceptibility, which are associated with

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Corresponding Author, Peter Milgrom, 1916 15th Ave E, Seattle, WA 98112 USA, dfrc@uw.edu.
Contact information
[1]Department of Biochemistry, University of California San Francisco, 1700 4th Street, QB3 Room 404, San Francisco, CA 94158 USA, jahorst@gmail.com
[2]Section on Oral Medicine, Department of Oral and Maxillofacial Diagnostic Sciences, University of Connecticut, 263 Farmington Avenue, Farmington, CT 06030 USA, Tanzer@uchc.edu
[3]Department of Oral Health Sciences, Box 357475, University of Washington, Seattle, WA 98195-7475 USA, dfrc@uw.edu

disclosure statement

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age. Dental caries in its various forms—early childhood caries (ECC), severe-early childhood caries (S-ECC), primary dental caries of the deciduous and permanent dentition, recurrent caries, and root surface caries—are diseases in which the products of sugar metabolism by certain bacteria that populate the tooth surface induce the development and progression of lesions.

These lesions (so called “cavities”) are the clinical expression of disease, in which dental plaque bacteria metabolize sugar into polymeric substances that stabilize their adherence to the tooth and into acids that demineralize the hard tissues of the tooth. The term caries lesion includes the spectrum of lost tooth structure ranging from “white spot” enamel demineralizations, through large cavitations that extend into dentin. The bacterial species involved in the disease process are substantially known, but vary among depths and sites of caries lesions. There is little evidence that any interventions currently in use by dentists reduce the incidence of dental caries as a *disease*. The most effective interventions now known decrease the incidence of new lesions and curtail lesion growth, and these will be a major subject of this chapter. Dentists, it should be noted, currently spend most of their time dealing with previously treated caries lesions, referred to as recurrent or secondary caries lesions. Population-focused prevention efforts seek to alter the dental plaque biofilm, by reducing dietary sugar exposure, and improving the resilience of the teeth.

In general, primary prevention attempts to address etiology, whereas secondary prevention aims to stop progress of disease. Confusion arises from failure to distinguish the difference between tooth-level (lesion) *versus* individual- and population-level (disease) prevention. We do not have adequate, facile means to detect caries activity before lesions have appeared; the apparent breakdown of tooth structures is a result of a disease process that started earlier. The presence of visible lesions is the best available diagnostic for disease and predictor of future disease, so this is what we use. Meanwhile, *cure* of caries is just as elusive as for most cancers or coronary heart disease; what we presently do is count the years since the last sign of disease, such as the appearance of a new lesion or growth of an existing lesion. Thus, once a person has had any caries lesions it is unclear whether intervention could target primary prevention of disease. The aim in this case is to reduce the impact of the disease -- secondary prevention.

This paper focuses primarily on interventions that enhance resistance to disease progress. Enhancing resistance is achieved through use of various fluorides, sugar substitutes, and mechanical barriers such as pit-and-fissure sealants. Relatively new to the discussion of primary and secondary prevention is the use of antimicrobials. Other key aspects of caries control are behavioral interventions (e.g., motivational interviewing) with patients and their caretakers (parents, guardians, grandparents, etc.) to promote use of disease transmission-reducing and resistance-enhancing agents. Behavioral intervention is necessary, as the interventions do have to be used in order to work.

A key means of risk reduction for primary prevention of dental caries on the population level is through decrease of frequency and duration of exposure to dietary sugar. Such public health efforts — through present and potential government policies and industry food guidelines to improve overall nutrition—need to be part of dental public health practice. The

enormous increases in sugars consumption over the past 40 years, and concomitant increase of human metabolic diseases (diabetes, obesity, heart disease, and stroke) demonstrate that people/families generally are not able to control sugar intake on their own, and thus system-wide public health changes are needed. However, efforts of the sugar industry during the 1960s and 1970s resulted in a shift away from research and progress in this field;[1] but more recently, successful reductions in sugar consumption have been achieved by raising taxes, as in Mexico.[2]

Secondary prevention of caries requires early diagnosis and prompt treatment to reduce lesions' complications (pain, abscess, systemic infection, etc.) and occurrence of new lesions. Secondary prevention encompasses the concept of caries lesion *arrest*, because lesions that continue to grow can cause pain, tooth loss, and may serve as a reservoir of cariogenic bacteria that can initiate new lesions; antimicrobials are logical interventions. Lesions that grow also lead to escalating personal and public expense to replace parts of the dentition or, eventually, all of the dentition. The cascade of disfigurement of the dentition can impact social acceptance, growth patterns, and quality of life.[3] School-based screenings have been an important and widespread approach to early detection in secondary prevention, but have generally not led to either early diagnosis or prompt treatment, primarily because referrals are largely ineffective.[4] All school programs would be more effective if they employed additional secondary prevention strategies to non-surgically arrest lesions (discussed later in this chapter).

Timing of prevention efforts

Children

Timing for primary prevention of ECC (caries lesions in the primary dentition), to prevent transmission of cariogenic bacteria to children, should be focused on mitigation and prevention of colonization of the dental biofilm (plaque) by cariogenic bacteria, especially *Streptococcus mutans*, which occurs within a couple years after tooth eruption. The child's mother or other caregivers, through transmission of salivary bacteria, is the usual source of *S. mutans* that colonize young children.[5,6] When new mothers have low salivary mutans levels, their babies' colonization by these bacteria is greatly delayed as is the age and severity of caries lesions in those children; whereas, when mothers have high salivary mutans levels most of their babies are colonized younger and lesions appear within a couple years thereafter.[5]

There is also good evidence that habitual maternal use of xylitol chewing gum during the first years of life of the infant protects the child from *S. mutans* colonization, and the children get 71–78% fewer caries lesions (Table 1).[6–8] Thus, the first intervention should start with the caregivers, before the child has teeth.

Typically, cavities begin to appear early in the third year of life. In communities with very high disease burden, cavities appear within the first year after tooth eruption. Primary prevention aimed at increasing tooth resistance must begin before this, when children are unlikely to see a dentist. *S. mutans* and other cariogenic bacteria are unable to stably colonize the mouth until the teeth erupt, although they have been detected in the mouths of

predentate children.[9] Thus, intensive prevention efforts for children in high risk communities should start with female care deliverers before the time when teeth of their children erupt and continue after the teeth have come into the mouth, generally late in the first year of life. Scarce resources for dental public health are being deployed during preschool (e.g. Head Start, 3–5 years old), often with the mistaken notion that this is primary prevention; the disease has already manifested by this age.

With each newly exposed tooth surface that enters the mouth, the opportunity presents for colonization by cariogenic bacteria. In permanent teeth, lesions typically follow 2–4 years after eruption (Figure 1).[10] Often the rationale for justification of efforts focused on the prevention and treatment of caries in primary teeth is the overstated connection of caries in primary teeth to that in permanent teeth; the contribution is very small with relative risk ratios such as 2.6[11] and 1.4.[12] Thankfully, children are in school at this age and easier to reach through school-based delivery systems. While intensive interventions to get high risk children into dental clinics have raised annual dental clinic visits from, *viz* ~12% to 43%,[13] bringing dental care to schools is a more viable option for secondary prevention of decay of permanent teeth. Non-restorative and minimally invasive options are logical treatments for primary teeth and early erupting permanent teeth. The grand opportunity afforded by exfoliation is to slow lesions in the primary dentition until the teeth shed, while preventing lesions in the permanent dentition. The importance of this goal cannot be overstated.

