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The primary and mixed dentition, post-eruptive enamel maturation and dental caries: a review

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The mouth is in flux from the time the primary teeth begin to erupt, in the first year of life, through to the end of the 'mixed dentition' (i.e. the concurrent eruption of the permanent teeth and exfoliation of the primary teeth), at around 12 years of age. Primary teeth facilitate the development of the facial muscles and speech. They act as 'guides' for erupting permanent teeth. If lost prematurely, subsequent misalignment of permanent teeth can make them difficult to clean and possibly more caries-prone. During the mixed dentition phase, teeth are at relatively high risk of caries. Erupting teeth are difficult to clean and cleaning may be avoided because of tender gums and behavioural factors in children. Permanent enamel (and possibly primary enamel) undergoes post-eruptive maturation, accumulating fluoride, becoming harder, less porous and less caries-prone. Overall, primary teeth are more vulnerable to caries than permanent teeth. Widespread use of fluoride toothpaste has effected marked reductions in caries. Some evidence exists that fluoride delivered from toothpastes may be somewhat more effective in reducing caries in primary than in permanent teeth. However, caries remains a public health concern globally. New fluoride toothpaste formulations, optimised using in vivo fluoride delivery and efficacy studies, may improve the caries resistance of mineral deposited during post-eruptive maturation. Behaviour should not be ignored; new formulations will be more effective if used according to professionally endorsed recommendations based on sound science. Establishing good oral hygiene behaviour early in life can lead to lasting anti-caries benefits.

Key words: *Dental caries, dental public health*

INTRODUCTION

From the time the primary teeth begin to erupt, typically between 6 and 8 months of age, to the eruption of the second permanent molars, at around 12 years of age, the mouth, perhaps more than the rest of the body, is in an almost continuous state of flux. This period of change presents its own particular challenges with regard to the maintenance of healthy teeth. The aim of this paper is to review the changes that occur and the concomitant challenges in the context of dental caries. This is not an exhaustive review but covers salient differences between primary and permanent teeth, and the post-eruptive maturation of enamel. The possible roles of fluoride, metal ions and oral hygiene behaviour with regard to the anti-caries effectiveness of fluoride toothpastes are discussed.

ERUPTION

The crowns of the deciduous teeth begin to develop in the womb[1] and the mandibular central incisors typically begin to erupt into the mouth somewhere between 6 and 8 months of age. When the maxillary second molars erupt at around 29 months the eruption of the primary dentition is complete[2–5]. Both the timing and sequence of eruption differ between the maxilla and mandible. Between individuals, while the eruption sequence is usually the same, typical biological variation is seen in timing[6].

The eruption of the permanent dentition begins at around age 6 years, with the mandibular central incisors and first mandibular and maxillary molars, and is almost complete by around age 12 years with the eruption of the second molars. The mouth grows during this period and, as a result, the first permanent molars erupt behind the primary second molars, rather than displacing them. Before the eruption of the permanent teeth, sufficient room becomes available in the growing mouth for gaps to develop between the primary teeth to make space for the larger permanent teeth that will replace them. As the remaining permanent teeth erupt into the mouth, either displacing primary teeth or erupting behind the first permanent molar, the primary teeth are exfoliated, also starting at around age 6 years and finishing

at around age 12 years[2–5]. This period, when the mouth contains both primary and permanent teeth is often described as the 'mixed dentition'[7].

