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Meta-analysis of the prevalence of tooth wear in primary dentition

ABSTRACT

Aim To conduct a meta-analysis of all the studies published in literature over the past three decades on the prevalence of dental erosion in preschool children.

Materials and methods The Pubmed data base revealed only one systematic review on the prevalence of tooth wear in children up to 5 years old. The search included works published from January 1982 to September 2012, using the following combinations of keywords: 1) "dental erosion" AND "children"; 2) "dental erosion in primary dentition"; 3) "dental" AND "attrition" AND "prevalence". The inclusion criteria for papers on tooth wear were the deciduous dentition observed only on the palatal and buccal sides with the distinction of erosion, attrition and abrasion. We took into consideration only randomized control trials. We excluded articles not written in English, case reports, historical and forensic studies, in vitro and in vivo studies. In case of doubt and/or when an abstract was not available, the full text copy of the article was examined. The first search on Pubmed revealed 29 articles, the same found in the study of Kreulen [2010], however we selected only multicentric studies focused on children of age below 5 years old, in which only the primary dentition (D) and only anterior teeth (incisors) were considered.

Results Both forest plot and scatter plot showed the prevalence of dental erosion in primary dentition, and that older children had a more severe dental erosion.

Conclusion Dental erosion should be considered a paediatric dentistry pathological entity as well as dental caries, and it can be related to more severe systemic diseases such as Gastroesophageal reflux disease. In addition, taking care of these little patients is important because they might suffer hypersensitivity, and also pulpal pathology caused by the typical structure of

deciduous teeth, where the pulp cavity is wide and close to the dentine and the enamel.

Keywords Enamel dental erosion; Gastroesophageal reflux disease; Primary dentition.

Introduction

Dental erosion is defined as irreversible loss of dental hard tissue by a chemical process that does not involve bacteria. Dissolution of the mineralised tooth structure occurs upon contact with intrinsic or extrinsic acids in the oral cavity. This form of tooth loss is part of a larger picture of tooth wear, which also consists of attrition, abrasion, and possibly abfraction [Gandara and Truelove, 1999]. The term dental erosion (DE) is used to describe the physical results of a pathologic, chronic, localized loss of dental hard tissue that is chemically removed from the tooth surface by acid and/or chelation without bacterial involvement. The acids responsible for erosion are not products of the intraoral flora; they stem from dietary, occupational or intrinsic sources [Ten Cate and Imfeld, 1996]. Tooth erosion is a multifactorial condition and the causative factors may be divided into extrinsic and intrinsic types, appropriately represented by the diagram in Figure 1 [Drugmore and Rock, 2004]. Since the first report on tooth erosion published in 1892 by Darby, many definitions and classifications have been used in the dental literature to describe erosive lesions of the teeth [Darby, 1892], due to the fact that different authors chose different approaches.

Thus, nomenclature and classification were based on: 1) aetiology: extrinsic, intrinsic and idiopathic erosion; 2) clinical severity [Eccles, 1982]; 3) activity of progression; 4) localisation of erosion [Imfeld, 1996].

This lack of harmonisation among tools and diagnostic indices led the organisation of a workshop of experts in 2007: the outcome was a new scoring system based on common experience and knowledge, the Basic

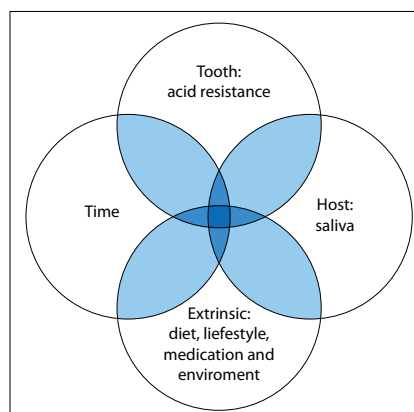


FIG. 1 Multifactorial aetiology of dental erosion: the overlapping factors may all be required to some extent to produce severe erosion.