Adults

The majority of dental treatment in adults is the consequence of failure of fillings placed earlier in life; most fillings are replacements, due to “recurrent caries” at the margins of or under old restorations. Dentists attribute the failures to the filling materials, but significant evidence to the contrary is now in the literature. [14,15] Excising lesions with a dental drill neither stops the initiation of new lesions, nor eliminates caries risk factors that led to the failure. Regardless, preventing recurrence is not primary prevention at either the disease or tooth level. On the other hand, altering patient resistance to coronal and root caries is primary prevention of lesions when the effort is focused especially on those at highest risk.

A major increase in the focus of public health efforts in adults should be on those who are transitioning into higher caries risk status, for example, when the quality and quantity saliva decreases (xerostomia) due to polypharmacy, radiation exposure of the salivary glands, methamphetamine abuse, Sjögren’s disease, etc. As well, root exposure following over-brushing and iatrogenic root surface damage attendant to mechanical instrumentation with the intent to control gingivitis and periodontal disease, and restoration of caries lesions that inadvertently damages the gingival attachment to the teeth and leads to root exposure, increase the number of at risk surfaces.

Efforts at the population level for adults are uncommon. Perhaps, to be effective, preventive interventions should be tied to other care encounters (periodontal care, primary medical care, and therapy for long term conditions such as substance abuse, heart disease, etc.) so that the seminal risk-increasing events (drug abuse, chemotherapy, onset of systemic disease, multiple prescriptions) are addressed before damage is seen. Senior centers (>60 years) and subsidized public housing for elders (e.g. HUD housing), assisted living and skilled nursing

facilities for older adults might be the focus of these efforts. The risk and the need for primary prevention are not static but change across the life course.

Prevention/arrest--approaches during early childhood

Caries does not occur without sugar. Rather, the evidence is overwhelming that the frequency of sugar consumption and the duration of sugar in the mouth are more powerful determinants of caries risk than is the quantity of its consumption.[16,17] Providing dietary guidance in dental public health programs at the earliest ages is imperative. Additionally, avoidance of sugar-enriched beverages such as juice drinks, sodas, and sports drinks at all ages is important, and the fallback strategy of rinsing with water after consumption of these artificial drinks are consumed may be useful, and should be studied further. Milk and baby formula should never be supplemented with sugar.

Patients with severe plaque due to a complete lack of oral hygiene, but fed solely through gastric tubes or intravenous ports, do not get caries lesions. Likewise, patients with the genetic defects of intestinal sucrase deficiency or hereditary fructose intolerance (fructose is half of the sucrose molecule), who therefore avoid dietary sucrose do not develop appreciable caries lesions and have barely detectable *S. mutans* in their mouths.[18,19] Increased frequency of simple sugar intake seems to have the largest effect on initiation of lesions. “Baby bottle tooth decay” resulting from cow milk or artificial “formula” is an important example: restriction of milk bottle exposure to 3–6 meal times depending on weight and age reduces the incidence of caries dramatically, while exposure throughout the night is to be strongly discouraged.[20]

It takes time for cariogenic dental plaque to accumulate to the point when it can deliver enough acid onto the tooth surface to dissolve enamel. Cavities do not occur in constantly cleaned teeth. Frequently disturbing plaque by any means works to prevent caries lesions.[21] Caregivers need to be taught how to clean the teeth while maintaining reasonable comfort for all involved. It is helpful to build a sense of control in the child by breaking up each episode of brushing into small bits with structured time (counting), even during infancy. Teeth can be cleaned anywhere. Sinks and bathrooms are not needed and it is frequently easier to clean a young child’s teeth on the floor or a sofa with the child’s head on one’s lap or between one’s legs (Figure 2).

Fluoride varnish decreases the amount of new caries lesions in school-aged children by 37%.[22] This effect was assumed to extrapolate to younger children. We ourselves had this hope, and documented safety of fluoride varnish in infants.[23] A surge in fluoride varnish use starting with the eruption of the first tooth has come in the last decade, but positive results have not followed. Table 2 details the outcomes of clinical trials on caries lesion prevention by fluoride varnish when starting the intervention before the 3rd birthday. Disappointingly, 5 of the 6 studies employing fluoride varnish alone show no prevention of new lesions.[24–30] The three studies that combine fluoride varnish with other interventions also showed no effect, or only the expected effect of the other interventions.[31,32]

One interpretation of this surprising disparity in the effectiveness of caries prevention by fluoride varnish at different ages is that differing balances of pathogenic and protective factors may occur at different ages. Perhaps dietary sugars, hygiene, and the composition of the dental plaque play a larger role than the enhancement of remineralization potential by fluoride varnish. It may also be that diverse varnishes vary in their effectiveness; no clinical testing has been done on the preventive effect of most fluoride varnishes currently on the market.[33] The lack of effect observed in these recent trials deserves further study. For now, we recommend use of interventions that consistently show an effect.

Promising work has been done on the combination of antimicrobial agents with fluoride varnish. Two clinical studies in toddlers (12–35 months old) show added benefit of painting povidone iodine onto the teeth immediately prior to fluoride varnish, every 2–4 months for at least 10 months. As summarized in Table 3, a clinical trial resulted in 80% fewer children having any signs of caries after 1 year of bi-monthly combined iodine-fluoride treatment, as compared to fluoride varnish only.[34] A cluster intervention showed that inclusion of povidone iodine resulted in 24% fewer children having any signs of disease after 10 months with ~2.5 treatments per child, and 31% less new lesions total.[35] Prevention of caries by antimicrobials is understudied in infants and toddlers.

Fluoride supplement tablets show 24% prevention of caries lesions in permanent teeth, but such an effect has not proven to be consistent for primary teeth, and the effect is no greater than topical fluoride rinses, varnish, or toothpaste, which presumably pose less risk of fluorosis.[36] If this is to be done, it is wise—especially if the children drink well water—that parents determine the level of fluoride in that water.

Community fluoridation

Following the discovery of the preventive effects of fluoride in water against dental caries, fluoride was added to water, milk, and salt. The scalability (amenability to general implementation) of this intervention arises from centralized production and existing government regulation of these vehicles. Water fluoridation is the most widely adopted, reaching more than 370 million people in 27 countries, with many studies demonstrating effectiveness and safety, with the sole exception of dental fluorosis as a possible side effect. The cost is roughly 20–50 cents per person per year in the U.S.[37] A recent Cochrane meta-analysis included 107 studies with an estimated average of 35% prevented fraction of caries lesions in the primary dentition (dmft), 26% prevention of lesions in permanent teeth (DMFT), and 15% prevention of any new lesions (primary disease prevention). The authors caution, however, that 72 of the studies were conducted prior to the widespread use of fluoride toothpaste, and that benefit from the combination is uncertain. Nevertheless, prevention by fluoride toothpaste is independent of fluoridated water exposure so one might expect a combined benefit. Twelve percent of recipients have esthetic concerns about dental fluorosis attributable to water fluoridation.[38]

Salt fluoridation reaches about 60 million people in Europe and more than 100 million in Latin America including Mexico. The cost is one-tenth of water fluoridation making it by far the least expensive and probably the most efficient method of caries prevention. While no

modern clinical trials on the caries preventive effects of salt fluoridation are known to us, older cluster-randomized studies consistently show 50% prevention of new lesions.[39]

Milk fluoridation offers the highest precision of fluoride dose, as the variation in quantity of milk intake is lower among children than that for tap water or salt, and can be further metered by single serving boxes. The cost is U.S. $1–2.50 per person per year, roughly 5 times that of water fluoridation. Accordingly, only about one million children receive fluoridated milk.[40] Despite many demonstration projects, a recent Cochrane review found only one placebo-controlled clinical trial of milk fluoridation, in which a 31% prevented fraction was observed in the primary dentition; the lesion increment in the permanent dentition of the control group was too low to make any conclusions.[41]

The reported target of these scalable fluoridation interventions generally was children. However, impact across the age spectrum can be achieved. In countries that predominantly consume processed food (such as the United States, Mexico, and Canada) and have high prevalence of dental caries, physicians recommend limiting salt intake due to exacerbation of hypertension associated with cardiovascular disease. Gestational hypertension bears the same concerns. Thus, while the cost effectiveness is provocative, the propriety of salt fluoridation for older adults or pregnant women should be approached with caution and further study. Most countries curtail milk consumption during later childhood. Community water fluoridation, therefore, appears to be the large scalable intervention of choice to prevent caries during adulthood.