HEALTH OF DECIDUOUS TEETH AND CHALLENGES DURING THE MIXED DENTITION PHASE; IMPLICATIONS FOR PERMANENT TEETH

Primary teeth are sometimes thought of by parents as 'practice teeth'[8] or that caries in primary teeth can be ignored, as they will be shed. However, this is untrue and problems in the primary teeth can lead to problems with the permanent teeth. The primary teeth act as 'guides' for the permanent teeth, helping to ensure that they erupt in the correct position. If the primary teeth are lost prematurely, for example through decay, there is a chance that remaining primary and permanent teeth may drift, and that unerupted permanent teeth will erupt incorrectly positioned[9], leading to crooked teeth that may be difficult to clean and possibly, more likely to experience caries. Primary teeth influence the development and growth of the facial and jaw muscles, and this is important in aiding the development of speech[3,6]. Perhaps it is stating the obvious, but the ability to chew food is an important part of the digestive process and hence proper nutrition; teeth missing through caries may hinder this. Links have been established between malnutrition and early childhood caries[10], although it is not entirely clear whether this is attributable to caries *per se* or a combination of caries and associated factors such as poor diet.

Although not inherent to the teeth themselves, other factors to consider are salivary flow-rate and the chemical composition of children's saliva when compared with those of adults. Anderson *et al.*[11] reported that both stimulated flow-rate and calcium concentration in 6- to 12-year-olds were considerably lower than in 19- to 44-year-olds. Both of these phenomena can adversely influence caries susceptibility[12,13]. The authors used salivary calcium concentrations to calculate the 'critical pH' (i.e. the pH below which it will start to dissolve) for hydroxyapatite, a commonly-used enamel surrogate[14,15], in children's saliva. A higher value was reported for children's than for adult's saliva as a result of the lower calcium concentration. They concluded that for thermodynamic reasons alone, primary enamel is at greater risk of net demineralisation than permanent enamel. When enamel, rather then hydroxyapatite, is considered, and where primary enamel is more soluble than permanent enamel, for reasons discussed below, the magnitude of this difference may be even greater. Other authors also report differences in flow rate, for example in the minor salivary glands[16] although some suggest that any differences that might exist do not persist into the teenage years[17]. If age does play a part then it would be useful to know when, and to what extent, any such transition in the composition of children's saliva might occur.

POST-ERUPTIVE MATURATION OF ENAMEL

It has long been believed that the process of 'post-eruptive maturation' plays an important role in reducing caries vulnerability in the post-eruptive phase[18–23]. Intuitively, one might think that newly erupted enamel would be at its strongest, before being subjected to the challenges of caries and erosion. However, this is not the case. Clinical data show that the teeth are most vulnerable to caries within the first 2–4 years of eruption[24]. The first permanent molar is especially sensitive to this effect and is sometimes used in isolation as an indicator of the clinical effectiveness of fluoride toothpastes during caries clinical trials[25,26]. This vulnerability is less pronounced where the risk of caries has been reduced, for example through changes in dental health behaviour, but the trend remains essentially the same[27]. Post-eruptive maturation of enamel involves both chemical and physical changes of the outer layers of enamel, following exposure to the oral environment[28–33].

Chemically, considerable amounts of fluoride[29,34] are incorporated into the enamel surfaces, along with other metal ions associated with enamel solubility, such as zinc[35]. These elevated concentrations may be lost in later life as surface enamel is worn away[36–38], and *in vitro* studies support this proposition[38,39]. However, it is important to note that surfaces where caries is most likely to occur are protected from wear. Here, elevated concentrations of fluoride may persist into later life[37]. It has also been suggested that zinc may be important in the process of post-eruptive mineralisation and may reduce the susceptibility of teeth to caries, based on *in vivo* studies[40]. Naturally occurring chemical impurities in enamel which, when present, increase its solubility, are lost during maturation[41,42].

Physically, the surface is remodelled, becoming much less porous[30,43–46] and harder. Some researchers have reported that at least 10 years may be needed to reach maximum hardness[47,48], but that significant increases occur after 2–3 years, up to a depth of 330 μm. Although predominantly a surface phenomenon, with changes such as fluoride incorporation occurring to depths of maybe 50–100 μm, at least one change occurs at considerably greater depths. Driessens *et al.*[49] reported that the crystallinity of enamel increased with post-eruptive age to a depth of at least 1 mm. In general, late eruption, relative to average timing, seems not to have any substantial effect[50]. Time spent in the oral cavity seems to be the predominant factor in maturation. In terms of vulnerability to demineralisation, it seems that susceptibility decreases for at least

10 years following eruption, as with increases in hardness[51]. Calcium (Ca)/phosphorus (P) ratios during demineralisation and remineralisation also change over many years[52–54], although some changes are detectable after a few months[46].

