

A literature review of dental erosion in children

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ABSTRACT

Dental erosion is increasingly recognized as a common condition in paediatric dentistry with complications of tooth sensitivity, altered aesthetics and loss of occlusal vertical dimension. The prevalence of erosion in children has been reported to range from 10% to over 80%. The primary dentition is thought to be more susceptible to erosion compared to the permanent dentition due to the thinner and less mineralized enamel. The aim of this paper was to critically review dental erosion in children with regards to its prevalence, aetiology, diagnosis and prevention. The associations between erosion and other common conditions in children such as caries and enamel hypoplasia are also discussed.

Keywords: Dental erosion, primary dentition, toothwear.

Abbreviation: GORD = gastro-oesophageal reflux disease.

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INTRODUCTION

Dental erosion, defined as the progressive, irreversible loss of dental hard tissues by a chemical process without bacterial involvement,¹ is currently considered a significant clinical challenge.² Even though erosion has been considered the major component of toothwear in children,³ it often co-exists with other forms of toothwear such as attrition (wear resulting from tooth to tooth grinding) and abrasion (wear resulting from tooth to other hard surfaces).⁴ As subjects with erosion in the primary dentition have increased risk of erosion in the permanent dentition,⁵ early diagnosis and prevention from an early age will help prevent damage to the permanent teeth. Erosion in children may be associated with many clinical problems such as dental hypersensitivity, altered occlusion, eating difficulties, poor aesthetics, pulp exposure and abscesses.^{6,7} The aim of this paper was to critically review the literature on dental erosion in children with regard to its aetiology, prevalence, associated clinical conditions and prevention.

Prevalence of dental erosion

As shown in Table 1, prevalence studies on erosion in children have reported a wide variation which is suggestive of the difficulty in finding a unified tooth-

wear index among researchers for measuring and detecting erosion lesions.⁸ The broad ranges in results are likely to be due to differences in the populations studied, as well as variations in consumption levels of acidic drinks.

Furthermore, the prevalence of erosion in children is likely to be affected by age as erosion lesions become easier to detect with increasing exposure of the teeth to acids. In the UK Children's Dental Health Survey (1993), the prevalence of erosion reaching dentine on the palatal surfaces of primary teeth was 8% and 24% in 2- and 5-year-old children respectively.⁹ In more recent studies (summarized in Table 1), the prevalence of erosion in the primary dentition ranged from 65% in 4–6-year-old UK children to 71% in 8–11-year-old German children.^{5,10} The first prevalence data on erosion in Australia was reported by Kazoullis *et al.*¹¹ who found that 78% of primary teeth in 5–15-year-old children showed erosion.

In the permanent dentition, the prevalence of erosion on the palatal surfaces was noted in 8% and 31% of 7- and 14-year-old UK children respectively.⁹ Investigations from other adolescent population groups have reported a general prevalence in the permanent dentition of around 10%⁵ to 90%¹² with Australian children having a prevalence of approximately 25%.¹¹ On the other hand, severe erosion has been noted in only 2% of adolescents.¹¹

Table 1. Prevalence of dental erosion in children reported in the past decade

Authors	Year	Country	No. of subjects	Age of subjects (yrs)	Prevalence of erosion	
Deery <i>et al.</i> ⁸¹	2000	UK	125	11–13	37%P	
Walker <i>et al.</i> ¹⁰	2000	UK	USA	129	11–13	41% P
			1726	4–6	65%pr	
			Total	7–10	61% P	
			11–14	52% P		
Al-Dlaigan <i>et al.</i> ⁸²	2001	UK	418	15–18	62% P	
				14	48% mild erosion	
					51% moderate erosion	
					1% severe erosion	
Ganss <i>et al.</i> ⁵	2001	Germany	1000	8–14	70.6% pr 11.6% P	
Ayers <i>et al.</i> ²⁰	2002	NZ	104	5–8	82% overall	
Al-Majed <i>et al.</i> ¹²	2002	Saudi Arabia	354	5–6	82% overall	
			862	12–14	95% overall	
Al-Malik <i>et al.</i> ¹⁷	2002	Saudi Arabia	987	2–5	31% overall	
Harding <i>et al.</i> ⁸	2003	Ireland	202	5	47% overall	
					21% erosion into dentine or pulp	
Dugmore <i>et al.</i> ⁶⁵	2004	UK	1753	12	59.7% P	
Bardsley <i>et al.</i> ¹⁹	2004	UK	2351	14	53% P	
Peres <i>et al.</i> ⁸³	2005	Brazil	499	12	13% P	
Luo <i>et al.</i> ⁷	2005	China	1949	3–5	5.7% overall	
Chadwick <i>et al.</i> ⁸⁴	2006	UK	10 381	5	53% pr	
			Total	8	About 10% P	
				12	About 30% P	
				15	About 30% P	
Wiegand <i>et al.</i> ¹⁶	2006	Germany	463	2–7	32% overall	
Kazoullis <i>et al.</i> ¹¹	2007	Australia	714	5.5–14.6	78% pr	
					25% P	
Mangueira <i>et al.</i> ⁸⁵	2009	Brazil	983	6–12	12.3% pr	
McGuire <i>et al.</i> ⁸⁶	2009	USA	1962		7.6% P	
						45.9% P

P: Permanent dentition.

pr: Primary dentition.