The importance of fluoride toothpaste

The sale of fluoride toothpaste has been profoundly successful. In the U.S., an average of three tubes of toothpaste are sold for every person annually. Across 70 clinical trials, 24% of caries lesions are prevented by using fluoride toothpaste compared to non-fluoride toothpaste, and this effect is not decreased by exposure to fluoridated water. Meta-analysis by concentration of fluoride shows a dose response that seems to reach maximum effect at 37% prevented lesions for the highest tested concentrations: 2400–2800 ppm F. Disease-level prevention (no new lesions) is seen in 12% of patients using over-the-counter (OTC) strength fluoride toothpastes, 1000–1500 ppm in primary or permanent teeth, compared to controls.[42] Significant heterogeneity in disease-level outcomes is seen. No placebo-controlled trials have been conducted in 30 years, while the free fluoride concentrations in these toothpastes have increased. One trial, conducted prior to the inception of adding fluoride into toothpaste, observed a 25% decrease in caries lesions with calcium phosphate toothpaste versus no toothpaste. Brushing without a dentifrice or with a dentifrice without fluoride has long been assumed to have an important effect on lesion prevention, but numerous trials have shown a lack of prevention of new lesions, even in supervised studies. Unquestionably, brushing *per se* inhibits gingivitis and that is a major reason dental providers stress its importance.

Children’s toothpastes (850 to 1150 ppm fluoride) for those under 6 years (when mineralization of all tooth crowns except the 2nd permanent molars has been completed), and pastes with as much as 5000 ppm F for older children or adults, are effective self-

administered topical drugs for primary prevention of tooth decay. The efficacy of these self-applied fluorides has been particularly well-documented in trials in the young permanent dentition in a wide range of populations (see above). As mentioned above, the prevalence of dental caries lesions drops 20–30% in populations using fluoridated toothpaste. Supervised tooth brushing in schools is effective. Rinsing is not encouraged generally after normal brushing, as exemplified by the Oral Health Foundation's "spit don't rinse" slogan.

Free postal delivery of toothpaste along with advice from home visitors not only reduces decay at the 24% rate seen in trials with direct administration, but reduces need for dental extractions in lower socioeconomic groups.[43] Therefore, distribution of toothpaste ultimately reduces pain, disfigurement, and further, more extensive and costly complications of caries – important goals. While successfully implemented at other ages, toothpaste use has not been effective as a public health measure in preventing ECC. Despite the fact that the American Dental Association recommends that appropriate amounts of fluoride toothpaste be used for high risk children of all ages,[44] many healthcare workers continue to discourage early adoption of fluoridated toothpaste to reduce the risk of fluorosis. However, retrospective study of an Australian population exposed to fluoride toothpaste early in life shows a relatively small increase in fluorosis, and at the same time demonstrates extensive protection against tooth decay. The highest meta-analysis estimate of increased risk of fluorosis by toothpaste is two-fold.[45] Most fluorosis is of no clinical significance. Furthermore, fluorosis is not related to quality of life measures.[46] If fluorosis concerns persist, roughly one third less fluorosis occurs when delaying use until after the first birthday.[44,45]

Parents need guidance from primary care providers about how to choose a fluoride-containing toothpaste. They are confused by the labeling and advertising. They also need instruction on how to brush a child's teeth. Often parents think they need to brush the teeth in the bathroom awkwardly trying to do this with the child sitting on the sink. They can't see the teeth or keep the mouth open. Many parents and caregivers think that children can brush their own teeth, even when they are very young. They need to be taught that a parent should model good brushing, and also brush the child's teeth themselves until the surprisingly old age when children can effectively brush (circa 7 years). Some parents believe that the teeth may be damaged by brushing, or that 3 year olds can brush properly: parents need accurate information.

Fluoride rinses, foams, varnish, and high fluoride toothpastes and gels

Clearly, fluoride can prevent caries lesions in school-aged children and older adults in any of the available delivery systems – 26% by lesion, and 12% by disease.[47] Industry and academic efforts to optimize benefits have focused on minimizing application frequency and protocol duration, while maximizing the prevented fraction of caries lesions. Massive development efforts have gone into the various delivery approaches: rinses, varnishes, foams, gels, etc. Varnish seems to be the endpoint of single-agent fluoride-only materials, as the protocol demands only seconds, varnish can be favorably flavored, and twice per year application appears to maximize effect. Still, the greater use of foams may be due to their more favorable textures. While daily fluoride exposure may contribute to control of dental

plaque bacteria, it must be remembered that professionally applied fluorides operate mostly by increasing the remineralization of the enamel surface. It is not surprising that these single agent therapies top out at ~37% prevention. In view of the small differences in effects between topical fluorides, it is not surprising that adoption is so low even 20 years after introduction of fluoride varnish into the U.S. According to industry experts, it is estimated that only half of dental offices use fluoride varnish (personal communication, Kevin Thomas of Elevate Oral Care).

Fluoride rinses result in 27% fewer lesions, or 23% fewer permanent teeth with lesions.[48] Rinsing with fluoride *instead* of brushing with it achieves similar outcomes.[49] Rinsing with fluoride may be particularly useful for prevention in teenagers or others who are old enough to rinse but have trouble with motivation or dexterity to brush, as rinsing is simpler. Only one study of fluoride varnish explicitly in addition to fluoride toothpaste has been done, though most studies of varnish to prevent lesions in children occurred in the background of fluoride toothpaste; their effects were equivalent. No significant differences are seen among rinses, gels, varnishes, and toothpastes in the few available studies,[49] though these studies appear too small to have been able to detect a difference if one had existed. Not accounting for the recent studies in 1–3 year old children summarized in Table 2 above, the prevented fraction of lesions estimated for each topical fluoride is as follows: daily OTC toothpaste 24%, daily prescription (5000 ppm) toothpaste 37%, daily rinse 26%, semi-annual gel 21%, and semi-annual varnish 37%.[42,47]

Considering cost effectiveness and additive benefit, the best approach to using fluoride for primary prevention as the risk increases appears to be normal strength fluoride toothpaste until the completion of permanent tooth crown formation (circa 8 years), then 5000 ppm fluoride toothpaste, each together with fluoride varnish two times per year.

Stannous fluoride in toothpaste has been understudied, perhaps because of concerns about tooth surface staining and taste. The color change of the dentition’s surface is probably due to oxidized porphyrins from dead bacteria and oxidized tin. There is some evidence of effectiveness but none of the studies are modern placebo-controlled randomized clinical trials. Blinding of examiners and participants is problematic. Meanwhile, the potential activity of the tin ion against dental caries in available OTC products should be further evaluated.

Silver diamine fluoride as a treatment and preventive for caries

Silver diamine fluoride is a topical treatment for caries lesions and a primary preventive for newly exposed high risk surfaces such as first molar fissures or roots.[50] Its mechanism(s) of action is under investigation. However, silver diamine fluoride has double the concentration of fluoride (~5%) as that in varnish, is 25% weight/volume silver ions, and has 8% ammonia, in water.[51] It is currently presumed that the high fluoride content allows for more effective diffusion into enamel and dentin, that the silver kills bacteria upon contact, and differentially stays in de-or hypo-mineralized tooth structures, both hardening the structures and reactivating upon exposure to bacterial metabolic byproducts thereby preventing their reinvasion, and that the ammonia stabilizes the solution and serves as an antiseptic to add to

microbial kill on contact.[52–54] This hypothesized triple mechanism seems well-suited for caries since this material treats the disease etiology.