While most data relate to newly-erupted permanent enamel, limited evidence also exists for maturation of primary enamel. Sabel *et al.*[55] reported post-eruptive changes in concentrations of trace elements in primary enamel. During a longitudinal study into caries-related events[56], early changes were more pronounced on the aproximal surfaces of newly-erupted permanent teeth than on adjacent primary enamel surfaces, that had spent many years in the mouth. Given that primary enamel is more vulnerable than permanent enamel, this suggests that primary enamel also undergoes maturation, as might be expected.

POST-ERUPTIVE MATURATION; POSSIBLE MECHANISMS

While phenomena associated with post-eruptive maturation have been well-characterised, there is no definitive explanation for the mechanism. One proposed explanation lies in the difficulty of cleaning partly-erupted teeth, and that cleaning may actually be avoided because of gingival tenderness, allowing a build-up of plaque at the gum margin[22,57]. Consequently, numerous demineralisation and remineralisation events may lead to lesions that are active but very superficial and subclinical in nature (i.e. pre-cavitated), with dissolution followed by subsequent mineral redeposition from calcium and phosphate in the oral fluids. Presumably, since hardness increases and porosity decreases during maturation, net remineralisation occurs, and this process would eventually take place over the entire surface of the crown. In the presence of fluoride, and metal ions such as zinc, incorporation of these species into the deposited mineral would be expected to occur. In fact, this does occur[35,36] and the mechanism proposed above might help explain an apparent contradiction. At near-neutral pH, the thermodynamic driving force for incorporation of fluoride into enamel by passive diffusion is insufficient to explain the incorporation of fluoride except in small amounts, very superficially (i.e. the first three layers at the atomic level)[58]. However, in reality, substantial amounts of fluoride are deposited. The effect of the numerous demineralisation and remineralisation events discussed above could explain this apparent anomaly, with substantial amounts of fluoridated mineral being deposited during periods of remineralisation, replacing mineral lost during demineralisation but without substantial net change in overall mineral content. The effect of pH during demineralisation on the nature of mineral deposited will likely be important in terms of its subsequent solubility, with low pH both accelerating the deposition of fluoridated apatites, even as the native enamel is dissolving, and maintaining enamel porosity to facilitate ingress of new mineral[59,60].

Data from early caries clinical trials lend support to the maturation proposition and particularly to the importance of fluoride. During one such trial, fluoride effected a greater caries reduction in teeth that erupted during the trial, than in those that were already present in the mouth at the start of the trial, but that had not previously been exposed to fluoride[61]. This finding, together with the affinity of newly-erupted enamel for fluoride when compared with mature enamel[62], supports the proposition that maturation reduces caries vulnerability and that fluoride may play an important role. With regard to possible fluoride mechanism, the potential importance of some limited enamel demineralisation and incorporation of fluoride is perhaps best illustrated by the findings from an *in situ* study where enamel specimens were exposed intraorally to a limited cariogenic challenge ('primed') and subsequently, to *ex situ* treatment with fluoride[63]. This regime conferred a substantial caries benefit during a subsequent, prolonged period of intraoral cariogenic challenge, with the primed enamel being more resistant than the surrounding unprimed enamel, despite having been demineralised to some extent during priming.

MATURATION, WHERE NEXT?

Overall, it is probably fair to say that most chemical data were reported before current consensus was reached on fluoride's predominantly topical mechanism of action. More recent reports tend to have focused on changes in enamel porosity during maturation as a means of evaluating the potential of caries diagnostic devices, rather than being studies into maturation *per se*[46,64,65]. Further understanding of the roles of fluoride and metal ions may facilitate the development of delivery systems to accelerate, and perhaps enhance, post-eruptive maturation. Given the (possibly) suboptimal salivary calcium concentrations and flow rates in children alluded to above[11], some means of elevating salivary calcium, particularly during acidic challenges and in the presence of fluoride[59,66], may also be beneficial.