As many epidemiological studies employed indices to report on prevalence of erosion, it is interesting to review the more commonly used ones with respect to the scale, choice of key teeth and validity.^{13,14} A popular toothwear index used is the one proposed by Smith and Knight.¹⁵ In this index, four sections of each tooth (i.e., buccal/labial, palatal/lingual, cervical and incisal/occlusal) are examined visually and recorded separately, scoring each surface, ranging from 0 (no loss of enamel surface characteristics) to 4 (complete loss of enamel and pulp exposure). These values are then compared to a set of maximum acceptable toothwear scores that have been proposed for each decade of life. This allows the extent and distribution of the toothwear in patients to be measured. However, the maximum acceptable toothwear score places the 0 to 25-year age bracket in one group and then incrementally moves upwards with every age decade. This index is arguably more relevant for adults and the required details prove lengthy to record.⁶ An important aspect of this index is that it measures toothwear irrespective of aetiology and hence is not exclusively designed for diagnosis of erosion.¹⁶

A number of studies^{7–12,17} have utilized modified versions of the Toothwear Index proposed by Smith

and Knight,¹⁵ as with those employed in the UK Children's Dental Health Survey 1993.⁹ The toothwear score used ranges from 0 (no evidence of toothwear) to 3 (loss of enamel and dentine resulting in pulp exposure) and 9 being a score where assessment could not be made due to tooth either missing or having a large restoration. Such indices measured erosion only in the categories of loss of enamel, dentine or exposure of the pulp and were not sufficiently sensitive to follow-up subtle changes in tooth surface loss.¹⁶ Some studies^{7,18} have recorded wear only on certain surfaces, scoring the palatal and/or buccal surfaces for erosion and disregarding occlusal/incisal wear from further analysis in an attempt to separate attrition and abrasion from lesions caused primarily by acid erosion.^{8,19} It could be argued, however, that toothwear occurs as a result of the interaction between all three types of wear,²⁰ with erosion potentially affecting any tooth surface. This could explain some of the variations seen in results among the studies that have taken all surfaces into account for evaluation and analysis as opposed to those that have limited the number of assessed surfaces. Some studies have limited the examination of dental erosion to the incisors and first permanent molars.^{12–17,21} Such a system does not provide any information about the

distribution of the erosive lesions across the whole dentition.

An index for the clinical classification of erosive lesions has been proposed by Lussi.²² This index, which also is an extension of Smith and Knight index,¹⁵ separates the grading of erosion between the facial and the oral and occlusal surfaces. As erosion, attrition and abrasion are difficult to distinguish from one another in their initial stages, only the lesions that are considered to be definitely a result of an acidic challenge are classified above grade 0. For the facial surfaces, grade 1 is loss of enamel, grade 2 is involvement of dentine for less than one half of the tooth surface, and grade 3 is involvement of dentine for more than half of the tooth surface. On the other hand, the occlusal surfaces are graded as either grade 1 being slight erosion with no dentine involvement, and grade 2 as severe erosion with dentine involvement. Grade 0 is no erosion, including surfaces with a smooth silky appearance, and absence of developmental ridges being a possibility in either of the surfaces. However, this index fails to record the initial signs of dental erosion.

A classification of dental erosion has also been proposed by Aine *et al.*²³ and was utilized in their study on both primary and permanent teeth affected by gastro-oesophageal reflux disease (GORD). The erosion lesions are classified according to their clinical appearance, being graded from 0 (no erosion) to 3 (dentine exposure on occlusal surfaces or dentine affected on other surfaces). This index is limited in that neither site specificity of the lesion nor the extent of the involved surface is considered.⁶ Kazoulis *et al.*¹¹ have expanded on this index by recording the highest score given to a tooth surface to assign an erosion grade for each tooth. The erosion index was then calculated for each subject using the grade score for each tooth, divided by the total number of teeth scored, having assigned an arbitrary cut-off value of 1.06 or greater to signify severe erosion.

Numerous other indices have been proposed within the literature, including those utilized by Larsen *et al.*,²⁴ Wiegand *et al.*,¹⁶ Khan *et al.*²⁵ and Bartlett *et al.*²⁶ However, the search for the ideal index and accurate rates of prevalence of dental erosion continues. The ideal index to assess dental erosion would need to fulfil a number of criteria. Ideally, it should have definitive parameters to assess differing extents of dental erosion, easily separate erosive lesions from other defects of dental hard tissue, be able to monitor changes of severity over time and be concurrently easy to use, with good inter- and intra-examiner agreement.²²

Considerations which can potentially accentuate differences amongst prevalence rates include differences in age of participants, the sample population studied, as well as the differences in consumption of acidic beverages among groups. Furthermore, most of the

epidemiological studies have analysed erosion on specific teeth and do not provide information about the distribution and severity of the erosive lesions across the whole dentition.¹⁶

Clinical appearance of dental erosion

A spectrum of changes may be observed on the tooth surface which results from erosion. The early stage of erosive toothwear appears as a smooth, silky glazed surface.²⁷ The clinical manifestation can include loss of surface anatomy, increased incisal translucency, absence of enamel, and chipping of the incisal edges.²⁸ As erosion progresses, rounding of the cusps, grooves and incisal edges occurs,²⁷ progressing towards loss of occlusal morphology. On smooth surfaces it manifests as a concave loss of tooth surface, with a dished out, hard, smooth appearance,¹⁶ where the width of the lesion often exceeds its depth.²⁷ Common sites of erosion in primary teeth are the occlusal aspects of the molars and the incisal/palatal surfaces of the maxillary incisors (Figs 1 and 2).²⁹ Symmetrical erosive dentine exposures on the cuspal inclines of the molar teeth are commonly described as cup or bowl-shaped lesions. Khan *et al.*²⁵ found the mandibular first permanent molars to be a site prone to cup lesions and predictive of age of onset and severity of dental erosion.

One of the main clinical challenges regarding erosion is to correctly distinguish it from other forms of wear. Occlusally, erosion can be differentiated from attrition lesions as the latter often is flat, glossy and with distinct margins.²⁷ Additionally, in the case of attrition, the lesions are also evident on the antagonist teeth.²⁷ Cervical lesions rarely occur in children as the primary dentition is not present intraorally for sufficient time to allow for this process to take place. However, all the processes of attrition, erosion and abrasion have a



Fig 1. Clinical photograph showing marked dental erosion on the incisal and palatal surfaces of the maxillary central and lateral primary incisors. Loss of anatomical contour and rounding of enamel edges is evident. Thinning of enamel has resulted in a pink hue being evident, representing pulpal tissues close to the palatal aspect.