Clearance by the US Food and Drug Administration (FDA) in 2014 and availability of the product in 2015 have catalyzed adoption. No dental product has had such rapid adoption. We estimate that 10% of US dentists now have a supply. Canada approved the same product in early 2017. The FDA recently designated silver diamine fluoride with Breakthrough Status, which is a commitment to a drug application for a life-threatening disease for which there is no medical treatment.

Nine clinical trials document caries arrest by treatment of cavitated dentin lesions with silver diamine fluoride in children and older adults. Twice per year application apparently maximizes the arrest effect that increases to 90% after 2 years of treatment.[55,56] Maintenance of arrest seems to depend on at least annual re-application.[57,58] Furthermore, after 2.5–3 years, 70% fewer lesions are observed on the untreated surfaces of patients whose lesions were treated with silver diamine fluoride.[55,59] This observation of fewer new lesions from treating only existing lesions appears to surpass that for operative treatment, which is approximately 38% after 2 years.[15] Prevention of new lesions is also documented, where application to high risk surfaces once per year is equivalent or more effective than fluoride varnish four times per year in children or older adults.[60,61]

The only known side effect of silver diamine fluoride is the staining of lesions. The silver tarnishes to black. This color change is an index of the effectiveness of the treatment, where the entire lesion turning black indicates success: all lesions that are completely black are apparently arrested. Some lesions that are arrested do not turn entirely black, but this is fairly obvious from the shiny dentin; all demineralized (carious) or hypomineralized dentin or enamel will stain black. Parents and caregivers generally do not object to the stains in primary teeth when the treatment is explained and the alternative is operative treatment.[62] The carious dentin is hardened by the treatment to twice normal dentin hardness.[63]

Application is simple (dry and apply), such that any dental or medical provider can provide the treatment. Nurses and hygienists who can provide care at remote sites such as schools or nursing homes should be encouraged to adopt silver diamine fluoride to manage dental caries lesions. Monitoring is simple. The cost of the material is commensurate with fluoride varnish.

Sealants for primary prevention

Sealants form mechanical barriers that isolate the pits, grooves, and defects in the biting (occlusal) surfaces of the teeth from the dental plaque and dietary constituents. They also can fill defects in smooth surfaces. Sealants were developed in part because water fluoridation was not as effective a decay preventive on occlusal pit and fissure surfaces as on the smooth surfaces of the teeth (buccal, lingual, and proximal), and also to treat early lesions.[64]

Sealants continue to be used in public health even as evidence—both histological and clinical— has mounted that topical fluorides are effective and less expensive. Placement of

plastic resin sealants is a time-intensive and technique-sensitive procedure requiring skilled personnel and significant patient tolerance, e.g. compared to application of topical fluorides. Effective resin sealant application in children requires four hands. Studies have shown no statistically significant difference in the preventive effects of resin sealants versus fluoride varnish. [65,66] Sealants in public health, placed on the basis of poverty status alone, are expensive, and may be an inefficient use of scarce resources. Nonetheless, direct cost effectiveness comparison is important, for example to account for the consolidation of efforts into fewer visits by sealants than for topical fluorides. A recent study comparing the two again showed no significant differences in the prevention of pit and fissure lesions by 6 monthly applications of fluoride varnish or sealants in first permanent molars, but a careful analysis showed cost savings of US$88.53 (£68.13) per patient using the varnish.[67]. Also, resin sealants do not alter the risk for caries lesions on untreated surfaces, while fluoride varnish is easily applied to other surfaces.

While public health outreach efforts to deliver sealants for first permanent molars are successful, almost no similar efforts have been made to place sealants in second permanent molars. This lack of coverage of older children and adolescents is important because caries lesions that develop during adolescence often go untreated and can result in expensive visits to the emergency department of hospitals a decade later.[68]

Resin sealants are considered the standard of care for prevention of lesions on the treated surfaces, or as treatment for non-cavitated lesions.[69] They should only be placed in children or adolescents who have clearly documented past caries experience or large amounts of plaque on their teeth. The presence of fillings is not always a good indicator of past caries lesion experience as diagnosis of caries lesions among dentists is highly variable. The best indicator of dental caries is frank cavitation. Also, sealants may be indicated if a child has a medical condition that directly or indirectly impacts salivary flow, or where medications contain sugar, as in syrups. Although the focus is on first and second permanent molars, defects or deep fissures in primary molars in caries active children merit the use of sealants.

Significant evidence suggests that the replacement of resin sealants by high viscosity glass ionomer cements should be considered. Glass ionomer cement sealants release fluoride and metal ions into the parts of the tooth most susceptible to caries, and do not require a dry field to be created in the moist environment of the mouth. When the bulk of the cement is lost, some material remains in the deepest parts of the grooves to provide mechanical protection. When lesions are used as an endpoint (instead of retention), resin and glass ionomer cement sealants show equivalence in meta-analyses at 2, 3, 4, and 5 years after placement.[70] There is markedly greater ease of application: the tooth is brushed, then the material is mixed and pushed into the pits and grooves of all teeth. This increases the speed and therefore presumably the cost-effectiveness. There is also evidence of superior prevention on untreated surfaces by glass ionomer cements. For example, a study of 2,557 children in Italy demonstrated 35% prevention of caries lesions on the distal of the second primary molars when using glass ionomer cement compared to resin.[71] Since resin sealants will not be successful unless placed in a very dry field, glass ionomer cement sealants may offer a better alternative generally, and particularly if the molar tooth is erupting, the child is unable to

cooperate, or if the operator is working alone. Patient preference and cost-effectiveness should be studied further.

Resin sealants, when placed well, have a relatively high retention rate. However, the goal is not to retain a material; it is to prevent caries lesions. There is misunderstanding that the Oral Health 2020 goals state that sealants should be retained in certain percentages of children; the actual goals state that certain percentages of children should have their teeth sealed. The monitoring of sealant retention is a surrogate measure specific to resin sealants. The effectiveness of glass ionomer cement sealants does not rely on retention. Meanwhile, there is no innate therapeutic value to resin, such that leaking resin margins create a micro environment that promotes tooth decay. Public health sealant programs have mistakenly been set up to monitor the surrogate marker of sealant presence rather than actual lesions as the response variable. These systems need to adapt to new data, and implement interventions that lead to the best clinical outcomes.

Xylitol and other polyols

Sugar substitutes have long been sought and some studied extensively: xylitol, a 5 carbon sugar alcohol has proved to have unexpected antibacterial effects specific for *S. mutans* by compromising its metabolism and colonization.[72] Studies began with a remarkable series of mostly human clinical trials. In the Turku Sugar Studies, Scheinin and Mäkinen substituted the non-fermentable polyol xylitol for virtually the entire sugar content of the dietary components of dental students and faculty at a dental school in Finland during a 3 year period.[73] This substitution resulted in remarkable caries incidence reduction, by comparison with similarly fructose-substituted and conventional sucrose-containing foods. Essentially no new lesions were seen in the xylitol group, while 7.2 were gained in the sucrose group.[74] It is, however, impractical for humans to make such complete dietary substitutions due to gastrointestinal intolerance to >50g per day of xylitol, being a characteristic of all dietary additives/substitutes poorly absorbed from the gut.