DIFFERENCES BETWEEN PRIMARY AND PERMANENT TEETH

Superficially, while primary and permanent teeth are similar both structurally and chemically, there are several important differences between them. Some of these are apparent upon visual inspection; for example, there are fewer primary teeth than there are permanent teeth – 20 *versus* 32 respectively – and they

are smaller than permanent teeth, so that the full set of primary teeth can be accommodated in children's smaller mouths. The primary teeth have flatter contact surfaces and the crowns are more bulbous[2,4], hence the different surfaces are less well-defined. Overall, differences between the different types of primary teeth are less well-defined. Although the cusps are more pointed than in permanent teeth, their somewhat softer enamel is soon worn down, masking this difference. Primary teeth are also whiter than permanent teeth, the latter being the result of the more porous nature of primary enamel[67,68].

Physically, the enamel on primary teeth is thinner[2,69–71] and more permeable[72]. Overall, it is less dense than permanent enamel[73–75]. However, while it is generally held that primary enamel is softer and more easily worn[76–78] reports vary as to the magnitude of the difference in hardness[79]. With regard to density, the situation is complicated. Differences exist between types of tooth, for example molars and incisors, within individual teeth (e.g. occlusal and cervical sites) and in the density gradient from the tooth surface to the enamel–dentine junction[73,80–83]. In general, however, it is reasonable to say that differences in both hardness and density do exist but may be more subtle than is commonly suggested, especially near to the enamel–dentinal junction[73].

The pulp chamber in primary teeth is larger[2], relative to the rest of the tooth, when compared with the permanent teeth, and is therefore relatively close to the tooth surface. Microstructurally, the proportion of interprismatic enamel (the more soluble fraction in permanent enamel) is higher and the prism-junction density is higher than in permanent enamel[84]. Primary teeth have a less well-structured crystal arrangement; the prisms are smaller in primary than in permanent enamel[70], but conversely, the crystallites tend to be somewhat larger[85]. Overall, however, the arrangement of the crystallites and prisms is apparently similar[86]. While some authors report considerable amounts of 'prismless' enamel at the surface of primary enamel[86,87], others do not[88]. One consequence of some of these structural differences may be the much higher permeability of primary enamel[89].

Chemically, while both primary and permanent enamel are composed predominantly of calcium-deficient carbanato-hydroxyapatite[90], some differences do exist. Carbonate, the impurity in enamel that most increases its solubility, is present in primary enamel in greater amounts[91,92]. Primary enamel may be comparatively deficient in phosphate[90] and concentrations of trace elements differ significantly[93–95]. Some of these trace elements, for example strontium and zinc, are implicated in susceptibility to caries[96,97]. Fluoride concentrations tend to be higher in the outer layers of permanent enamel[98,99].

Although not the focus of this review, it is worth mentioning *in vitro* studies of erosion in primary and permanent teeth. Here, simple solubility can be studied without very relevant, but potentially confounding, variables such as plaque and lesion porosity. Reported data are mixed, with some showing primary enamel to be more vulnerable to erosion[77,100,101] and others showing little or no difference[76,102,103]. However, these studies used a wide range of erosive challenges, from the earliest stages of erosive demineralisation to fairly aggressive challenges, making comparison difficult. A further complication is the use of an acquired salivary pellicle, with its protective effect[104,105] in some studies[102] but not in others. In general, however, primary enamel seems to be more susceptible to more aggressive, or at least to cumulative, erosive challenges[106]. *In situ* data are scarce but support this proposition[107].