Fig 2. Marked dental erosion on maxillary left primary canine tooth on the incisal aspect, and dentine exposure on the buccal cuspal inclines of the first and second primary molar teeth.

compounding effect on the final clinical manifestation, with levels of salivary protection further influencing the outcome for the dentition. Enamel softened by the erosive process may be more susceptible to the effects of abrasion and attrition.⁶ Attrition of the incisal edges in the primary dentition is extremely difficult to distinguish from erosion in the later stages.¹⁶

Intrinsic sources of acid as causes of dental erosion

Gastric acid is hydrochloric acid produced by the parietal cells in the stomach³⁰ and has a pH of 1–1.5.³¹ Clinical manifestation of dental erosion occurs when teeth are exposed to acid over several months.³¹ Common causes for the presence of the gastric acid in the oral cavity include GORD, eating disorders, chronic vomiting, persistent regurgitation and rumination.^{4,30,31} Erosion resulting from gastric acids is often initially noted on the palatal surfaces of the maxillary incisors.³⁰ In more severe cases as erosion progresses, the palatal surfaces of the maxillary premolars and molars will become involved and eventually the erosion pattern becomes more widespread, involving the occlusal and facial surfaces of the teeth.³⁰

A recent systematic review of GORD in children has reported a pooled sample-size-weighted average prevalence of 23.4% in children with asthma and 3.8% in children without asthma.³² In spite of its common

occurrence, there is limited literature on the oral health of children with GORD. GORD is defined as the involuntary passage of gastric contents into the oesophagus.³³ In recent reviews, a strong association was shown between the prevalence of GORD and dental erosion.^{32,34} The reported prevalence of dental erosion among GORD children varies greatly and ranges from 24% (no control group was considered in this study),³⁵ 46% (compared with 40% in the control group),³⁶ 76% (compared with 24% in the control group)³⁷ and up to 87% (no control group was considered in this study).²³ The variation in the results may be attributed to differences in age, sample sizes used and the relative lengths of time the teeth were exposed to gastric acid.³⁷

According to Ersin *et al.*,³⁷ 76% of their 38 GORD subjects have dental erosion compared to 24% in the control group. The authors attributed the results to be directly related to reflux. Aine *et al.*²³ have also found similar results, reporting 87% of their 17 GORD subjects to have erosion lesions. In addition, they found that the signs of dental erosion among two children led to GORD diagnosis. Furthermore, the results showed that while diurnal reflux could be damaging, reflux during sleep caused the greatest loss of dental hard tissue, presumably due to the reduced amount of saliva during sleep. Aine *et al.*²³ thus suggested early diagnosis of silent GORD through dental erosion patterns noted on the patient's dentition. Linnett *et al.*³⁶ have suggested other oral modifying factors such as salivary flow rate and buffering capacity to be assessed in cases of dental erosion.

Extrinsic sources of acid as sources of dental erosion

Medicaments, lifestyle, diet, environmental and occupational factors are possible extrinsic sources of acid involved in the aetiology of dental erosion.³⁸ Prolonged and frequent use of acidic medications has the potential to cause erosive lesions in teeth.³⁹ The most direct mechanism is through the relative acidity of the medications which are often in the form of liquid and effervescent tablets for children. As the solubility of weak acids and bases is pH dependent, acidic preparations are often necessary for drug dispersion.⁴⁰ In addition, these acidic medications improve palatability and patient compliance. Nunn *et al.*⁴⁰ has assessed the pH of commonly prescribed drugs used long-term by children with renal disease, which included antibiotics, cardiovascular medications, gastrointestinal drugs and potassium supplements. All but two of the drugs tested were found to have a pH well below the critical pH of 5.5 for enamel demineralization, with the most erosive potential being that of two effervescent tablets due to their citric acid content.⁴⁰ Supplemental vitamin C tablets have very high levels of acidity and are known to cause erosion, especially when consumed frequently

and left in direct contact with teeth.^{38,39} In the past few years, use of supplemental vitamin C (L-ascorbic acid) has become very popular.³⁹ Al-Malik *et al.*¹⁸ have found that children who were given vitamin C supplements had up to 4.7 times greater risk of erosion. Results from other clinical surveys also report similar positive correlations between the consumption of vitamin C supplements and erosion.^{28,41}

Chronic use of chewable aspirin tablets, as may be used in patients with juvenile rheumatic arthritis, can also result in dental erosion. A number of case studies support this finding.^{42,43} Sullivan and Kramer⁴³ considered a group of 42 children with juvenile rheumatoid arthritis and found that the 25 children who were chewing their daily aspirin tablets showed dental erosion, whereas children who swallowed tablets without chewing exhibited no erosion. Medications may also be responsible for erosion through reducing the salivary flow rate and/or buffering capacity of saliva. The bicarbonate level in saliva is directly correlated with salivary flow rate, thus saliva produced at a low flow rate will have a lower pH and a lower buffering capacity.³⁸ As reported by Hellwig and Lussi,³⁹ medications that can lead to such effects include tranquilizers, antihistamines, anti-emetics and antiparkinsonian medicaments. As a result of the xerostomic effect of such drugs, some patients may also increase their consumption of carbonated drinks.²¹

