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RESEARCH ARTICLE

PREVENTION OF EARLY CHILDHOOD CARIES- A REVIEW

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ABSTRACT

The expression "dental caries" is utilized to represent the outcomes, signs, symptoms, and side effects of a localized chemical disintegration of the tooth surface (enamel and dentin) caused by dental plaque and mediated by saliva[1]. Caries is considered as disease with high incidence among childhood chronic conditions, where it is also well-thought-out to cause harm on both population and individual well- being[2,3]. When comparing it with other common diseases, dental caries is five times as frequent as asthma and seven times as common as hay fever[4]. The American Academy of Pediatrics demonstrates that dental and oral infections keep on infecting children and, specifically, very young children. In primary teeth, dental caries is a preventable and reversible disease if treated in early stages, but when left untreated it will lead to pain, bacteremia, alteration in growth and development, premature tooth loss, speech disorder, increase in treatment costs, loss of confidence, and negatively affect successor permanent teeth. The definitions used previously to describe this bacterial disease were related to cause and the improper utilization of nursing bottle. These terms are used interchangeably: "Early childhood tooth decay", "early childhood caries (ECC)", "bottle caries", "nursing caries", "baby bottle tooth decay", or "night bottle mouth"[5,6]. The expression "ECC" was proposed more than 20 years ago during a workshop supported by the Centers for Disease Control and Prevention (CDC) trying to scope the consideration upon the various issues, such as financial, sociopsychological, and behavioral, which contributes to the formation of caries at such initial years, instead of attributing its manifestation solely on feeding bottles[7]. This article explores the various means of preventive strategies, including anticipatory guidance and future approaches to prevent ECC, interprofessional education and practise also explores the implications of ECC epidemiology for evolving health service delivery and financing approaches

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INTRODUCTION

ECC has been described as a chronic, dynamic, progressive, diet-dependent, fluoride-mediated infectious disease that results in dental lesions that are reversible at early stage[8]. Caries has also been characterized as a "complex" disease, such as diabetes, which arises from the combined actions of genetic, environmental, and/or infectious factors, along with risk-conferring behaviours[9]. Dental caries involves a shift in the balance between protective factors (that aid in remineralization) and destructive factors (that aid in demineralization) to favour demineralization of the tooth structure over time[10].

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ECC may manifest as either an isolated lesion or a rampant case, progressing rapidly in the primary dentition, beginning in infancy and affecting preschool children. If left untreated, it may result in pain and infection[8]. ECC is also largely preventable[11]. Infant oral health practices, use of fluorides and sealants, and recall preventive visits are opportunities to assess caries risk, offer anticipatory guidance, promote sound oral health practices, and provide risk-based primary prevention[12]. A first dental visit by age one and establishment of a dental home can mitigate a child's risk of oral disease over a lifetime[13]. Until recently, the standard of care of the dental profession once carious lesions manifest has been to rely primarily on surgical and restorative treatment[14]. Young children with ECC who are not cooperative and children with special health-care needs are commonly sedated or treated under general anaesthesia. Despite receiving such costly treatment[15,16,17,18], high rates of new and recurrent caries occur[19,20,21,22].

It is now accepted that surgical repair alone does not address the underlying etiology of the disease[9]. It is well established that caries is a dynamic process that can progress or regress, depending on a multitude of factors that can alter the normal balance of demineralization and remineralization. The balance of pathologic factors can be altered in favor of protective factors to slow down or completely halt the disease process. This may result in caries arrest, if not also preventing the onset of new disease. On the other hand, in individuals with active caries, without altering the caries balance to effectively manage the disease itself, new and recurrent caries are likely to occur[9,23,24]. In fact, previous caries experience is the best predictor of future caries experience in primary teeth[25].

DISEASE MANAGEMENT HEALTH CARE: Disease management (DM) has been defined as a system of coordinated healthcare interventions in which patient self-care efforts are significant[19]. There is in fact no single definition available for DM. Other descriptions of Disease Management(DM) activities include "educating patients/parents about their disease and how they can better manage it," "actively monitoring patients' clinical symptoms and treatment plans following evidence-based guidelines," "coordinating care for the disease among all providers," and "providing feedback on individual patients and support to physicians about patients' status between office visits as well as up-to-date information on best practices for particular patients"[26].

EVIDANCE BASED DISEASE MANAGEMENT: Published studies and reports have described Disease Management (DM) of caries through use of various chemotherapeutics. Outside of the United States, silver diamine fluoride has been found to be effective in arresting caries without removal of carious dentin and in preventing recurrence of ECC[27,28,29,30]. A case report suggests the potential of silver nitrate applications followed by fluoride varnish to manage ECC by arresting the decay[31]. Pilot studies suggest the promise of an antimicrobial agent such as topical iodine[32,33,34.] and emerging non fluoride remineralizing products such as casein phosphopeptide and calcium phosphate products[35,36], used alone or in combination with topical fluoride, to prevent and manage ECC. Xylitol, a sugar substitute that is a part of the polyol family, has been shown to reduce plaque formation and bacterial adherence and inhibit enamel demineralization. Studies have found that xylitol can decrease mutans streptococci (MS) levels in plaque and saliva and can reduce caries rates in young children and their mothers, along with decreasing the transmission of MS from mother to child[34]. Another study found that xylitol syrup (8 g/d) reduced ECC by 50–70% in children 15–25 months of age[37].