COMPARATIVE SUSCEPTIBILITY TO CARIES OF PRIMARY AND PERMANENT TEETH: CLINICAL AND LABORATORY DATA

In general, clinical data suggest that primary enamel is more prone to lesion formation and progression than is permanent enamel[57,108–113]. One reviewer, investigating the end of the 'caries decline' in Norway, concluded that whatever the possible explanation(s) for the reversal of the caries decline, it affected more 5-year-olds than it did 15-year-olds, suggesting that when the risk of caries increases, the primary teeth are more vulnerable[114]. The combination of faster lesion progression in primary enamel, its relative lack of thickness and the proximity of the relatively larger pulp chamber may be particularly insidious[113]. One noteworthy observation is that perhaps 30% of total experience of caries occurs in the primary teeth, despite their relatively short residence time in the mouth[115].

While well-designed *in situ* studies are probably the best preclinical indicator of anti-caries efficacy, data from studies where both primary and permanent enamel were used are scarce. Sønju-Clasen *et al.*[112] showed that in the absence of fluoride, primary enamel demineralised to a considerably greater extent than did permanent enamel. However, when a fluoride mouth-rinse was used, no difference was seen. Further, demineralisation in permanent enamel was about the same regardless of whether or not the fluoride rinse was used, and was relatively superficial in both cases, suggesting a modest cariogenic challenge, making assessment of any fluoride effect difficult to assess. The lack of fluoride effect in permanent enamel and modest cariogenic challenge suggest that this study may have simulated the clinical situation for low caries-risk individuals. However, caries risk is variable, both between and within individuals, and studies incorporating a range of cariogenic challenges,

for example by including a variable sucrose challenge[116], would be desirable. Further, a range of substrates, including sound enamel, and enamel lesions simulating different stages of caries, from early softening through to relatively advanced subsurface, should yield useful information on the relative susceptibilities of primary and permanent enamel.

Data from *in vitro* studies are both contradictory and difficult to compare. Many researchers have used one particular type of demineralising system (i.e. a fixed pH and degree of undersaturation at least at baseline), rather than a range of demineralising conditions representing the full 'spectrum' of cariogenic conditions found at the plaque–enamel interface. Presumably, this was to address a specific hypothesis, to allow interstudy comparisons or perhaps because the study authors simply had more experience with their particular system. These studies have generated useful data, but have also led to apparent contradictions. Sabel *et al.*[117] found a positive relationship between lesion depth and porosity in primary enamel but, unfortunately, a permanent enamel comparison group was not included in their study. Some authors have reported no difference between enamel types[118] whereas others have reported[109,119–121] or implied[122] substantial differences. While the acidic challenge used by Issa *et al.*[118] does not appear to have been low, the resulting lesions were relatively superficial nonetheless. Overall, the relationship seen in erosive lesions between differences in behaviour and strength of acid attack (i.e. that primary enamel is more soluble but that the difference is only revealed by a relatively aggressive acidic challenge) may also hold true for carious lesions.

Finally, a point worthy of note is that although remineralisation of lesions in primary enamel has been demonstrated in pH-cycling studies[123], there is a paucity of mechanistic data relating to remineralisation in primary enamel. Given the importance of remineralisation in the caries process in permanent teeth, the study of remineralisation of lesions in primary enamel warrants much more attention.

EFFECT OF FLUORIDE ON THE PRIMARY DENTITION

Broadly speaking, trends in primary teeth have mirrored those in permanent teeth, although some anomalies apparently exist. Caries incidence in the permanent dentition has decreased steadily following the widespread introduction of fluoride toothpastes. In the primary dentition, there appears to have been a similar decline, but less constant, and with a plateau in the 1990s[124,125]. As with permanent teeth, it is reasonable to attribute this decline (or these declines) predominantly to the widespread use of fluoride and particularly fluoride toothpastes.