A number of studies have looked at the association between erosion and medications used to treat asthma and have provided conflicting results.⁴⁴⁻⁴⁷ Asthmatic medications may cause erosion through the acidic nature of the aerosols on the teeth.⁴⁶ In addition, prolonged use of the beta-2 agonists in drugs such as salbutamol and terbutaline can lead to xerostomia, which indirectly contributes to erosion through reducing the modifying and protective effects of saliva.⁴⁶ Drugs used as bronchodilators have also been implicated through relaxing the smooth muscle and affecting the oesophageal sphincter, thus potentiating the effect of acids via the gastro-oesophageal reflux.⁴⁶ Furthermore, patients may increase their acidic drink consumption due to the increased dry mouth effect from the drugs.⁴⁶ However, to date many of the studies have not been able to show a direct association between asthma, asthmatic medications, their side effects and dental erosion, most likely due to the multifactorial cause of dental erosion.^{46,47}

Diet and lifestyle make up a large component of the extrinsic factors involved in dental erosion. Many population studies on children have shown a direct correlation between consumption of carbonated drinks, fruit juices and dental erosion in children,^{7,8,12,18,48} with excessive consumption of acidic drinks and food being reported as the most important extrinsic factor contributing to dental erosion.⁴⁹ The recent dramatic

increase in consumption of acidic fruit juices, fruit drinks and carbonated beverages is now thought to be the leading cause of dental erosion observed among children and adolescents.²⁹

Factors that can affect the erosive potential of acidic drinks are pH and buffering capacity. It is known that the greater the buffering capacity of the drink, the longer it will take for saliva to neutralize the acid.⁵⁰ Also, the physical and chemical properties of acidic foods and beverages can affect their erosive potential³⁸ through their effect on the clearance rate from the oral cavity. Previously, it was thought that the total acid level (titratable acid) of dietary substances was more important than the pH.³⁸ However, these hypotheses have now been refuted with research showing that the main chemical parameter likely to significantly correlate with erosion is the initial pH of the dietary intake.^{51,52} The type of acid used in the beverage may also affect erosive potential as those drinks containing acids with calcium-chelating properties, such as citrate, may cause erosion at higher pH levels.³⁸ The calcium, phosphate and fluoride contents of food and beverages can further explain the severity of erosive attack as they determine the degree of saturation with respect to tooth minerals,⁵⁰ whereby solutions that are under-saturated with respect to enamel tissue will dissolve it. Phosphoric acid, citric acid and sodium citrate are commonly found in carbonated and sports drinks.⁵² Both phosphoric acid and citric acid are triprotic acids that can release up to three hydrogen ions in solution, while phosphate and citrate can sequester calcium ions.⁵² Studies have shown that the majority of beverages sold from school canteens, as well as commercially-available baby drinks exhibit erosive potentials.^{52,53}

The clinical appearance of dental erosion will further vary among individuals depending on behavioural factors. These include the manner that the dietary acid is introduced into the oral cavity (e.g., using a straw, baby bottle, sipping, gulping), the frequency of exposure and duration of the erosive content being in contact with teeth.⁴⁹ The eating, drinking and swallowing habits that increase the direct contact time of acidic foods and drinks with the teeth have an obvious direct correlation with dental erosion.³⁸ On the other hand, the consumption of foods and drinks after or in conjunction with the erosive substance may help neutralize as well as clear the acidic agent from the oral cavity.⁴⁹

The timing of the erosive agent being introduced is of significance, in particular among children who go to bed with drinking bottles, as night-time exposure to acidic drinks has been suggested to be more destructive due to the much reduced salivary flow rate.⁴⁹ The consumption of sports drinks during or after strenuous activity, at times where the individual is dehydrated, also increases the erosive potential of the drink.⁵⁰

Susceptibility of primary teeth to erosion

Due to structural differences, primary teeth are more susceptible to the complications of erosion compared to permanent teeth. Dentine involvement as a consequence of erosion may occur more rapidly in the primary as opposed to permanent dentition due to the thinner enamel layer^{48,54} and morphological differences.⁵⁵ Also, in immature teeth with large pulps, erosion is more likely to lead to pulpal inflammation and exposures.⁵⁶ Johansson *et al.*⁴⁸ reported microhardness of enamel in primary teeth to be less than in permanent teeth. This is due to less mineralization and specifically, the enamel surface not being as mature as that of the permanent teeth, with a lower degree of crystallite arrangement.⁴⁸ Furthermore, primary enamel contains more water⁵⁷ and has increased permeability compared to permanent enamel.⁵⁸ This may further explain the relatively more rapid progression of erosion in the primary teeth.⁴⁸

The slower salivary sugar clearances and the lower salivary flow rates in children may contribute to increased susceptibility for erosion in children.⁴⁸ The buffering capacity of saliva is dependent on flow rates and is responsible for neutralizing and clearing acids that cause dental erosion.^{59,60}

Role of saliva and other biological factors in protection against erosion

Saliva protects the enamel from erosion through a number of mechanisms, including the formation of the pellicle.⁶⁰ The acquired pellicle is a biofilm, free of bacteria,² composed of calcium-binding proteins that occur in saliva.⁶¹ Young and Khan⁶⁰ have shown that the distribution pattern of erosion in adults is affected by variations of the thickness of the acquired salivary pellicle, with sites of greatest thickness (i.e., lingual aspect of mandibular permanent molars) having the lowest incidence of cervical erosion.

Of relevance are the distinct differences found in the chemical composition, rate of formation and ultra-structural appearance between the pellicle on primary and permanent teeth.⁶² It has been observed that the rate of formation of the pellicle is initially slower on primary enamel, with the adsorption process levelling out at a thickness corresponding to one-third of the pellicle on permanent enamel.⁶² Also, the differences in amino acid composition may be indicative of the presence of different types and amounts of proteins in the acquired pellicle on primary enamel compared with that on permanent enamel.⁶² These findings may be suggestive of primary teeth being more prone to erosion compared to permanent teeth.