PREVENTION STARTS WITH MOTHER: Even if caries in pre-school children has been known since the beginning of the 20th century and, despite the progress in cures, early childhood caries (ECC) is still a public health problem, affecting both underdeveloped and industrialised nations[38].ECC is considered as one of the most prevalent diseases in childhood globally. Sugar has a pivotal role in caries development, mostly during the first years of life[39]. This, together with poor oral hygiene, increases the chance of caries onset tremendously. Thus, it is clear that preventive measures are focused on oral health-related behaviours and dietary habits. Based on these premises, paediatric dentists should focus on the mother's education in order to avoid the development of caries. The paediatric dentist has the duty to be the mothers' dentist, before of the child's. Mothers should be educated on their diet and oral hygiene and on the correct nutrition of the child, starting from the first month of age, limiting the child's intake of sugars and avoiding free sugars up to 2 years of age; dentists should inform the parents on when they have to start cleaning teeth (Fig 1) and when fluoride is needed[38]. Caries preventive measures should be established as early as possible. For a pediatric dentist, the only real success is not treating ECC with the latest technology, but visiting ECC-free children[38]

CHANGING ADVICE FOR CHANGING CHILD: This section describe the content of the six areas of Anticipatory

Fig 1, 2 Oral hygiene maintenance of child by the mother/caretaker

Guidance (AG): oral and dental development, fluoride use, diet and nutrition, oral hygiene, habits, and injury prevention[40]. We use the term "prethree" to describe children in the first 36 months of age throughout this chapter. Application of these topics to a child at any particular age in the prethree period requires an assessment of lifestyle, child development, family function, and parental ability. Feeding illustrates this dynamism of transition with a child primarily fed from birth by mother with bottle or breast with limited food choices and a fledgling dentition. The child may quickly transition into a day care setting and will begin solid foods at some point, so feeders and food may increase in complexity. The maxillary primary incisors historically present the first caries risk from excessive bottle use, but as the dentition matures and the diet changes, the posterior teeth become most susceptible to caries[40]. The ambulatory child may have access to foods at his or her discretion, and food may be used as a motivator or behavioral control rather than just for nutrition.

Children who are new to a dental home may need to have all elements of Anticipatory Guidance to the left of their age line provided because the dentist cannot assume the behaviors or risk have already been dealt with. The principles of Anticipatory Guidance should be remembered in the context of the other three overall goals of health supervision that are disease detection, disease prevention, and health promotion. Anticipatory Guidance should be seen as the hands-on, direct application of preventive strategies[40]

FLUORIDES AG IN PRETHREE CHILD: Fluoride remains one of the three most critical areas of AG because of its known benefits for oral health and the potential risks of misuse. but the content of discussion in all phases of AG is the same. Is systemic intake optimal? Is the child using fluoride toothpaste and doing so appropriately? Is the presence of fluoride products in the home safe? At every supervision visit, fluoride needs to be reviewed because of the dynamic nature of exposure. A good way to approach fluoride in this age period is as one would a medication. Indications, benefits, dosages, route of administration, side effects, and refill information should all be covered[40]. Fluoridated dentifrices have proved their effectiveness as an effective anticarious agent. Recent reviews indicate that fluoridated dentifrices reduce caries by approximately 25%[41,42]. Fluoridated dentifrices have fluoride available as sodium fluoride, stannous fluoride, and monofluorophosphate.

All three of these fluoride compounds are recognized for effectiveness in the reduction of caries by the FDA, and they all exhibit similar cariostatic effects. Most dentifrices have a fluoride level of 1000 or 1100 ppm, but 1500 ppm is also available. There is a 5000ppm fluoride dentifrice, but it must be professionally prescribed. The risk of swallowing fluoridated dentifrices is higher among younger children and children who tend to use “child-flavored” dentifrices in greater amounts and for longer time of brushing[43,44,45]. For this reason, it is recommended that a pea-sized amount of dentifrice be applied to the toothbrush by the child’s caregiver to prevent ingestion of undesirable amounts of toothpaste[46]. Tooth brushing should be performed by an adult caregiver for children until at least age 5, when coordination improves with the child’s tooth brushing. When the children begin to brush their own teeth, the dentifrice should still be dispensed by the caregiver, as well as having the brushing evaluated by the caregiver. Tooth brushing should be performed twice per day[47]. When a child is old enough to effectively expectorate, more than a pea-sized amount of dentifrice can be used to increase the Fig 3 Amount of fluoridated toothpaste recommended level of fluoride exposure (Fig 3)

Fig. 3. Amount of fluoridated toothpaste recommended

DIET AND NUTRITION CONSIDERATION OF PRETHREE CHILD: Bottle-feeding ad lib and nocturnally continues to occur with frequency and has been documented in a large number of working families well into the third year of life[48], so in the 3-year AG paradigm, entry at any age demands the dentist ask about the bottle. Clinicians can no longer assume that simple admonition about the risk of nocturnal bottle use will result in behaviour change. The pressures of work, single parenting, and caretaking multiple children may make ad lib and nocturnal bottle or sippy cup feeding a convenient behaviour modification tool for parents. Reasonable and workable alternatives need to be offered to families to break the habit (Fig 4).

Fig. 4. Sippy cup with plain water an alternative to break the habit

Poor sugar control looms large as a reason for the early childhood epidemic. The amount of sugar in the prethree child population has grown in the form of carbonated beverages and sweetened juices, displacing milk as the beverage of choice. The transition from bottle to solid food should be the latest point at which consideration of sugar intakes is done in AG and in many instances delaying until then can result in dental caries. At an initial AG visit, sugar intake should be screened, and if needed, a more detailed diet history done to identify amount, frequency, and type of sugar consumed. The dentist needs to have a thorough awareness of the prethree diet to be able to make realistic recommendations for alternatives. This may require the assistance of a dietician. A simple mandate to reduce sugar intake without workable alternatives is doomed to failure[40].

A final consideration is the safety of dental-friendly diet alternatives. Traditionally, these have been nutritious but also high in fat and salt. The negative contribution of dietary fat and sodium in dental snacks at this age is not well understood, but concerns about obesity stem from our understanding that dietary habits are fixed in the first 2 years of life. Dental personnel cannot recommend high-sodium, fatty snacks to benefit oral health if they contribute to obesity and systemic problems such as elevated blood pressure and diabetes. In addition, choking on nuts or chunks of hot dogs is a real risk, and parents need to be instructed on how to prepare and serve these tooth-friendly food alternatives[40].