Erosive Wear Examination (BEWE) scores for diagnosis of erosive lesions. Scoring varies from 0, which corresponds to no surface loss to 3, where there are distinct defects, and hard tissue loss >50% of the surface area. This index is a simple partial scoring system that guides the practitioner in case management, and makes it easier to assess lesions on all teeth [Bartlett et al. 2009]. Differently from abrasion, in which a mechanic action causes the loss of dental surfaces, in the case of DE enamel demineralisation is caused by chemical substances. In this situation, the enamel becomes weaker even when occlusal/masticatory forces are normal. The aetiology of DE can be found in extrinsic and intrinsic causes. Acids in the mouth originate from three main sources: acidogenic bacteria, ingested extrinsic and dislocated intrinsic acids through the backflow of gastric contents. Clearance of acids from the oral cavity depends to a large extent on the salivary flow rate and the saliva buffering capacity. A low salivary flow rate and poor buffering capacity allow prolonged retention of extrinsic and intrinsic acids in the mouth, which accelerate the erosive process.

The extrinsic factors involved in DE are: environmental, diet, medications, and lifestyle, though environmental and lifestyle cannot be taken into consideration in the case of children because of the lack of reliable data available; however, based on the volume of published material on the subject, the role of diet in the aetiology of DE has received great attention, while the role of medications is less extensively explored. The recent increased consumption of fruit juices, and carbonated beverages is still the main factor causing DE in children and teenagers [Lussi and Jaeggi, 2004]. Even if the total acid level (titratable acid) of dietary substances is considered more important than their pH, the erosive potential of a substance is not exclusively dependent on pH value and type of acid, but is also strongly influenced by its titratable acidity (the buffering capacity), calcium-chelation properties, mineral content and adhesion to the dental surface. Acids such as citric acid have a double action and may be very damaging to the tooth surface; besides the effect of hydrogen ions, acid anions (citrate) may form a complex with calcium, reducing the supersaturation of saliva and dissolving the crystal surface [Serra et al., 2009]. The intrinsic gastric acid is a hydrochloric acid produced by the parietal cells in the stomach and has a pH of 1–1.5. Clinical manifestation of DE occurs when teeth are exposed to acid over several months. Common causes for the presence of the gastric acid in the oral cavity include gastroesophageal reflux disease (GERD), eating disorders, chronic vomiting, and persistent regurgitation and rumination. In spite of its common occurrence, there is limited literature on the oral health of children with GERD. The first modern description of DEs associated with GERD was made by Howden in a case report [Howden, 1971]. In recent years, GERD has been described as an important aggravating factor of DEs and DE is now considered a comorbid syndrome with an established aepidemiological

association with GERD. Thus, it is not surprising that some authors have advocated that the examination of the oral cavity, in search for “atypical” DEs, should be an integral part of the physical examination of the patient with suspected GERD. On the other hand, other authors have denied, at least in children, that DEs may represent a relevant problem in GERD patients [Pace et al, 2008]. The dental enamel is a highly mineralised tissue of ectodermal origin that lacks any metabolic activity once formed. The characteristic macroscopic features of erosive lesions include a polished appearance, cupping of incisal edges and cusps and, enamel loss affecting the labial surfaces. The loss of tooth surface is disproportionate to the age of the subject. There are no studies published on the progression of erosion in deciduous teeth. These teeth often have aprismatic enamel on their surface, and their chemical composition differs somewhat from that of permanent teeth. When examined by electron microscopy, these lesions appeared as deep porosities and showed destruction both in the oral and facial surfaces. 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 and morphological differences. Also, in immature teeth with large pulps, erosion is more likely to lead to pulpal inflammation and exposures. Johansson et al. [2001] reported the microhardness of enamel in primary teeth to be less than that found in permanent teeth. This is due to the lower degree of mineralisation and specifically, the enamel surface not being as mature as that of permanent teeth, with a different crystallite arrangement. Furthermore, primary enamel contains more water and has a higher permeability compared to permanent enamel. This may further explain the relatively more rapid progression of erosion in primary teeth. Saliva has been considered the most important biological factor influencing DE. Ion effect of salivary calcium and phosphate protects the mouth from erosive agents, thanks to neutralisation and buffering of acids which slow down the rate of enamel dissolution. Erosion may be associated with low salivary flow and low buffering capacity. Saliva also plays a role in forming the acquired pellicle, which may act as a diffusion barrier or a perm-selective membrane, avoiding direct contact between acids and the tooth surface, protecting it against erosion [Serra et al., 2009]. It is believed that after neutralisation and/or clearing of the erosive agent, remineralisation of some of the softened enamel will take place through salivary calcium, fluoride and phosphate deposition. Of relevance are the distinct differences found in the chemical composition, rate of formation and ultrastructural appearance between the pellicle on primary and permanent teeth. 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. Also, the differences in amino acid composition may be indicative of the presence of