The buffering capacity of saliva is another important factor involved in protection of the dentition from erosion through its ability to neutralize acids that cause

dental erosion.⁶⁰ The active constituents of saliva which provide buffering capability include phosphate and bicarbonate ions.⁶¹ The buffering capability of saliva may also play a role in preventing dental caries.^{61,63} The flow rate of saliva has also been implicated as an important host factor, providing protection against both erosion and caries.^{60,63} It is believed that after neutralizing and/or clearing the erosive agent, remineralization of some of the softened enamel will take place through salivary calcium, fluoride³⁸ and phosphate deposition.⁴⁹ Both the quantity and quality of saliva may explain the observed differences between the extent of dental erosion among individuals who are subjected to similar dietary challenges.³⁸

Other biological factors involved in protection against erosion include the anatomy of the teeth and soft tissues, the soft tissue movements of the tongue and buccal mucosa and swallowing patterns, all of which could affect the retention or clearance pattern of the erosive agent.⁴⁹

Association of erosion with dental caries

A number of recent research reports have paid particular attention to the possible association between dental erosion and caries.^{11,17,18,21} Both dental erosion and dental caries are multifactorial in nature. However, there are distinct differences between them. The acids responsible for the process of erosion are either intrinsic (gastric acid reflux), extrinsic or occupational and are not produced by the oral flora²¹ as opposed to caries which develops due to acids produced by the oral flora and their effects on the tooth surface.¹⁸ Another important difference between the two processes is site specificity, with dental erosion often being located in areas that are plaque-free as opposed to dental caries which is often located in sites of plaque accumulation.

Kazoullis *et al.*¹¹ carried out dental examinations on 714 children, recording dental erosion, presence of enamel defects as well as dental caries. The authors found dental erosion to be strongly associated with caries experience. Children who had caries present were more likely to also have severe dental erosion in their primary teeth as well as in their permanent dentition. Other studies have found similar associations.^{17,18,21} Al-Malik *et al.*¹⁸ investigated the possible association between dental erosion and caries in a sample of 987 children, using a cross-sectional study including dental examination and questionnaire survey. The results showed a direct relationship between dental erosion and dental caries, and that the dietary factors related to erosion may also have contributed to caries and/or rampant caries. The authors have speculated that ascorbic acid, which was used in the research, may not only have a direct effect on the enamel, but may be indirectly contributing to the process of rampant caries.

However, it is noted that only 54 of the subjects used in this study were given the vitamin C supplements.

Erosion and caries may share common risk factors.¹⁷ Most of the acidic food and drinks consumed by children that contribute to dental erosion also contain cariogenic sugars, thus being capable of inducing both processes in a proportion of cases.^{17,21,64} Individuals who fail to maintain a non-cariogenic diet may also fail to maintain a non-erosive diet.⁶⁵ This lack of dietary care may potentiate both erosive and cariogenic tooth tissue loss in the same patient. Caries experience has also been strongly correlated with presence of dental erosion as well as being a predictor for dental erosion.²¹ Al-Malik *et al.*¹⁷ carried out a study utilizing exfoliated or extracted teeth, which were assessed visually and histologically. The results of the study showed that both erosion and caries may occur in the same teeth and tooth surfaces in some cases. These results were confirmed in another survey, suggesting that the occurrence of erosion and caries can be concurrent and that caries may be a risk factor for erosion in some children.¹⁷ In some cases, the rapid and destructive nature of caries may superimpose on erosion, and remove clinical evidence of erosion.

Dental caries and erosion share similarity in that lack of saliva and its protective factors may make the oral cavity more susceptible to both of these conditions.^{11,60} Furthermore, both conditions being chronic processes, require time to develop. Histologically, when experimental erosion is induced *in vitro*, enamel sections show a complete loss of surface morphology and an irregular lesion surface.^{54,64} A subsurface zone of enamel was found in at least one study which showed a translucent appearance under polarized light, reminiscent of the translucent zone seen in early enamel caries.⁶⁴ However, this finding was not supported in a study by Al-Malik *et al.*⁵⁴ where no such zone could be detected histologically. Another interesting correlation between erosion and caries arises from the postulation that the acidic oral environment is likely to encourage the growth of acidophilic *Streptococcus mutans*, thus increasing a subject's susceptibility to caries.³⁷ A number of studies have supported such a postulation, with results showing higher numbers of *S. mutans* in children with erosion and GORD.^{36,37,66}

It is important to appreciate that dental caries and erosion can occur independently, so that associations between the two conditions may not always be found. For example, investigations such as those of Auad *et al.* reported no significant link between dental caries and erosion in their samples.⁶⁷⁻⁶⁹

Association of erosion with enamel hypoplasia

Enamel hypoplasia is the incomplete or defective development of enamel due to a disturbance of the

ameloblast cells during enamel formation.⁷⁰ The prevalence rates of developmental enamel defects in the primary dentition have been reported to vary considerably among studies, ranging from 4% to 60%.⁷¹

With the eruption of teeth, hypoplastic lesions are exposed to the factors responsible for erosion and dental caries.⁷² A recent study examining 714 children aged 5.5 to 14.6 years reported a strong relationship between presence of dental erosion and enamel hypoplasia in both the primary and permanent dentition.¹¹ It has been hypothesized that the altered mineralization associated with enamel hypoplasia and enamel defects may lead to a greater rate of dissolution by acids, therefore being a risk factor for dental erosion as well as resulting in secondary tooth structure loss through attrition and abrasion.¹¹ The affected teeth may also have an increased potential for becoming carious.⁷⁰⁻⁷²

Identification of risk factors

For appropriate preventive methods for erosion to be instigated, a confident diagnosis must have been reached. The relative contribution of intrinsic and/or extrinsic sources of acid need to be identified as causative factors. Biological factors such as saliva, acquired pellicle and the tooth structure as well as behavioural factors such as eating and drinking habits, level of hydration during exercise and oral hygiene should be assessed.⁷³ In order to establish the risk factors involved, a comprehensive six-day diet diary should be completed by the parent/guardian and reviewed by the clinician.⁷⁴ The medical history should consider any medication that may be acidic or may be reducing the salivary protection of the dentition. Patients should be questioned regarding history of reflux, frequency of vomiting and any GORD history should be further evaluated. Where intrinsic acids have been identified, it is prudent to ensure that the patient has been further evaluated by health professionals for the condition. Otherwise referral is appropriate for further assessment and management.²³