APPLICATION OF ORAL HYGIENE TO PRETHREE CHILD: Once teeth erupt, an age-appropriate brush should be used. Because parents will be performing the cleaning, the appropriate brush is one with a long handle easy for an adult to

grasp and a small head to fit comfortably in the prethree's mouth (Fig 5, 6) An appropriate location to perform the cleaning would be a place where the parent can stabilize the child and have good access to and visualization of the mouth. In today's busy world, this is often the bathroom in conjunction with other hygiene activity. Most likely, the prethree child will "fuss" with brushing. Parents need to be creative and innovative to create a "fun" time. This may include distraction with music, singing, or an egg timer watched by the child[40].

Other than with the at-risk infant, a fluoridated dentifrice before age 2 is not indicated. If you feel it would reduce the child's risk, then a fluoride-containing dentifrice can be recommended, but used sparingly. Prethree children's ability to expectorate is limited and messy at best. Flossing is generally not recommended for the prethree child until the interdental contacts have been established, and even then, it will be the parent's responsibility.

Fig 5, 6. Application of oral hygiene to prethree child

Flossing may introduce an additional unnecessary and burdensome step that has little support from evidence as to its anticaries benefit[40] In providing AG to parents for oral hygiene procedures, it is important to demonstrate application of dentifrice, a full "round" of tooth cleaning, and positioning. Do not assume that a parent can effect plaque removal without some instruction. Critical to a successful home hygiene program is its integration into the lifestyle of the family. Considerations must include location, timing, selection of devices and their expense, positioning, and problem solving relative to other needs of the child[40].

INTERPROFESSIONAL COLLABORATION: Health outcomes for children are determined at the level of the individual, family, and community[49]. Research has shown that improving health outcomes is far more complex than having access to medical and dental care alone[50]. Achieving and maintaining good health require the active and collaborative participation of the professional health-care team, individual and family, and community. Thus, a framework for understanding children's oral health outcomes should incorporate biologic, psychosocial, and environmental determinants. Any program or partnership hoping to improve health, including oral health, must be evidence based and consider the family, community, health-care infrastructure, and policy[4.] Care coordination, at both the system and service delivery levels, has been identified as a fundamental strategy to achieve these aims[51], and while this makes intuitive sense, collaborative practice remains a care delivery paradigm shift[52]. In 2013, The Macy Foundation described the need to align interprofessional educational efforts with clinical redesign so practices can effectively deliver team-based care. Their recommendations included:

- Engage patients, families, and communities in the design, implementation, improvement, and evaluation of efforts to link interprofessional education (IPE) and collaborative practice.
- Reform the education and lifelong career development of health professionals to incorporate interprofessional learning and team-based care.
- Accelerate the design, implementation, and evaluation of innovative models linking IPE and collaborative practice.
- Revise professional regulatory standards and practices to permit and promote innovation in IPE and collaborative practice.
- Realign existing resources to establish and sustain the linkage between IPE and collaborative practice[53].

DENTAL HOME: The concept of the dental home is an evolving one, with many conceptual elements borrowed from the definition of the medical home propagated by the American Academy of Pediatrics (2002). In 2002, Nowak and Casamassimo introduced the concept of the dental home as a primary health concept to address the early childhood caries epidemic and other aspects of oral health to the general dental profession[54.] A working definition, according to the American Academy of Pediatric Dentistry (AAPD) (2013), is as follows: The dental home is the ongoing relationship between the dentist and the patient, inclusive of all aspects of oral health care delivered in a comprehensive, continuously accessible, coordinated and family centered way. Establishment of a dental home happens no later than 12 months of age and includes referral to dental specialists when appropriate.

Definitions of the dental home may vary and will likely change to meet the dynamics of disease occurrence, population shifts, scientific advances, and changes in the health-care system. Some aspects of the dental home are rudimentary to health care such as physical and fiscal access and cultural effectiveness in providing care to families with diverse backgrounds and with children with special needs. Other aspects of the dental home address elements of the oral health-care system that are somewhat new such as family centeredness and care coordination. This provides some insight into what the dental home encompasses and what dental practices need to do to develop the concept into a working system[40].

Treatment Modalities: Early diagnosis of enamel demineralization allows for early intervention to remineralize enamel and to evaluate the reason for demineralization. The oral balance of demineralization/remineralization must be controlled to prevent progression of early lesions and the initiation of new lesions[40].

ANTIMICROBIALS

CHLORHEXIDINE: Chlorhexidine has demonstrated antimicrobial effectiveness through numerous well-controlled clinical trials[55,56]. Chlorhexidine is1,6-bis-4-chloro-phenyldiguanidohexane, a synthetic cationic detergent. It has great bacteriostatic and bacteriocidal features and was originally used to treat dermatologic infections, wound surfaces, and eye and throat infections. When chlorhexidine was originally tested for efficacy in plaque control, 10 ml of a 0.2% chlorhexidine digluconate rinse demonstrated successful plaque control with subsequent inhibition of gingivitis[57,58]. Other studies have demonstrated the effectiveness of 0.12% chlorhexidine digluconate solution, the formulation available in the United States, to effectively reduce plaque and gingivitis[59,60]. The cationic chlorhexidine molecule binds to anionic compounds, such as free sulfates, carboxyl and phosphate groups, and salivary glycoproteins[61].

This action will reduce the adsorption of proteins to the tooth surface, delaying the formation of the dental pellicle. Chlorhexidine molecules also coat salivary bacteria, which alter the mechanisms of adsorption of bacteria to the tooth. The main side effects of chlorhexidine are staining of the teeth and taste and the content of ethyl alcohol. The stain on the teeth can be easily removed with a pumice prophylaxis. Since chlorhexidine can temporarily affect taste sensations, use around mealtimes is not recommended. It has been recommended that high-risk patients with high intraoral bacterial levels rinse 10 ml of 0.12% chlorhexidine digluconate solution once per day for 1 week every 6 months[62]. Since children less than 3 years of age would not be appropriate for rinsing, this would be more pertinent to mothers at high risk for caries development with high intraoral bacterial levels. Chlorhexidine is also available in gels and varnish; however, these are not currently available in the US marketplace. The gels containing chlorhexidine(Fig 7) have contained 1 or 2% chlorhexidine digluconate. The 2% gel has been shown to be effective when used as a dermatologic wound healing agent[63]. Gels with 1% chlorhexidine digluconate incorporated into the gel have shown efficacy in reducing caries when applied for 5 min/day over a period of 2 weeks[64].