Subsequently, controlled studies demonstrated that several exposures daily to high content xylitol-containing chewing gums or other confections with a high content of xylitol, thus greatly reducing the ingested load of xylitol (the remainder of the diet remaining essentially unaltered), also significantly inhibited caries prevalence and incidence. Notably, the greatest reduction of lesions occurred on the smooth surfaces of the teeth; the fissures and pits were least affected. The most remarkable of these longitudinal studies was carried out for 40 months with 10 year olds in Belize, a society with high sucrose consumption and high caries prevalence. Several studies have shown the biological bases of this effect to be essentially specific to the *S. mutans* among the oral flora.[72] Some have argued that this anti-caries effect of high content xylitol gums is merely a reflection of sweet taste and gum chewing that increase salivary flow and salivary buffers, with resultant clearance of food from the mouth and neutralization of acids in the plaque.[75] Nonetheless, the well-controlled Belize study that included gums of high content sorbitol alone and in combination with xylitol demonstrated a xylitol dose-response efficacy, and discounts this simple salivary flow and buffering explanation.[76] Additionally, large population studies of fluoride-containing toothpastes containing either 10% sorbitol or 10% xylitol also show augmentation of decay

preventive effects of the fluoride when containing 10% xylitol.[77,78] A recent smaller study did not show any benefit.[79] Mainstream toothpaste manufacturers in the US do not presently make their pastes with xylitol. A recent study of xylitol lozenges in adults with low caries lesion backgrounds showed no effect on coronal lesions, but reduction in root lesions.[80]

Other non-fermentable or slowly fermented (by dental plaque bacteria) sugar substitutes have been studied beside xylitol. Of long interest have been sorbitol and mannitol (6-carbon sugar alcohols, widely used in sugar free confections in the U.S.) and inducibly transported and catabolized in part to ethanol;[81] they are associated with modest caries reduction consistent with partial substitution of sugar confections in the diet.[82] Erythritol (a 4-carbon sugar alcohol analog of xylitol), when added in high concentration to a glucose-containing culture medium slows the growth of mutans streptococci, as does xylitol,[83] but unlike xylitol, is not associated with reduction of total streptococci in interdental plaque.[84] A 2-year cluster-randomized trial in children at low risk for lesion development (average 1.5 new lesions after 2 years), drinking fluoridated water, observed no difference between lesion scores for lozenges that contained either xylitol-maltitol (4.7g, 4.6g daily total, respectively) or erythritol-maltitol (4.5g, 4.2g daily total) versus participants not given lozenges.[85] Maltosyl-erythritol, a triose alcohol, has been reported to inhibit extracellular glucan synthesis from sucrose by *S. sobrinus*, one of the mutans streptococci prevalent on human teeth, but we know of no clinical studies with it.[86] The other clinical trial of erythritol (7.5g daily total) confections known to us showed prevention of caries lesions in children with respect to sorbitol or xylitol candies, though comparisons were performed on total lesions in each group rather than the standard ΔDMFS, which deflates variance metrics and is therefore questionable.[87]

Also notable as a sugar substitute is isomaltitol (Palatinit, Isomalt) which is now prominent in sugar free confections in the U.S. and many countries abroad. It is a 1:1 mixture of 2 synthetic disaccharide alcohols: glucosyl-sorbitol and glucosyl-mannitol. It is slowly fermentable,[88] claimed not to lower the pH in human dental plaque, unlike sugars, and is reportedly non-cariogenic in rats.[89] However, extensive literature search reveals no clinical studies to date that indicate it either reduces *S. mutans* colonization levels or reduces tooth decay incidence in humans.

Perhaps the most surprising and important public health data from studies of xylitol gum chewing come from the study of mothers who chewed xylitol gum daily postpartum, and whose initially three month old infants experienced delayed colonization by mutans streptococci, and dramatically lower levels of carious lesions at 5 and 7 years of age, even though the mothers were instructed to stop chewing xylitol gum when their children were 15 months old.[8] Confirmation of this pattern is seen in two other studies, as shown in Table 1. These data are illustrative of primary prevention of caries by prevention of mother/child transmission of the prime pathogen of caries. Thus, it can reasonably be concluded that xylitol is of interest in the realms of primary and secondary prevention of tooth decay.

Antibiotic and Antiseptic agents

Antibiotics and Immunization

Although chronic antibiotic use, as formerly common when rheumatic fever was prevalent in the US, was noted to sharply decrease caries prevalence among penicillin users,[90] it is universally deemed inappropriate to use antibiotics to inhibit caries because of allergic sensitization of the host and because of the risk of spread of antibiotic resistance among bacteria. Considerable progress has been made in efforts to develop immunization against the mutans streptococci, a group-specific or species-specific approach to caries control, leaving the rest of the oral flora relatively unaffected. To date, almost all work has been done in non-human experimental animals and *in vitro*. A proposal for human trials of vaccine in the United Kingdom has been turned down by its FDA-analogous agency with its feeling that all vaccines carry some, albeit generally rare, risk of significant adverse effects on the host, ex. Guillain-Barré syndrome, and that there already exist good non-immunological means to inhibit caries – sugar restriction, fluorides, sealants, etc. One must view the prospect of immunization against caries as distant. Nonetheless, at least one patent for an immunological has been sold to a drug company recently.

Antiseptic agents

Chlorhexidine and combinations with chlorhexidine

Several non-specific, albeit potent antiseptic agents have been of interest in secondary prevention of caries affecting crown (enamel) and root (dentin) surfaces. The most studied has been chlorhexidine, which kills most bacteria rapidly by disruption of their cell walls. This antiseptic is available in different forms and concentrations in various countries. It is importantly benign to the host's mucous membranes and skin. A 0.2% mouthrinse was of initial interest in Scandinavia for its inhibition of supragingival dental plaque and associated gingivitis, for which it was effective even in the absence of tooth brushing and flossing. A non-randomized study of caries inhibition using 1% chlorhexidine gel resulted in 56% fewer lesions after 3 years.[91] These studies, among others, led to attempts by a U.S. company to introduce an over-the-counter chlorhexidine mouthrinse, but the concentration of chlorhexidine that cleared the FDA was reduced to 0.12% and the product was required to be marketed by prescription only.

It was known by this time that chlorhexidine rinsing would reduce short term salivary mutans streptococcal titers by 1000-fold, but that those titers rebound to baseline in about 3 months. This provided part of the rationale for a study of whether operative dentistry to surgically remove recurrent carious lesions after prolonged rinsing with 0.12% chlorhexidine could provide better outcomes if patients were to then chew either xylitol gum, sorbitol gum, or no gum for a period of three months. It was observed that xylitol gum greatly delayed the rebound of mutans streptococci in saliva, while sorbitol gum chewing had no effect; typical rebound occurred, as it did in the no gum control group.[92] This dramatized the prospective utility of xylitol gum for secondary caries prevention, and argued against the idea that its effects were attributable simply to saliva flow stimulation, as discussed above.