Prevention

Conventional methods of prevention against dental erosion include dietary analysis and restricting contact with erosive foods and drinks, education of the child and parent, and optimization of salivary protective mechanisms. However, the success of such methods heavily relies on patient acceptance and cooperation. As a result, research has geared towards investigating agents that can be applied to teeth directly to prevent dental erosion. Such agents include Tooth Mousse (GC Asia Pty Ltd, Japan),⁷⁵ Sensodyne Pronamel (GSK Consumer Healthcare, St George's Avenue, Weybridge, Surrey, UK) and fluoride with laser irradiation.⁷⁶ Tooth

Mousse contains a phosphopeptide that stabilizes amorphous calcium phosphate (CPP-ACP). Its effectiveness on reducing dental erosion caused by citric acid⁷⁷ and an acidic sports drink⁷⁸ has been shown. The effectiveness of Tooth Mousse may relate to the lubricating potential of its component ingredients such as glycerol, as well as its remineralizing potential.⁷⁵ Other agents which have potential for prevention of dental erosion contain high levels of bioavailable fluoride together with potassium nitrate.⁷⁷ The preventive effects of fluoride and its associated compounds have been attributed to the formation of precipitates on the tooth surface, which act as a protective barrier against acid impact.^{79,80} One major disadvantage to the fluoride layer is its dissolvability in acidic environments.⁷⁹ Laser irradiation of dental enamel in conjunction with fluoride application has been shown to result in significant reduction of the solubility of enamel mineral, thus increasing the protective effects of fluoride against erosion.⁷⁶

CONCLUSIONS

Dental erosion in the primary dentition is commonly encountered in children and may continue into the permanent dentition. Its multifactorial aetiology and the associations with other dental conditions such as enamel hypoplasia and caries add complexity to the diagnosis, prevention and management of these conditions. The high prevalence of dental erosion reported in children calls for further research into its prevention, such as the use of protective additives to alleviate the erosive effects of acidic foods and beverages.

REFERENCES

- Pindborg JJ. Pathology of dental hard tissues. Copenhagen: Munksgaard, 1970.
- Lussi A. Erosive tooth wear – a multifactorial condition of growing concern and increasing knowledge. *Monogr Oral Sci* 2006;20:1–8.
- Millward A, Shaw L, Smith A. Dental erosion in four-year-old children from differing socio-economic backgrounds. *ASDC J Dent Child* 1994;61:263–266.
- O'Sullivan E, Milosevic A. UK National Clinical Guidelines in Paediatric Dentistry: diagnosis, prevention and management of dental erosion. *Int J Paediatr Dent* 2008;18 Suppl 1:29–38.
- Ganss C, Klimek J, Giese K. Dental erosion in children and adolescents – a cross-sectional and longitudinal investigation using study models. *Community Dent Oral Epidemiol* 2001;29:264–271.
- Linnett V, Seow WK. Dental erosion in children: a literature review. *Pediatr Dent* 2001;23:37–43.
- Luo A, Zeng XJ, Du MQ, Bedi R. The prevalence of dental erosion in preschool children in China. *J Dent* 2005;33:115–121.
- Harding MA, Whelton H, O'Mullane DM, Cronin M. Dental erosion in 5-year-old Irish school children and associated factors: a pilot study. *Community Dent Health* 2003;20:165–170.
- O'Brien M. Children's dental health in the United Kingdom. London: HMSO, 1993.
- Walker A, Gregory JR, Bradnock G, Nunn J, White D. National Diet and Nutritional Survey: young people aged 4 to 18 years. London: HMSO, 2000.
- Kazoullis S, Seow WK, Holcombe T, Newman B, Ford D. Common dental conditions associated with dental erosion in schoolchildren in Australia. *Pediatr Dent* 2007;29:33–39.
- Al-Majed I, Maguire A, Murray JJ. Risk factors for dental erosion in 5–6 year old and 12–14 year old boys in Saudi Arabia. *Community Dent Oral Epidemiol* 2002;30:38–46.
- Berg-Beckhoff G, Kutschmann M, Bادهle D. Methodological considerations concerning the development of oral dental erosion indexes: literature survey, validity and reliability. *Clin Oral Investig* 2008;12 Suppl 1:S51–S58.
- Young A, Amaechi BT, Dugmore C, *et al.* Current erosion indices – flawed or valid? Summary. *Clin Oral Investig* 2008;12(Suppl 1):59–63.
- Smith BG, Knight JK. An index for measuring the wear of teeth. *Br Dent J* 1984;156:435–438.
- Wiegand A, Muller J, Werner C, Attin T. Prevalence of erosive toothwear and associated risk factors in 2–7 year old German kindergarten children. *Oral Dis* 2006;12:117–124.
- Al-Malik MI, Holt RD, Bedi R. Erosion, caries and rampant caries in preschool children in Jeddah, Saudi Arabia. *Community Dent Oral Epidemiol* 2002;30:16–23.
- Al-Malik MI, Holt RD, Bedi R. The relationship between erosion, caries and rampant caries and dietary habits in preschool children in Saudi Arabia. *Int J Paediatr Dent* 2001;11:430–439.
- Bardsley PF, Taylor S, Milosevic A. Epidemiological studies of tooth wear and dental erosion in 14-year-old children in North West England. Part 1: The relationship with water fluoridation and social deprivation. *Br Dent J* 2004;197:413–416.
- Ayers KM, Drummond BK, Thomson WM, Kieser JA. Risk indicators for tooth wear in New Zealand school children. *Int Dent J* 2002;52:41–46.
- Dugmore CR, Rock WP. A multifactorial analysis of factors associated with dental erosion. *Br Dent J* 2004;196:283–286.
- Lussi A. Dental erosion. Clinical diagnosis and case history taking. *Eur J Oral Sci* 1996;104:191–198.
- Aine L, Baer M, Maki M. Dental erosion caused by gastroesophageal reflux disease in children. *ASDC J Dent Child* 1993;60:210–214.
- Larsen IB, Westergaard J, Stoltze K, Larsen AI, Gynzelberg F, Holmstrup P. A clinical index for evaluating and monitoring dental erosion. *Community Dent Oral Epidemiol* 2000;28:211–217.
- Khan F, Young WG, Law V, Priest J, Daley TJ. Cupped lesions of early onset dental erosion in young southeast Queensland adults. *Aust Dent J* 2001;46:100–107.
- Bartlett D, Ganss C, Lussi A. Basic Erosive Wear Examination (BEWE): a new scoring system for scientific and clinical needs. *Clin Oral Investig* 2008;12(Suppl 1):65–68.
- Ganss C, Lussi A. Diagnosis of erosive tooth wear. *Monogr Oral Sci* 2006;20:32–43.
- O'Sullivan EA, Curzon ME. A comparison of acidic dietary factors in children with and without dental erosion. *ASDC J Dent Child* 2000;67:186–192.
- Lussi A, Jaeggi T. Dental erosion in children. *Monogr Oral Sci* 2006;20:140–151.
- Bartlett D. Intrinsic causes of erosion. *Monogr Oral Sci* 2006;20:119–139.
- Scheutzel P. Etiology of dental erosion – intrinsic factors. *Eur J Oral Sci* 1996;104:178–190.