Fig 7. Chlorhexidine gel

The chlorhexidine does not diffuse as rapidly from a gel as a rinse; therefore, it needs a longer contact time, as well as direct application to the tooth surface, to be effective. In a longitudinal study using 0.2% chlorhexidine gel weekly in 10-monthold infants, it was found that no differences were observed when compared to a placebo group and to a treatment group at follow-up evaluations after 3 months[65]. A clinical trial evaluating the use of a 40%, by weight, chlorhexidine varnish in Chinese preschool children indicated a positive anticaries effect. The preschool children received 6-monthly applications of the 40% chlorhexidine varnish, and a control group received a placebo varnish at the same application intervals. At 2 years, the chlorhexidine group demonstrated a 37% reduction in caries compared to the control. This chlorhexidine varnish anticariogenic effect was also seen in children evaluated in other studies[66,67].

IODINE: Studies have indicated that topical iodine agents can significantly suppress levels of mutans streptococci[68,69]. Therefore, studies have examined the effectiveness of iodine agents to inhibit the development of early childhood caries. The application of 10% povidone iodine, to the tooth surfaces of 83 high-cariesrisk children (12–19 months), was performed every 2 months in a study for duration of 12 months. The children that received this treatment developed significantly fewer white spot lesions than a control group that received treatment with a placebo agent. Further research will indicate the long-term effects of iodine treatment, when it is being applied and when it has been removed as an antibacterial agent[40].

XYLITOL: Xylitol is a sugar substitute that has 40% fewer calories than sucrose[70]. Sugar alcohols, such as xylitol, sorbitol, mannitol, and maltitol, have been shown to be noncariogenic[71,72]. The literature indicates that xylitol also reduces the level of mutans streptococci in plaque and reduces the level of lactic acid produced by bacteria[73].Xylitol consumption in the range of 6–10 g/day, divided into at least three time periods, is effective in reducing bacteria levels and subsequent drop in acid production[74]. The influence of maternal xylitol consumption on the mother's transmission of bacteria to their child has also been evaluated[75,76]. Xylitol has been shown to reduce bacteria in the oral cavity; therefore, the potential for this to reduce the transmission from mother to child would be a means to reduce early childhood caries. Xylitol is also available in wipes to clean the teeth of infants (Fig 8) This can be an effective means of providing oral hygiene maintenance and xylitol at the same time. There are many other products that contain xylitol[74].

SALIVA: Saliva is very important in providing remineralization effects for tooth structure. Since saliva is supersaturated with calcium and phosphate, which bathes the teeth, remineralization can occur with the deposition of minerals into subsurface enamel lesions. Saliva is also important as a buffering agent. This is critical to control the pH of the oral environment. Buffering can be attributed, in part, to bicarbonate in stimulated salivary secretions and peptides, as well as amino acids in unstimulated saliva[77]. Furthermore, salivary proteins aid in antimicrobial activity by inhibiting bacterial growth[78]. Examples of these proteins would include histatins, lactoferrin, peroxidase, and lysozyme. An adequate salivary flow rate is considered to be approximately 1 ml/min. If salivary flow is reduced to less than 0.5 ml/min, interventions should be considered.

Fig 8. Various forms of Xylitol

Artificial saliva can be utilized, greater consumption of water can be recommended, and chewing gum has been shown to stimulate salivary flow[79]. Reduced salivary flow, which increases the risk of caries, would indicate the appropriateness of increased fluoride exposure and increased exposure to calcium- and phosphate-containing agents.

DIET EVALUATION: Dietary intake plays a role in the status of the oral cavity. Intake of sugar (sucrose) is known to decrease the pH level to the point of causing tooth demineralization[40]. In fact, any fermentable carbohydrate can initiate and progress carious lesions. As a part of risk assessment, intake of fermentable carbohydrates is important to know. Of particular importance is the frequency of intake. Each exposure can drop the pH; therefore, the greater the number of times fermentable carbohydrates enter the oral cavity (snacking, juice, and soda drinking), the greater the amount of times the pH within the oral cavity is prone to caries initiation/progression Practitioners can recommend that frequency of exposure to fermentable carbohydrates be reduced[80].

PROFESSTIONALLY APPLIED TOPICAL FLUORIDES:

FLUORIDE VARNISH: Fluoride varnishes, although available in Europe for years as an anticaries agent, is recognized by the US FDA as a device to be used as a desensitizing agent and a cavity-lining varnish[81]. Fluoride varnish is available as 5% sodium fluoride (22,600 ppm fluoride) and 1% difluorosilane (1,000 ppm fluoride).

There is minimal information regarding the effectiveness of fluoridated varnishes to enhance remineralization; however, early data indicate that fluoride varnish has the potential to aid in the remineralization of incipient caries[82,83.] Ekstrand and colleagues reported a low plasma fluoride level following placement of a 5% fluoride varnish, which was comparable to plasma fluoride levels experienced after toothbrushing with a fluoridated dentifrice[84]. This level is significantly lower than plasma fluoride levels seen after a professionally applied 1.23% acidulated phosphate fluoride[85]. Since the placement of fluoride trays in young children is difficult, cooperation is difficult with young children to use slow-speed suction to remove excess fluoride from the mouth as it dissipates from the delivery tray and the inability to ensure young children will not swallow fluoride in a tray delivery system— young children can benefit from fluoridated varnish. The ease of varnish application, safety, and efficacy, comparable to 1.23% acidulated phosphate fluoride gel, makes the use of fluoride varnish appropriate for young children[86] (Fig 9)

Fig 9. Professionally applied fluoride varnish

PROFESSIONALLY APPLIED FUORIDE GELS AND FOAMS: There are three professionally applied topical fluorides recognized by the American Dental Association (ADA): 1.23% acidulated phosphate fluoride, 2% sodium fluoride, and 8% stannous fluoride. All three of these professionally applied topical fluorides have demonstrated success in reducing caries; however, they are difficult to use with small children[87]. As previously discussed, tray-delivered fluoride is difficult in young children; therefore, fluoride varnish is preferable as a professionally applied topical fluoride.