Attempts have been made to deliver chlorhexidine in gels and varnishes to the teeth, with inconsistent demonstrations of efficacy. Fourteen clinical trials on prevention of caries with chlorhexidine products have shown markedly different results. While 3 trials using 1% chlorhexidine + 1% thymol, or 10% chlorhexidine show an effect to prevent root caries, no effects were seen in meta-analysis of the other 5 clinical trials in older adults,[93] and the eight in children.[94] Patients in the 3 trials showing an effect did not have access to routine prophylactic dental cleanings. The lack of effect seen for 40% chlorhexidine varnish was particularly concerning because a dose-response is expected for effective interventions. However, little or no evidence was reported that the chlorhexidine was released from the diverse matrices in these studies. The chlorhexidine may have been bound in the varnishes and never released; bioavailability needs to be tested before clinical trials. A large carefully executed multi-centered trial in the U.S., in which chlorhexidine release from varnish was verified, demonstrated a lack of caries prevention for 10% chlorhexidine varnish in adults.[95]

On the other hand, several chlorhexidine-containing vehicles have been shown in randomized trials to have an effect on caries lesions.[96] In a notable study, 1% chlorhexidine gel was applied to the teeth of teenagers using applicator trays for 1 min/day for 14 days, whenever their paraffin-stimulated salivary *S. mutans* levels were higher than $>2.5 \times 10^5$ cfu/mL, at baseline, and every 4th months thereafter. The strategy effected a short-term 2 to 3 log reduction of salivary titers, with long term 1 log reductions only in those starting with titers $>10^6$ cfu/mL. Pit and fissure sealants were also placed at baseline in the intervention arm. All participants rinsed with 0.2 % NaF every two weeks during the 3 school years of the study. 56% fewer lesions were seen across all intervention group patients, and 81% fewer among participants with $>10^6$ cfu/mL *S. mutans* at the start of the study. Thus, treating the risk factor of cariogenic bacteria salivary titers with an antimicrobial, illustrated efficacy.[91] Additionally, two placebo controlled trials showed significant prevention of interproximal caries lesions by flossing 1% chlorhexidine gel between the teeth. One study of 12 year olds applied the chlorhexidine by floss 4 times/yr for 3 years, resulting in 42% fewer new lesions and 68% fewer new fillings in the interproximal surfaces, compared to placebo quinine flavored floss and a no flossing control. The placebo floss group scores were not substantially different from the no flossing scores. No differences in *S. mutans* salivary titers were noted.[91] An analogous study in 4 year olds showed 43% fewer new lesions and 58% fewer new fillings in the interproximal surfaces.[97]

Iodine

Iodine-based disinfectants kill *S. mutans*, albeit not selectively. They have long been accepted as skin and mucosal disinfectants, and appear to be extremely safe. Three clinical trials describe 1-time use in children in operative dental treatment under general anesthesia. This additional intervention lowers *S. mutans* titers for ~6 months, but does not have an effect on clinical outcomes, as predicted by rebound of mutans levels.[98,99] However, repeated use of povidone iodine before fluoride varnish decreases incidence of caries lesions; as described in Table 3. This is one of the only interventions that has been shown to work in toddlers that also works in school-aged children.[100]

Arginine

Safety and buffering of dental plaque due to putative ionization of ammonium has led to interest in arginine somewhat recently. Various clinical trials ranging in size from 200 to 6000 participants on the use of toothpaste with 1.5% arginine, calcium carbonate, and fluoride all find nearly the same 20% reduction in caries lesions after 2 years compared to fluoride toothpaste.[101–103] Similar studies on reversal or arrest of root caries lesions show some effect.[104,105] The effect is purported to be a metabolic shift away from acid production, and change of the microbial profile towards health.[106] But arginine has a strongly cationic guanidino- functional group like the two guanidino- groups of chlorhexidine, and thus may actually function as an antiseptic. Eight percent arginine-containing toothpastes are marketed by Colgate specifically to reduce dentin hypersensitivity in the U.S., while toothpastes containing 1.5% arginine, calcium carbonate, and fluoride are available elsewhere. The manufacturer indicates that the product is safe for children.

Other agents

Fluoride, silver, xylitol, chlorhexidine with thymol, povidone iodine, and arginine all appear to be effective agents and, to some degree, function in an antibacterial manner. Also, myriad papers describe kill of *S. mutans* with extracts from natural products, such as high molecular weight cranberry extracts and numerous botanicals from Asia. Vitamin D also deserves more attention: 24 clinical studies on caries lesion prevention by vitamin D supplementation were conducted between the two World Wars, of which meta-analysis estimates 53% prevention.[107] The belief at the time of these studies was that vitamin D enhanced the quality of saliva. Work in Canada demonstrated a relationship between both maternal and children's blood vitamin D levels and caries experience.[108,109] This, however, was not observed in the U.S. nation-wide NHANES data.[110] While vitamin D is widely added to foods such as milk, it has additionally come into common practice in the U.S. to promote vitamin D supplementation during pregnancy and infancy, which may thus produce benefits; effects should be monitored.

Salivary stimulants

Currently no clinical studies known to us have evaluated the effects of medications that stimulate salivary production against dental caries. While saliva provides a natural defense against caries disease, and the most dramatic severity of disease occurs when saliva flow is severely decreased (xerostomia), no clinical trials have been performed to evaluate the possibility of protective effects against caries by muscarinic agents in patients with xerostomia. Xerostomia is a common unintended side effect of many drugs with anticholinergic effects, or of radiation therapy, and Sjogren's disease. Of course, these studies would have to weigh the incidence of caries lesions, as manifested in the long term, versus the acute manifestations of the cholinergic (muscarinic) agent -- desired salivation, lacrimation, perspiration, intestinal cramping, and defecation, and potentially bradycardia that are characteristic responses to muscarinic drugs.

Sealants for caries arrest

In the pediatric chapter of *Pathology of the Hard Tissues of the Teeth*, Black instructs "Leave the decayed material in the dentin where it is" when describing interproximal disking and use of silver nitrate to treat caries.[111] Many infections resolve with use of antibiotics, which do not kill nor remove every causative microbe, rather they tip the balance in favor of the host response and, when properly selected, to preferential survival of benign indigenous bacteria. Similarly, treating caries lesions by sealing them to remove access to host dietary nutrients or further insult, tips the balance in favor of the host response. This is not new. Even the cautious ADA Council on Scientific Affairs states: "sealants can prevent the progression of early non-cavitated carious lesions."[69] The goodness of the seal is the important factor. The abilities of the odontoblasts to specifically sense and secrete antimicrobial peptides that kill cariogenic bacteria, and to keep inflammation away from the inner pulp, seem to have been overlooked.[112] As well, reactionary dentin (tertiary) forms under slowed or arrested lesions, serving to distance the pulp from active microbes. Thus, most caries lesions should simply be sealed rather than excavated.

In the Hall technique, discussed below, caries lesions are sealed with a preformed stainless steel or acrylic strip crown and glass ionomer luting cement; this is the single-most effective caries lesion treatment in primary teeth besides extraction.[113] The Journal of the American Dental Association has published studies for decades that demonstrate long term clinical and microbiological success from sealing in caries lesions that progress well into the dentin.[114]

Bacteria die when cut off from nutrients. A recent systematic review in the J Dent Res reports less pulp exposures and symptoms with incomplete excavation: "incomplete lesion removal seems advantageous compared with complete excavation, especially in proximity to the pulp."[115] Leaving some bacteria in a tooth with no signs or symptoms of pulpal pathology is becoming the standard of care; this is done when sealing in initial lesions as recommended by the ADA, while some bacteria are nearly always found in sound or affected dentin. Meanwhile, therapeutic sealants can be combined with chemotherapeutic interventions such as silver diamine fluoride or silver nitrate provide.[116] Sealing caries lesions simply requires a good seal, which in turn demands either circumferential contact with healthy enamel, or complete coverage as with the Hall technique.

Atraumatic restorative treatment

Atraumatic restorative treatment (ART) is the simple operative procedure of partial lesion removal focusing on developing clean margins, followed by placement of high viscosity glass ionomer cement. Neither advanced equipment nor electricity is needed. Treatment of single surface lesions is highly useful, while success in multiple surface lesions has higher failure rates.[117] The conceptual novelty with respect to traditional operative dentistry has motivated various U.S. professional organizations to attempt to rename ART "scoop and fill glass ionomers" or "interim therapeutic restorations". However, the inventors of the technique and the World Health Organization already named this and recommended its use worldwide, 20 years ago.