32. Tolia V, Vandenplas Y. Systematic review: the extra-oesophageal symptoms of gastro-oesophageal reflux disease in children. *Aliment Pharmacol Ther* 2009;29:258–272.
33. Davies AE, Sandhu BK. Diagnosis and treatment of gastro-oesophageal reflux. *Arch Dis Child* 1995;73:82–86.
34. Pace F, Pallotta S, Tonini M, Vakil N, Bianchi Porro G. Systematic review: gastro-oesophageal reflux disease and dental lesions. *Aliment Pharmacol Ther* 2008;27:1179–1186.
35. Meurman JH, Toskala J, Nuutinen M, Klemetti E. Oral and dental manifestations in gastroesophageal reflux disease. *Oral Surg Oral Med Oral Pathol* 1994;78:583–589.
36. Linnett V, Seow WK, Connor F, Shepherd R. Oral health of children with gastroesophageal reflux disease: a controlled study. *Aust Dent J* 2002;47:156–162.
37. Ersin NK, Oncag O, Tumgor G, *et al.* Oral and dental manifestations of gastroesophageal reflux disease in children: a preliminary study. *Pediatr Dent* 2006;28:279–284.
38. Zero DT. Etiology of dental erosion: extrinsic factors. *Eur J Oral Sci* 1996;104:162–177.
39. Hellwig E, Lussi A. Oral hygiene products and acidic medicines. *Monogr Oral Sci* 2006;20:112–118.
40. Nunn JH, Ng SK, Sharkey I, Coulthard M. The dental implications of chronic use of acidic medicines in medically compromised children. *Pharm World Sci* 2001;23:118–119.
41. Al-Dlaigan YH, Shaw L, Smith A. Dental erosion in a group of British 14-year-old school children. Part II. Influence of dietary intake. *Br Dent J* 2001;190:258–261.
42. Grace EG, Sarlani E, Kaplan S. Tooth erosion caused by chewing aspirin. *J Am Dent Assoc* 2004;135:911–914.
43. Sullivan RE, Kramer WS. Iatrogenic erosion of teeth. *ASDC J Dent Child* 1983;56:192–196.
44. McDerra EJ, Pollard MA, Curzon ME. The dental status of asthmatic British school children. *Pediatr Dent* 1998;20:281–287.
45. Shaw L, Al-Dlaigan YH, Smith A. Childhood asthma and dental erosion. *ASDC J Dent Child* 2000;67:102–106.
46. Dugmore CR, Rock WP. Asthma and tooth erosion: is there an association? *Int J Paediatr Dent* 2003;13:417–424.
47. Sivasithamparam K, Young WG, Jirattanasopa V, *et al.* Dental erosion in asthma – a case-control study from south east Queensland. *Aust Dent J* 2002;47:298–303.
48. Johansson AK, Sorvari R, Birkhed D, Meurman JH. Dental erosion in deciduous teeth—an in vivo and in vitro study. *J Dent* 2001;29:333–340.
49. Lussi A, Jaeggi T, Zero D. The role of diet in the aetiology of dental erosion. *Caries Res* 2004;38 Suppl 1:34–44.
50. Lussi A, Jaeggi T. Chemical factors. *Monogr Oral Sci* 2006;20:77–87.
51. Seow WK, Thong KM. Erosive effects of common beverages on extracted premolar teeth. *Aust Dent J* 2005;50:173–178.
52. Cochrane NJ, Cai F, Yuan Y, Reynolds EC. Erosive potential of beverages sold in Australian schools. *Aust Dent J* 2009;54:238–244.
53. Hunter L, Patel S, Rees J. The in vitro erosive potential of a range of baby drinks. *Int J Paediatr Dent* 2009;19:325–329.
54. Al-Malik MI, Holt RD, Bedi R, Speight PM. Investigation of an index to measure tooth wear in primary teeth. *J Dent* 2001;29:103–107.
55. Hunter ML, West NX, Hughes JA, Newcombe RG, Addy M. Erosion of deciduous and permanent dental hard tissues in the oral environment. *J Dent* 2000;28:257–263.
56. Harley K. Tooth wear in the child and the youth. *Br Dent J* 1999;186:492–496.
57. Bonte E, Deschamps N, Goldberg M, Vernios V. Quantification of free water in human dental enamel. *J Dent Res* 1988;67:880–882.
58. Linden LA, Bjorkman S, Hattab F. The diffusion in vitro of fluoride and chlorhexidine in the enamel of human deciduous and permanent teeth. *Arch Oral Biol* 1986;31:33–37.
59. Crossner CG, Hase JC, Birkhed D. Oral sugar clearance in children compared with adults. *Caries Res* 1991;25:201–206.
60. Young WG, Khan F. Sites of dental erosion are saliva-dependent. *J Oral Rehabil* 2002;29:35–43.
61. Ten Cate AR. Oral histology – development, structure and function. USA: Mosby, 1994.