Caseinphosphopeptide–amorphous calcium phosphate: Caseinphosphopeptide–amorphous calcium phosphate (CPP–ACP) (Fig 10) has received significant attention over the past decade to aid in the control of caries. CPP stabilizes ACP in metastable solution[88]. Through multiple phosphoseryl residues, CPP binds to forming nanoclusters of ACP, preventing their growth to the critical size required for nucleation and phase transformation. The CPP–ACP attaches to plaque, the ACP being released onto the tooth surface. Not only does this provide calcium and phosphate for tooth remineralization but also acts as a buffering agent when the intraoral pH becomes more acidic.

CPP–ACP rinse: There has been evidence that enamel subsurface lesions can be remineralized with CPP-stabilized

calcium phosphate solutions[89]. Although these remineralizing solutions can be effective at remineralizing enamel, children at age 3 and less would have a difficult time with a rinse and other delivery systems of CPP–ACP would be more appropriate.

CPP–ACP gum: Studies have also shown the effectiveness of CPP–ACP contained in sugar-free gum to remineralize subsurface enamel lesions[90,91]. The trademark name for CPP–ACP is Recaldent™. Gums containing CPP– ACP offer benefits from the delivery of bioavailable calcium and phosphate, as well as improving salivary flow, which is supersaturated with calcium and phosphate. An additional benefit can occur if xylitol is used as the sweetener in the gum, xylitol exhibiting anticariogenic effects on bacteria. A clinical trial evaluating a sugar-free gum containing CPP–ACP chewed for 10 min three times daily by 2720 adolescents demonstrated a significant reduction in lesion progression, as well as enhancement of lesion reversal when compared to a sugar-free control gum[92]. Although these gums containing CPP–ACP enhance remineralization of subsurface enamel lesions, children at age 3 and less may not have the ability/ coordination to chew gum. If children are unable to chew gum, application of CPP–ACP in another form would be appropriate.

CPP–ACP paste: CPP–ACP is available in a paste form, which is referred to as MI Paste (GC America Inc, Alsip, IL). This CPP–ACP-containing MI Paste is not only available in North America but is also available in Australia and New Zealand with the product name Tooth Mousse. The paste can be applied to the teeth gently with a rubber cup or gloved finger by the dental professional and can be applied at home by the patient, or parent of the patient, using a finger or toothbrush.

The paste is recommended to be placed on the labial surfaces of the teeth, in a pea-sized amount, every day before bedtime[93]. Ingestion of this agent has been classified as safe for patients of all ages. Since saliva flow decreases when sleeping, the CPP–ACP paste would be expected to have a greater contact time and subsequent benefit if applied prior to bedtime. Although CPP–ACP paste may be of benefit, particularly in children with compromised salivary flow, a randomized clinical trial on high-caries-risk children showed no benefit over the control over 1 year with five times per week application[94].

CPP–ACP paste with fluoride: A new paste was recently introduced to the marketplace that contains CPP–ACP with 900 ppm fluoride (MI Paste Plus, GC America Inc, Alsip, IL). This fluoridated paste has bioavailable calcium and phosphate, yet also has approximately the same amount of fluoride available as that provided in dentifrices. CPP has been shown to stabilize amorphous calcium fluoride phosphate. MI Paste Plus compared to MI Paste remineralizes subsurface enamel lesions better[93]. This is attributed to the fluoride availability that enhances the precipitation of calcium and phosphate. Although this fluoridated CPP–ACP paste is effective in enamel remineralization, it is not indicated in young children. The entire fluoride content of the paste is expected to be swallowed; therefore, the concern for increased potential for fluorosis limits the recommendation for use of fluoridated CPP–ACP in young children.

Fig 10. Various forms of CPP-ACP

Other CPP–ACP carriers: CPP–ACP has also been incorporated into dental sealants and dental varnishes. A slow release of the calcium and phosphate would seem to be beneficial; however, little research is presently available for these carriers of CPP–ACP, and further information should become available in the near future.

Conclusion

Our goal, as oral health-care professionals, is to provide our patients and their families with the education and tools to increase the probability that they will have the best oral health possible. This translates into identifying the risk for disease early so that we can prevent the manifestations of disease. When we do not succeed in this, we need to have effective tools to treat and manage the disease process. Research in medicine, biotechnology, and materials science has both direct and indirect applications in dentistry that are yet to be investigated[40]. Prevention and management of this devastating disease in children will be best accomplished by coordinating efforts with the many individuals and groups who are dedicated to the well-being of children. Many of these groups were discussed in this review and include professional dental associations, families, public health workers, and researchers. With the dedication of all of these individuals, we can make a difference in the lives of our youngest citizens[40]

REFERENCES

1. Fejerskov, O., Kidd, E. Dental caries: the disease and its clinical management. Hoboken (NJ): John Wiley & Sons; 2009
2. Chu CH, Fung DS, Lo EC. Dental caries status of preschool children in Hong Kong. Br Dent J 1999 Dec;187(11):616-620.
3. Stecksén-Blicks C, Sunnegårdh K, Borssén E. Caries experience and background factors in 4-year-old children: time trends 1967-2002. Caries Res 2004 Mar-Apr;38(2):149-155.
4. Bagramian RA, Garcia-Godoy F, Volpe AR. The global increase in dental caries. A pending public health crisis. Am J Dent 2009 Feb;22(1):3-8.
5. Dilley GJ, Dilley DH, Machen JB. Prolonged nursing habit: a profile of patients and their families. ASDC J Dent Child 1980 Mar-Apr;47(2):102-108.
6. Ismail AI, Sohn W. A systematic review of clinical diagnostic criteria of early childhood caries. J Public Health Dent 1999 Summer;59(3):171-191.