Looking specifically at fluoride delivered from toothpastes, again there is a paucity of data relating to the primary dentition. A recent review[126] concluded that only a small number of caries clinical trials[26,127–131] were sufficiently well-designed to yield useful data. There are not, for example, sufficient data to confirm a dose–response relationship, as is the case for permanent enamel[132,133], although it is hard to imagine that a dose–response relationship does not exist. Lima *et al.*[131] reported that a 1100 ppm F toothpaste was more effective than a 500 ppm F toothpaste, but only in high-risk subjects (although this might well be expected). In their recent review, Dos Santos *et al.*[134] reported that 'standard' toothpastes (1000–1500 ppm F) were effective at all levels when compared with 'low F' (<600 ppm F) toothpastes, which were only effective at surface level. *In vitro* and *in situ* studies do tend to support the case for a fluoride dose–response[135–140], although whether the dynamic range and magnitude of response to fluoride are similar to those in permanent teeth cannot be discerned. While fluoride, when delivered from some sources such as water fluoridation and varnishes, may be somewhat less effective at reducing caries in the primary than in the permanent dentition, there is some limited evidence that when delivered from toothpaste, it may more effective[141–143]. One can only speculate on possible explanations but a much deeper understanding of the interaction of fluoride with the primary teeth is needed if its effectiveness is to be optimised.

CARIES DECLINE AT AN END?

Since their introduction over 50 years ago, the widespread use of fluoride-containing toothpastes is generally considered to have effected a marked reduction in caries incidence[144]. However, despite programmes to increase awareness of the role of brushing with fluoride toothpaste, together with diet modification and regular dental check-ups, caries remains a ubiquitous problem[145]. Further, recent reviews have concluded that the decline in caries may be at an end or even in reversal, with levels increasing in some cases[114,146]. Regardless, even in countries where the incidence of caries is relatively low, an unacceptable level of the disease persists[147]. There is a clear need for continued efforts to reduce caries.

TOWARDS MORE EFFECTIVE AND EFFICACIOUS FLUORIDE TOOTHPASTES

The development of more efficacious anti-caries toothpastes continues apace. Given that fluoride concentration in toothpastes sold for non-prescription use is limited by regulatory bodies in most markets to somewhere between

1,000 and 1,500 ppm[148,149], formulations are optimised for fluoride delivery. Alternatively, fluoride toothpastes with complementary agents, such as calcium-based remineralising agents, are a promising route to caries reductions[59].

However, it is not enough simply to make fluoride toothpastes available for use, no matter how efficacious they may be. The effect of behaviour during use is just as important and should not be ignored. A product with outstanding efficacy will not be effective in reducing caries unless it is used correctly, and hence delivers fluoride to the mouth efficiently. Incremental improvements in plaque removal through self-performed brushing are unlikely to deliver a substantial caries benefit[150], especially when compared with the benefits of fluoride toothpaste[151]. However, increased brushing frequency has been linked to reductions in caries during clinical trials[152] and the effect of establishing good brushing behaviour has been demonstrated during intervention studies[26,153]. Further, when behaviour is changed early in life, the caries benefits can continue for many years beyond the intervention period. This caries benefit, resulting from modified behaviour, was also seen in teeth that were unerupted during the intervention period and therefore cannot be attributed to, for example, enhanced fluoride incorporation during post-eruptive maturation during the intervention period[153].

One means of increasing duration and frequency of brushing may be through the design of toothpastes with improved sensory characteristics. There is evidence of a significant relationship of brushing for longer when toothpaste is rated highly for taste and consistency[154]. While conclusive clinical evidence is lacking, increased brushing time has been linked to increased enamel rehardening *in situ*[155] and more efficient fluoride delivery to saliva[156].

Finally, the relationship between incidence of caries in the primary dentition and the permanent dentition has been well-established over many years[157–165]. This relationship is most likely based on diet coupled with oral hygiene practises and highlights the importance of establishing these practises at an early age, to confer anti-caries benefits on the permanent teeth, to help them last a lifetime.

WHAT STUDIES ARE NEEDED TO FILL THE GAPS IN OUR KNOWLEDGE?