62. Sonju Clasen AB, Hanning M, Skjorland K, Sonju T. Analytical and ultrastructural studies of pellicle on primary teeth. *Acta Odontol Scand* 1997;55:339–343.
63. Leone CW, Oppenheim FG. Physical and chemical aspects of saliva as indicator of risk for dental caries in humans. *J Dent Educ* 2001;65:1054–1062.
64. Smith AJ, Shaw L. Baby fruit juices and tooth erosion. *Br Dent J* 1987;162:65–67.
65. Dugmore CR, Rock WP. The prevalence of tooth erosion in 12-year-old children. *Br Dent J* 2004;196:279–282.
66. O’Sullivan EA, Curzon ME. Salivary factors affecting dental erosion in children. *Caries Res* 2000;34:82–87.
67. Auad SM, Waterhouse PJ, Nunn JH, Moynihan PJ. Dental caries and its association with sociodemographics, erosion, and diet in schoolchildren from southeast Brazil. *Pediatr Dent* 2009;31:229–235.
68. Truin GJ, van Rijkom HM, Mulder J, van’t Hof MA. Caries trends 1996–2002 among 6- and 12-year-old children and erosive wear prevalence among 12-year-old children in the Hague. *Caries Res* 2005;39:2–8.
69. Kunzel W, Cruz MS, Fischer T. Dental erosion in Cuban children associated with excessive consumption of oranges. *Eur J Oral Sci* 2000;108:104–109.
70. Silberman SL, Trubman A, Duncan WK, Meydrech EF. A simplified hypoplasia index. *J Public Health Dent* 1990;50:282–284.
71. Seow WK. Enamel hypoplasia in the primary dentition: a review. *ASDC J Dent Child* 1991;58:441–452.
72. Boston DW, Al-bargi H, Bogert M. Abrasion, erosion, and abfraction combined with linear enamel hypoplasia: a case report. *Quintessence Int* 1999;30:683–687.
73. Lussi A, Jaeggi T. Erosion – diagnosis and risk factors. *Clin Oral Investig* 2008;12 Suppl 1:S5–S13.
74. Young WG. Tooth wear: diet analysis and advice. *Int Dent J* 2005;55:68–72.
75. Ranjitkar S, Rodriguez JM, Kaidonis JA, Richards LC, Townsend GC, Bartlett DW. The effect of casein phosphopeptide-amorphous calcium phosphate on erosive enamel and dentine wear by toothbrush abrasion. *J Dent* 2009;37:250–254.
76. Moslemi M, Fekrazad R, Tadayon N, Ghorbani M, Torabzadeh H, Shadkar MM. Effects of Er,Cr:YSGG laser irradiation and fluoride treatment on acid resistance of the enamel. *Pediatr Dent* 2009;31:409–413.
77. Rees J, Loyn T, Chadwick B. Pronamel and tooth mousse: an initial assessment of erosion prevention in vitro. *J Dent* 2007;35:355–357.
78. Ramalingam L, Messer LB, Reynolds EC. Adding casein phosphopeptide-amorphous calcium phosphate to sports drinks to eliminate in vitro erosion. *Pediatr Dent* 2005;27:61–67.
79. Schlueter N, Hardt M, Lussi A, Engelmann F, Klimek J, Ganss C. Tin-containing fluoride solutions as anti-erosive agents in enamel: an in vitro tin-uptake, tissue-loss, and scanning electron micrograph study. *Eur J Oral Sci* 2009;117:427–434.
80. Wiegand A, Attin T. Influence of fluoride on the prevention of erosive lesions – a review. *Oral Health Prev Dent* 2003;1:245–253.
81. Deery C, Wagner ML, Longbottom C, Simon R, Nugent ZJ. The prevalence of dental erosion in the United States and the United

- Kingdom on a sample of adolescents. *Pediatr Dent* 2000;22:505–510.
82. Al-Dlaigan YH, Shaw L, Smith A. Dental erosion in a group of British 14-year-old school children. Part I: Prevalence and influence of differing socioeconomic backgrounds. *Br Dent J* 2001;190:145–149.
83. Peres KG, Armenio MF, Peres MA, Traebert J, De Lacerda JT. Dental erosion in 12-year-old schoolchildren: a cross sectional study in southern Brazil. *Int J Paediatr Dent* 2005;15:249–255.
84. Chadwick BL, White DA, Morris AJ, Evans D, Pitts NB. Non-carious tooth conditions in children in the UK, 2003. *Br Dent J* 2006;200:379–384.
85. Manguiera DF, Sampaio FC, Oliveira AF. Association between socioeconomic factors and dental erosion in Brazilian schoolchildren. *J Public Health Dent* 2009;69:254–259.
86. McGuire J, Szabo A, Jackson S, Bradley TG, Okunseri C. Erosive tooth wear among children in the United States: relationship to race/ethnicity and obesity. *Int J Paediatr Dent* 2009;19:91–98. Erratum in: *Int J Paediatr Dent* 2009;19:222.

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