7. Schroth RJ, Brothwell DJ, Moffatt ME. Caregiver knowledge and attitudes of preschool oral health and early childhood caries (ECC). Int J Circumpolar Health 2007 Apr;66(2):153-167.
8. Edelstein BL, Chinn CH, and Laughlin RJ. 2009. Early childhood caries: definition and epidemiology. In: Berg JH and Slayton RL (eds), Early Childhood Oral Health. Ames, IA: Wiley-Blackwell
9. Fontana M and Wolff M. 2011. Translating the caries management paradigm into practice: challenges and opportunities. J Calif Dent Assoc 39:702–8
10. Featherstone JD. 2000. The science and practice of caries prevention. J Am Dent Assoc 131:887–99
11. Ramos-Gomez F and Ng MW. 2011. Into the future: keeping healthy teeth caries free: pediatric CAMBRA protocols. J Calif Dent Assoc 39:723–33.
12. Ng MW, Fida Z. Early childhood caries disease prevention and management. InEarly Childhood Oral Health 2016 (pp. 47-66). John Wiley & Sons, Inc, Hoboken, New Jersey.
13. American Academy of Pediatric Dentistry. 2012. Guideline on infant oral health care. Pediatr Dent 35:137–41.
14. American Academy of Pediatric Dentistry. 2008. Policy on early childhood caries (ECC): classifications, consequences, and preventive strategies. Pediatr Dent 30:40–3
15. Duperon DF. 1995. Early childhood caries: a continuing dilemma. J Calif Dent Assoc 23:15–6, 18, 20–2 passim
16. Griffin SO, Gooch BF, Beltran E, Sutherland JN, and Barsley R. 2000. Dental services, costs, and factors associated with hospitalization for Medicaid-eligible children, Louisiana 1996–97. J Public Health Dent 60:21–7
17. Kanellis MJ, Damiano PC, and Momany ET. 2000. Medicaid costs associated with the hospitalization of young children for restorative dental treatment under general anesthesia. J Public Health Dent 60:28–32.
18. Ramos-Gomez FJ, Huang GF, Masouredis CM, and Braham RL. 1996. Prevalence and treatment costs of infant caries in Northern California. ASDC J Dent Child 63:108–12
19. Almeida AG, Roseman MM, Sheff M, Huntington N, and Hughes CV. 2000. Future caries susceptibility in children with early childhood caries following treatment under general anesthesia. Pediatr Dent 22:302–6
20. Berkowitz RJ, Moss M, Billings RJ, and Weinstein P. 1997. Clinical outcomes for nursing caries treated using general anesthesia. ASDC J Dent Child 64:210–1, 228
21. Eidelman E, Faibis S, and Peretz B. 2000. A comparison of restorations for children with early childhood caries treated under general anesthesia or conscious sedation. Pediatr Dent 22:33–7
22. Graves CE, Berkowitz RJ, Proskin HM, Chase I, Weinstein P, and Billings R. 2004. Clinical outcomes for early childhood caries: influence of aggressive dental surgery. J Dent Child (Chic) 71:114–17
23. Featherstone JD. 2006. Caries prevention and reversal based on the caries balance. Pediatr Dent 28:128–32
24. Llena Puy C and Forner Navarro L. 2008. Evidence concerning the medical management of caries. Med Oral Patol Oral Cir Bucal13:E325–30
25. Zero D, Fontana M, and Lennon AM. 2001. Clinical applications and outcomes of using indicators of risk in caries management. J Dent Educ 65:1126–32.
26. Congressional Budget Office. 2004. An Analysis of the Literature on Disease Management Programs. Washington, DC: Congressional Budget Office
27. Chu CH, Lo EC, and Lin HC. 2002. Effectiveness of silver diamine fluoride and sodium fluoride varnish in arresting dentin caries in Chinese pre-school children. J Dent Res 81:767–70.
28. Llodra JC, Rodriguez A, Ferrer B, Menardia V, Ramos T, and Morato M. 2005. Efficacy of silver diamine fluoride for caries reduction in primary teeth and first permanent molars of schoolchildren: 36-month clinical trial. J Dent Res 84:721–4
29. Rosenblatt A, Stamford TC, and Niederman R. 2009. Silver diamine fluoride: a caries “silver- fluoride bullet”. J Dent Res 88:116–25
30. Yee R, Holmgren C, Mulder J, Lama D, Walker D, and Van PalensteinHelderman W. 2009. Efficacy of silver diamine fluoride for Arresting Caries Treatment. J Dent Res 88:644–7.
31. Duffin S. 2012. Back to the future: the medical management of caries introduction. J Calif Dent Assoc 40:852–8
32. Amin MS, Harrison RL, Benton TS, Roberts M, and Weinstein P. 2004. Effect of povidone-iodine on Streptococcus mutans in children with extensive dental caries. Pediatr Dent 26:5–10.
33. Lopez L, Berkowitz R, Spiekerman C, and Weinstein P. 2002. Topical antimicrobial therapy in the prevention of early childhood caries: a follow-up report. Pediatr Dent 24:204–6.
34. Milgrom P and Chi DL. 2011. Prevention-centered caries management strategies during critical periods in early childhood. J Calif Dent Assoc 39:735–41
35. Milgrom P, Zero DT, and Tanzer JM. 2009b. An examination of the advances in science and technology of prevention of tooth decay in young children since the Surgeon General’s Report on Oral Health. AcadPediatr 9:404–9
36. Robertson MA, Kau CH, English JD, Lee RP, Powers J, and Nguyen JT. 2011. MI Paste Plus to prevent demineralization in orthodontic patients: a prospective randomized controlled trial. Am J Orthod Dentofacial Orthop 140:660–8.
37. Milgrom P, Ly KA, Tut OK, Mancl L, Roberts MC, Briand K, and Gancio MJ. 2009a. Xylitol pediatric topical oral syrup to prevent dental caries: a double-blind randomized clinical trial of efficacy. Arch PediatrAdolesc Med 163:601–7
38. Paglia L. Oral prevention starts with the mother. European journal of paediatric dentistry. 2019 Sep 1;20(3):173-
39. Paglia L, Friuli S, Colombo S, Paglia M. The effect of added sugars on children’s health outcomes: Obesity, Obstructive Sleep Apnea Syndrome (OSAS), Attention-Deficit/Hyperactivity Disorder (ADHD) and Chronic Diseases. Eur J Paediatr Dent 2019; 20(2);127-132.
40. Berg JH, Slayton RL. Early childhood oral health. John Wiley & Sons; 2015 Oct 26.
41. Twetman S, Axelsson S, Dahlgren H, Holm AK, Kallestal C, Lagerlof F, Lingstrom P, Mejare I, Nordenram G, Norlund A, Petersson L, and Soder B. 2003. Caries preventive effect of fluoride toothpaste: a systematic review. Acta OdontolScand 61:347–55
42. Marinho VCC, Higgins JPT, Logan S, and Sheiham A. 2005. Fluoride toothpastes for preventing dental caries in