With regard to differences in the relative caries vulnerabilities of primary and permanent enamel, it is probably fair to say that while primary enamel is the more vulnerable, there is a lack of comprehensive understanding at all levels, from simple mechanistic studies through to caries clinical trials, particularly with regard to the effect of fluoride and possibly metal ions. For post-eruptive maturation, there is a considerable body of evidence describing what happens – at least in the permanent teeth – but it is not altogether clear why it happens. The cost and complexity of caries clinical trials is ever-increasing, and ethical considerations preclude the extended use of a non-fluoride placebo in most cases (correctly, in the author's opinion). Thus, well-designed *in situ* studies, supported by *in vitro* studies to elucidate mechanisms, are a promising means of gaining further understanding of differences between primary and permanent enamel, and of post-eruptive maturation. More comprehensive *in vitro* and *in situ* studies are needed. Lesion type, or enamel status (i.e. sound or partially demineralised) at baseline in particular can have a pronounced effect on subsequent behaviour *in situ*[166–169] and *in vitro*[170–173]. Even during a single cariogenic challenge, in any particular individual, neither pH nor concentrations of calcium and phosphate will remain constant[174]. Therefore, studies using sound enamel and a range of lesion types, representing the 'spectrum' of caries status, from early softening through to advanced subsurface lesions, in both net demineralising and remineralising environments, should be considered. These should lead to greater understanding of the differences between primary and permanent enamel, with the potential to affect their vulnerability to caries under a range of conditions.

SUMMARY

Between birth and the age of 12 years, the mouth is in flux, through the eruption of the primary teeth, post-eruptive maturation, and then the mixed dentition period. The latter phase is perhaps the most complex of all, with concurrent exfoliation of the primary dentition, eruption of the permanent dentition and the post-eruptive maturation of the permanent teeth. The permanent teeth are often at highest risk of caries during this period for at least two reasons. Erupting teeth are difficult to clean, as they are not yet fully aligned with their neighbours, and, in any event, cleaning may be avoided completely because of tender gums. In the months and years following eruption, primary and permanent enamel undergo post-eruptive maturation, which is thought to reduce vulnerability to caries. During post-eruptive maturation, the enamel surfaces accumulate fluoride and metal ions, become harder, less porous and hence less caries-prone.

Although the primary teeth remain in the mouth for but a short time, they are important for a number of reasons, including guiding the erupting permanent teeth and the development of speech. Premature loss can cause problems with the permanent dentition and rather than being regarded as practice teeth, they

should be cared for well. Problems resulting from lack of proper care can lead to problems in the permanent teeth, which ideally must last a lifetime. Primary teeth need specialised attention, perhaps more so than the permanent teeth in some respects. They are more vulnerable to caries because of differences in their chemical composition and their physical properties, including thinner, softer enamel that is more vulnerable to dissolution by cariogenic acids.

Caries is still a major public health concern in populations globally, despite the success of fluoride toothpastes in reducing its incidence. There is a need for greater understanding of the role of fluoride toothpastes in the post-eruptive maturation of all teeth and in caries reductions in primary teeth. The limited evidence available suggests that they may be somewhat more effective in the primary dentition, but more information is needed, for example with regard to a dose–response relationship. It is unlikely that a sufficient number of full-scale caries clinical trials will be conducted in the near future to answer these questions. Therefore, properly-designed *in situ* studies, supported by *in vitro* mechanistic studies, are needed. These should be designed to include the spectrum of the disease state in terms of both risk and lesion status, and should yield valuable information to guide the development of more effective fluoride toothpastes. However, the effect of behaviour should not be overlooked. Rather, it must be an integral part of strategies to make more efficacious fluoride toothpastes more effective in use. Good oral hygiene habits, especially if established at an early age, can translate into benefits in the permanent teeth that can last a lifetime if cared for properly.

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Conflict of interest

Author Lynch is employed by GlaxoSmithKline Consumer Healthcare.

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