Hall technique crowns for arrest

The Hall technique for placing stainless steel crowns is shockingly easy and effective. An appropriately sized preformed crown is selected and cemented using glass ionomer cement, with neither excavation of lesion nor other mechanical or chemical preparation. The presumed therapeutic mechanism is multiple: seal the tooth from inflow of extrinsic nutrients in attempt to deprive cariogenic bacteria, and strengthen the caries-weakened dentin with fluoride and metal ions from the cement.

Evidence supporting the Hall technique includes a 5-year split-mouth randomized controlled trial in 132 children, which not only evaluated minor (need for retreatment) and major (infection) failures, but also personal factors such as treatment preference. This study even followed 73% of teeth to exfoliation.[113] Another similar randomized trial followed 148 children. Concerns raised about the possibility of low quality restorations in the control group in the first clinical trial were addressed in the second trial, in which all restorations were performed or supervised by pediatric dentists.[118]

Behavioral interventions

Current guidance from the American Academy of Pediatric Dentistry (AAPD)[119] and others states that toothbrushing of all dentate children should be performed twice daily with a fluoridated toothpaste and parents should use a ‘smear’ of toothpaste to brush the teeth of a child less than 2 years of age. Despite the recommendation, there is no evidence for the effectiveness of dentist anticipatory guidance or counseling. Studies of this topic, though imperfect,[120] are beginning to appear in the literature.[121] While fluoride toothpaste is effective in the primary dentition (see above), the results question whether the age 1 dental visit is an efficacious means of informing and guiding parent and child behavior.

Trials of traditional advice-based counseling have been neither promising nor rigorous.[122] A search of trials on the effect of dietary interventions, for example, alone or in combination with other behavioral interventions on dental caries of children identified 13 trials. Self-reported increases in fruit and vegetable consumption was reported, but no changes in sugar consumption nor hygiene were achieved. Limitations of the studies included not having an intense phase as well as a maintenance phase of the intervention to maintain change, short follow-up, and not including caries as outcome. Moreover, the trial designs not only underestimate the need to impart specific parenting skills required to improve self-efficacy of caregivers in child’s oral hygiene or child’s sugar sweetened beverage intake, but also lack an environmental component designed to limit access to cariogenic foods. A better approach to controlling the etiologic sugar consumption, is governmental policy. Studies of Mexico’s tax on sugar-sweetened beverages show 10% decrease in nation-wide consumption after 2 years, and over $1B in government income.[2] A similar tax in Berkeley California demonstrated 10% decrease in sugar beverages, and 16% increase in water consumption.[123] Similar system-wide interventions should be adopted throughout North America and elsewhere, and research should be done to monitor effects on caries.

Many low-income parents have difficulty acting on health recommendations and in following through with intentions to attend classes and clinics.[124] An intervention that involves parents in identifying *their needs* and helps them overcome barriers to act on their needs is necessary.

Traditional health education is insufficient to change parental behavior in at-risk populations. Health education in dental and medical settings is frequently an attempt to persuade. What appears to be a convincing line of reasoning to the dental or medical professional falls on deaf ears or results in reluctance to change. Patients have reservations about "being told what to do." especially by a stranger.[125] More fundamental is the possibility that direct persuasion, whatever the degree of a patient's readiness to change, pushes the patient into a defensive position. While health education has not been successful, there have been promising results using Motivational Interviewing For example, Harrison and Wong reported that children whose mothers received at least two counseling sessions using Motivational Interviewing regarding children's oral health needs and disease prevention, had significantly less tooth decay than children in a comparison group.[126] The motivational approach featured one-to-one counseling by a lay worker, personalization of recommendations and telephone follow-up of mothers. An experimental study by Weinstein, Harrison and Benton compared a brief counseling intervention, again using Motivational Interviewing, with traditional oral health education to reduce tooth decay in a sample of 240 high-risk infants, 6 to 18 months of age. A 50% reduction in tooth decay was associated with the Motivational Interviewing intervention. [127–129]

Motivational Interviewing is a client-centered yet directive counseling approach. The conceptual basis is founded in the theory and research on self-regulation. Self-regulation models view individuals as active participants in reducing gaps between their perceived current status and immediate and long-term goals; health and illness behaviors are the result of the individual's representation of health threats and perceptions of the relevance of actions for managing or controlling these threats. The intervention approach that follows from the theory builds on client-centered counseling skills. It differs from traditional client-centered counseling in that the skills (i.e., open-ended questioning, affirmations and the reinforcement of self-efficacy, reflective listening, and summarizing) are used in a highly directive manner that moves clients toward self-examination and awareness of the problem and to understand how their current behavior is at odds with their desired goal. Motivational Interviewing utilizes the Stages of Change model to understand the process of change and select specific strategies to move clients from a stage of inaction to action.

Case management is the facilitation, coordination, and monitoring of services, the purpose of which is to provide individuals with the ability to engage in actions to better their health. Case management helps identify barriers that may preclude or interfere with client actions, helps develop strategies to overcome these barriers, and, at times provides advocacy for clients. In recent years, dental case management for families and children with low incomes was found to enhance dentist participation in Medicaid, and utilization of dental services and result in increased oral health literacy.[130] Other studies have used case management to integrate dental and medical care[131] and to overcome barriers to accessing dental school services.[132]

An age-guided model for dental public health

Assembling this chapter into an actionable recommendation for controlling dental caries using currently available materials is straightforward. Centralized approaches should include fluoridating the water and distributing fluoride toothpaste. Scalable hands-on approaches should include application of povidone iodine and fluoride varnish to high risk children with increased frequency at younger ages, silver diamine fluoride to all caries lesions, and glass ionomer cement to seal cavitation with circumferential coronal tooth structure (the combination of silver diamine fluoride and glass ionomer cement should be encouraged. When lesions in primary teeth are large the Hall technique should be employed. These hands-on approaches should be delivered in the field by hygienists, therapists, or assistants, where patients frequent: WICs, Head Starts, schools, Planned Parenthood, and long-term care facilities. The target populations are young children before 3 years of age and 6–8, pregnancy, and older age. Older caregivers of young children should be sought with pick-up and drop-off to child day programs. School supervised brushing programs should be widely implemented at young ages, with a “spit not rinse” approach starting at 8 years of age. Xylitol gum should be given to new mothers through WICs for at least one year when the child is ages 3 to 15 months, and similarly to older caries active caregivers. Vitamin D levels should be surveyed regionally during pregnancy and infancy and supplemented accordingly. Finally, assessment of the time of initial *S. mutans* colonization and the time of first apparent lesions for the target population of each public health unit should be done to inform the selection and timing of interventions.

The mainstay of caries prevention continues to be fluoride and control of sugar exposure, but metal ions, antiseptics, polyols, and vitamins may contribute as well. The mechanistic bases of protection are clear, in most cases, and comprehensive: fluorides increase the resilience of the tooth, reducing sugar exposure affects the cariogenic flora and acid production in the dental plaque, xylitol, silver and iodine decrease the load of *S. mutans*, and xylitol decreases transmission of *S. mutans*. These interventions are appropriate to address the life events, during which people are most susceptible to experiencing caries lesions and passing down the infection.

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Key Points

- Scarce public health resources should be directed toward intensive primary prevention of dental caries in toddlers and preschool-aged children, specifically prevention of transmission of cariogenic bacteria from mothers and frequent caretakers to children in the first years after tooth eruption.
- Expansion of school programs to include more strategies to atraumatically arrest lesions would increase program effectiveness.
- The risk and the need for primary prevention are not static but change across the life course.
- Public water and salt fluoridation, and taxes on sugar consumption are cost effective approaches to decrease disease risk and increase resistance to disease. Fluoride toothpaste should be distributed widely.
- Fluoride is not sufficient to control dental caries in high risk patients. Topical therapies and dietary modifications that decrease transmission of cariogenic bacteria or impact the capacity of dental plaque organisms (e.g. antimicrobials) to cause cavities should be employed.