children and adolescents. Cochrane Database Syst Rev 4:CD002278.
43. Levy SM, Maurice TJ, and Jakobsen JR. 1992. A pilot study of preschoolers' use of regular flavored dentifrices and those flavored for children. Pediatr Dent 14:388–91
44. Naccache H, Simard PL, Trahan L, Brodeur JM, Demers M, Lachapelle D, and Bernard PM. 1992. Factors affecting the ingestion of fluoride dentifrice by children. J Public Health Dent 52:222–6
45. Adair SM, Piscitelli WP, and McKnight-Hanes C. 1997. Comparison of the use of an adult and a child dentifrice by a sample of preschool children. Pediatr Dent 19:99–103.
46. American Academy of Pediatric Dentistry. 2007c. Fluoride therapy. Reference manual 2006–7. Pediatr Dent 28(Suppl):95–6.
47. Chestnut IG, Schafer S, Jacobsen APM, and Stephen KW. 1998. The influence of tooth-brushing frequency and postbrushing rinsing on caries experience in a caries clinical trial. Community Dent Oral Epidemiol 26:406–11
48. Hammer LD and Bryson S. 1999. Development of feeding practices during the first 5 years of life. Arch PediatrAdolesc Med 153:189–94.
49. Fisher-Owens SA, Gansky SA, Platt LJ, Weintraub JA, Soobader MJ, Bramlett MD, and Newacheck PW. 2007. Influences on children's oral health: a conceptual model. Pediatrics 120(3):e510–20
50. Aday L and Andersen R. 1974. A framework for the study of access to medical care. Health Serv Res 9(3):208–20
51. McDonald KM, Sundaram V, Bravata DM, Lewis R, Lin N, Kraft S, McKinnon M, Paguntalan H, and Owens DK. 2007. Care coordination. In: Shojania KG, McDonald KM, Wachter RM, and Owens DK (eds), Closing the Quality Gap: A Critical Analysis of Quality Improvement Strategies. Technical Review 9.7 (Prepared by the Stanford University-UCSF Evidence-based Practice Center under contract 290-02-0017), Vol 7. Rockville, MD: Agency for Healthcare Research and Quality. AHRQ Publication No. 04(07)-0051-7
52. Bisognano M and Kenney C. 2012. Pursuing the Triple Aim: Seven Innovators Show the Way to Better Care, Better Health, and Lower Costs. San Francisco, CA: Jossey-Bass Publishers.
53. Cox M and Naylor M. 2013. Transforming patient care: aligning interprofessional education with clinical practice redesign. In: EDITOR. Proceedings of a Conference sponsored by the Josiah Macy Jr. Foundation in January 2013; New York: Josiah Macy Jr. Foundation
54. Nowak AJ and Casamassimo PS. 2002. The dental home: a primary care concept. J Am Dent Assoc 133:93–8
55. Lang NP and Brecx MC. 1986. Chlorhexidine digluconate—an agent for chemical plaque control and prevention of gingival inflammation. J Periodontal Res 21(Suppl):74–89.
56. Anderson MH. 2003. A review of the efficacy of chlorhexidine on dental caries and the caries infection. Can Dent Assoc J 31:211–14.
57. Davies RM, Jensen SB, Schiött CR, and Löe H. 1970. The effect of topical application of chlorhexidine on the bacterial colonization of teeth and gingiva. J Periodontal Res 5:96–101
58. Löe H and Schiött CR. 1970. The effect of suppression of the oral microflora upon the development of dental plaque and gingivitis. In: McHugh WE (ed.), Dental Plaque. Edinburgh: E&S Livingstone, pp 247–55.
59. Lang NP and Briner WW. 1984. Chemical control of gingivitis in man. J Am Dent Assoc 109:223
60. Siegrist BE, Gusberti FA, Brecx MC, Weber HP, and Lang NP. 1986. Efficacy of supervised rinsing with chlorhexidine digluconate in comparison to phenolic and plant alkaloid compounds. J Periodontal Res 16(Suppl):60–73
61. Rölla G and Melsen B. 1975. On the mechanism of the plaque inhibition by chlorhexidine. J Dent Res 54(Special Issue B):57–62
62. Featherstone JDB. 2006. Caries prevention and reversal based on the caries balance. Pediatr Dent 28:128–32
63. Asboe-Jörgensen V, Attström R, Lang NP, and Löe H. 1974. Effect of chlorhexidine dressing on healing after periodontal surgery. J Periodontol 45:13–17.
64. Zickert I, Emilson CG, and Krasse B. 1982. Effect of caries preventive measures in children highly infected with Streptococcus mutans. Arch Oral Biol 27:861–8.
65. Wan AKL, Seow WK, Purdie DM, Bird PS, Walsh LJ, and Tudehope DI. 2003. The effects of chlorhexidine gel on Streptococcus mutans infection in 10- month-old infants: a longitudinal, placebo-controlled, double-blind trial. Pediatr Dent 25:215–22
66. Achong RA, Briskie DM, Hildebrandt GH, Feigal RJ, and Loesche WJ. 1999. Effect of chlorhexidine varnish mouthguards on the levels of selected oral microorganisms in pediatric patients. Pediatr Dent 21:169–75.
67. Forgie AH, Paterson M, Pine CM, Pitts NB, and Nugent ZJ. 2000. A randomized controlled trial of the caries-preventive efficacy of a chlorhexidine-containing varnish in high-caries-risk adolescents. Caries Res 34:432–9
68. Lopez L, Berkowitz RJ, Zlotnik H, Moss M, and Weinstein P. 1999. Topical antimicrobial therapy in the prevention of early childhood caries. Pediatr Dent 21:9–11.
69. Lopez L, Berkowitz R, Spiekerman C, and Weinstein P. 2002. Topical antimicrobial therapy in the prevention of early childhood caries: a follow-up report. Pediatr Dent 24:204–6.
70. Lindley MG, Birch GG, and Khan R. 1976. Sweetness of sucrose and xylitol. Structural considerations. J Sci Food Agric 27:140–4.
71. Hayes C. 2001. The effect of noncariogenic sweeteners on the prevention of dental caries: a review of the evidence. J Dent Educ 65:1106–69.
72. Roberts MC, Riedy CA, Coldwell SE, Nagahama S, Judge K, Lam M, Kaakko T, Castillo JL, and Milgrom P. 2002. How xylitol-containing products affect cariogenic bacteria. J Am Dent Assoc 133:435–41
73. Trahan L. 1995. Xylitol: a review of its action on mutans streptococci and dental plaque—its clinical significance. Int Dent J 45:77–92.
74. Ly KA, Milgrom P, and Rothen M. 2006. Xylitol, sweeteners, and dental caries. Pediatr Dent 28:154–63
75. Söderling E, Isokangas P, Pienihäkkinen K, and Tenovuo J. 2000. Influence of maternal xylitol consumption on acquisition of mutans streptococci by infants. J Dent Res 79:882–7.
76. Söderling E, Isokangas P, Pienihakkinen K, Tenovuo J, and Alanen P. 2001. Influence of maternal xylitol consumption on mother-child transmission of mutans streptococci: 6-year follow-up. Caries Res 35:173–7.
77. Van Wuyckhuyse BC, Perinpanayagam HE, Bevacqua D, Raubertas RF, Billings RJ, Bowen WH, and Tabak LA. 1995. Association of free arginine and lysine

concentrations in human parotid saliva with caries experience. J Dent Res 74:686–90
78. Tabak LA. 2006. In defense of the oral cavity: the protective role of the salivary secretions. Pediatr Dent 28:110–17
79. Donly KJ and Brown DJ. 2005. Identify, protect, restore: emerging issues in approaching children's oral health. Gen Dent 53:106–10
80. Featherstone JDB. 2006. Caries prevention and reversal based on the caries balance. Pediatr Dent 28:128–32.
81. Beltran-Aguilar ED, Goldstein JW, and Lockwood SA. 2000. Fluoride varnishes: a review of their clinical use, cariostatic mechanism, efficacy and safety. J Am Dent Assoc 131:589–96.
82. Seppä L. 1988. Effects of a sodium fluoride solution and a varnish with different fluoride concentrations on enamel remineralization in vitro. Scand J Dent Res 96:304–9
83. Attin T, Hartman O, Hilgers RD, and Hellwig E. 1995. Fluoride retention of incipient enamel lesions after treatment with a calcium fluoride varnish in vivo. Arch Oral Biol 40:169–74
84. Ekstrand J, Koch G, and Petersson LG. 1980. Plasma fluoride concentration and urinary fluoride excretion in children following application of the fluoride-containing varnish Duraphat. Caries Res 14:185–9.
85. Ekstrand J, Koch G, and Petersson LG. 1983. Plasma fluoride concentration in pre-school children after ingestion of fluoride tablets and toothpaste. Caries Res 17:379–84.
86. Weyant RJ, Tracy SL, Anselmo TT, Beltrán-Aguilar ED, Donly KJ, Frese WA, Hujoel PP, Iafolla T, Kohn W, Kumar J, Levy SM, Tinanoff N, Wright JT, Zero D, Aravamudhan K, Frantsve- Hawley J, and Meyer DM. 2013. Topical fluoride for caries prevention: executive summary of the updated clinical recommendations and supporting systematic review. J Am Dent Assoc 144:1279–91.
87. Ripa LW. 1989. Review of the anticaries effectiveness of professionally applied and self-applied topical fluoride gels. J Public Health Dent 49(Special Issue):297–309.
88. Reynolds EC. 1998. Anticariogenic complexes of amorphous calcium phosphate stabilized by casein phosphopeptides: a review. Spec Care Dent 8:8–16.
89. Reynolds EC. 1997. Remineralization of enamel sub-surface lesions by casein phosphopeptide- stabilized calcium phosphate solutions. J Dent Res 76:1587–95
90. Shen P, Cai F, Nowicki A, Vincent J, and Reynolds EC. 2001. Remineralization of enamel subsurface lesions by sugar-free chewing gum containing casein phosphopeptide-amorphous calcium phosphate. J Dent Res 80:2066–70
91. Lijima Y, Cai F, Shen P, Walker G, Reynolds C, and Reynolds EC. 2004. Acid resistance of enamel subsurface lesions remineralized by a sugar-free chewing gum containing casein phosphopeptide-amorphous calcium phosphate. Caries Res 38:551–6
92. Morgan MV, Adams GG, Bailey DL, Tsao CE, and Reynolds EC. 2006. CPP-ACP gum slows progression and enhances regression of dental caries. J Dent Res 85(Special Issue B):2445
93. Walsh LJ. 2007. Clinical applications of Recaldent products: which ones to use where. Aust Dent Pract 18(3):144–6
94. Sitthisettapong T, Phantumvanit P, Huebner C and DeRouen T. 2012. Effect of CPP-ACP paste on dental caries in primary teeth: a randomized trial. J Dent Res 91:847–52

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