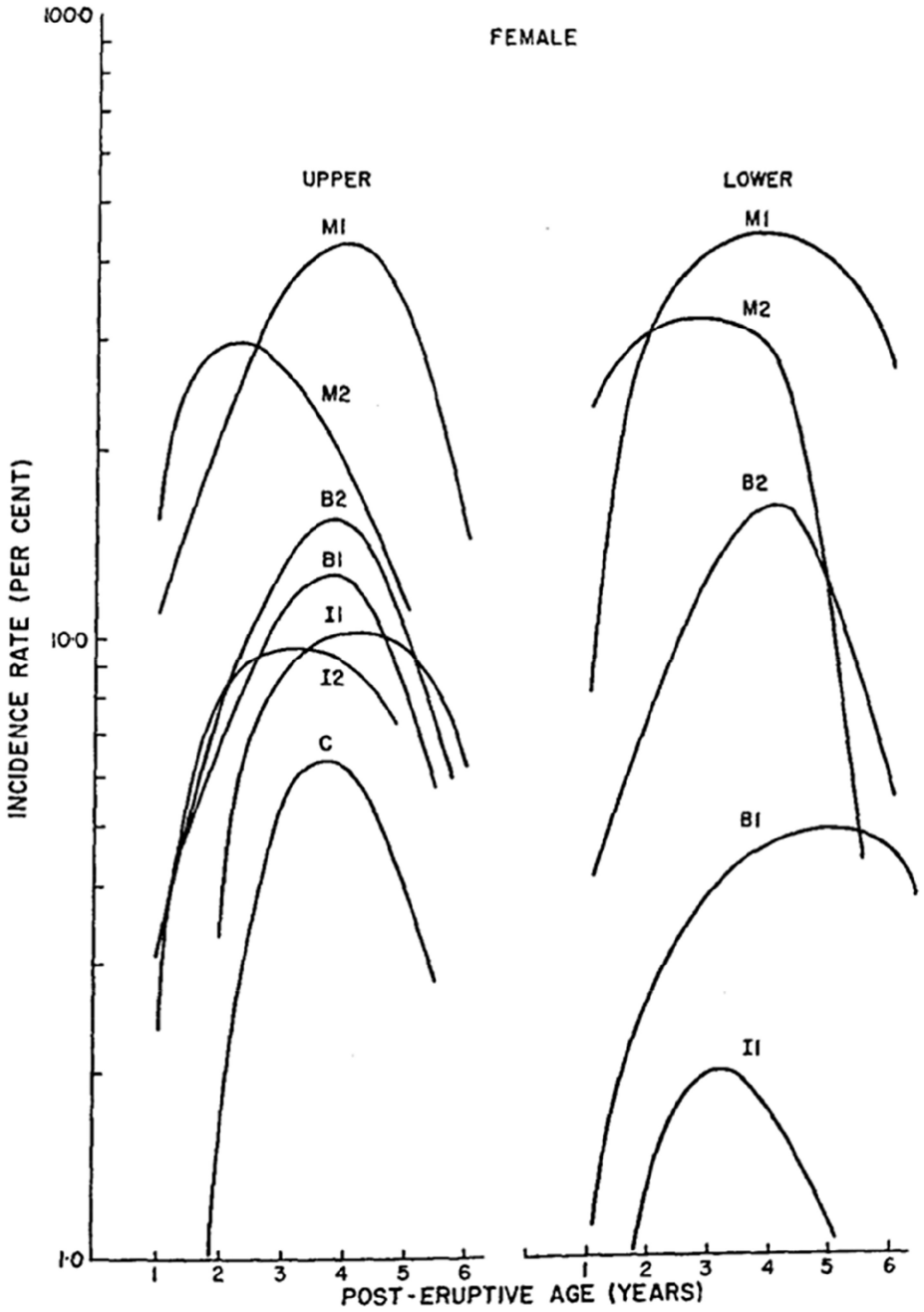

Figure 1.

Annual probability of caries attack (100 Q_x) of the permanent teeth, female children, Kingston, New York. Semi-logarithmic scale. Probabilities are slightly lower in male children. I1: central incisor, I2: lateral incisor, C: canine, B1: 1st bicuspid, B2: 2nd bicuspid, M1: 1st molar, M2: 2nd molar. *From* Carlos JP, Gittelsohn AM. Longitudinal studies of the natural history of caries—II. Arch Oral Biol. 1965;10(5):739–751; with permission.

Figure 2.

Figure Legend: One of the authors (PMM) brushing his grandson's teeth with fluoridated toothpaste.

Table 1

Prevention of caries lesions in children by treating mothers with xylitol gum.

first author	publication year	pubmed ID	mother-child diads	age at start (months)	xylitol frequency	duration of intervention	evaluation time	control	prevented fraction
Isokangas	2000	11145360	195	3	4/day	21 mon	5 yo	NaF 2/year	71%
Alamoudi	2014	24888659	60	10–36	3/day	3 mon	2y later	NaF 2/year	78%
Thorild	2006	17164069	173	6	3/day	1 year	4yo	NaF/xylitol/sorbitol	71%

Abbreviations: NaF sodium fluoride, mon months, y year, yo year old.

Table 2

Prevention of caries lesions with fluoride varnish in children starting at 1–3 years of age. 9 recent clinical trials evaluated prevented fraction of caries lesions in toddlers, documenting an unfortunate lack of effect.

first author	publication year	pubmed ID	children in study	age at start (months)	frequency (annual)	duration (years)	combined intervention	prevented fraction
Slade	2011	20707872	666	18–47	2	2	Guidance	31%
Lawrence	2008	18422711	1,275	6–71	2	2	none	25%
Tickle	2016	27685609	1,248	24–48	2	3	F toothpaste	25%
Ramos-Gomez	2012	21999806	361	12–36	2	2	maternal CHX	none
Jiang	2014	25448437	450	8–23	2	2	none	none
Agouropoulos	2014	25123352	424	24–71	2	2	none	none
Oliveira	2014	24481085	200	12–59	2	2	none	none
Memarpour	2015	25895964	140	12–36	3	1	none	none
Anderson	2016	26795957	3,403	12	2	2	none	none

Abbreviations: F fluoride, CHX chlorhexidine.

Table 3

Prevention of dental caries by placement of povidone iodine before fluoride varnish in toddlers.

first author	publication year	pubmed ID	patients	age at start (months)	frequency (annual)	duration of intervention	follow up	control	notes	prevented fraction
Lopez	2002	12064491	72	12–19 months	q 2months	1 year	1 year	F varnish only		80%
Amin	2004	15080351	25	2–7 years	q 2months	6 months	1 year	none	post-GA, no F varnish	71%
Simratvir	2010	20578661	30	μ = 4.2 years	q 3 months	1 year	1 year	water	post-GA, no F varnish	100%
Tut	2010	20337902	614	5–6 years	q 4 months	10 months	10 months	F varnish only		46%
Milgrom	2011	22126926	172	12–30 months	q 4 months	10 months	10 months	F varnish only		24%
Xu	2009	19417885	61	6–9 years	q 1 week	1 month	1 year	foam	foam iodine, no F varnish	0%
Zhan	2006	16913243	22	2–6 years	once	once	1 year	PBS, F gel	post-GA, F gel	0%

Abbreviations: F fluoride, μ mean, q *quater* (Latin: every), GA general anesthesia, PBS phosphate buffered saline.