different types and amounts of proteins in the acquired pellicle of primary enamel compared with that of permanent enamel. These findings may be suggestive of primary teeth being more prone to erosion compared to permanent teeth [Taji and Seow, 2010].

Aim of the work

The aim of the work was a meta-analysis of the literature on the prevalence of DE/tooth wear in children with primary dentition and the effects of DE.

Materials and methods

The Pubmed Library revealed only one systematic review on the prevalence of tooth wear in children up to 10 years old. The search considered articles published in the last three decades, from January 1982 to September 2012, using the following combinations of keywords: 1) "dental erosion" AND "children"; 2) "dental erosion in primary dentition"; 3) "dental" AND "attrition" AND "prevalence". The type of review was accomplished following the PRISMA (Preferred Reporting Items for Systematic Review and Meta-Analyses) statement (Fig. 2). The inclusion criteria for papers on tooth wear were the deciduous dentition observed only on the palatal and buccal sides with the distinction of erosion, attrition and abrasion. We took in consideration only randomised controlled trials (RCT). We excluded articles not written in English, even if Italian, German, Spanish and French articles were analysed, case reports, historical and forensic studies, *in vitro* and *in vivo* studies. We also excluded those studies that did not use a clinical tooth wear index, did not show specific age groups, did report on wear related to a specific age or not reporting wear on objectively. General study characteristics and study quality parameters were recorded. The full text of the articles was examined. The first search on Pubmed data base revealed 50 articles, from which 29 papers were identified but only three articles met the inclusion criteria and were recruited for the critical examination: 1) Al Malik, 2002; 2) Jones and Nunn, 1995, and; 3) Luo et al., 2004. The articles were selected for inclusion and analysis if the following criteria were met: human studies; deciduous dentition; studies designed as prospective studies; only MRC (Multicentric Randomized Cluster); use of the same parameters to assess the prevalence of dental erosion; accuracy in statistical analysis, with the type of statistical analysis mentioned and sufficient data for statistical calculations.

Results

Fifteen studies out of the Pubmed research were deemed of good quality but only three of them were considered of medium-high quality (Table 1). Retrospective designs without inclusion of consecutively

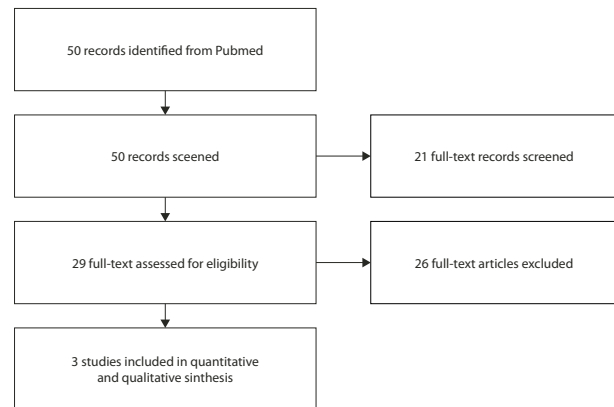


FIG. 2 PRISMA diagram for study design.

Author	Age (y)	%DE-	%DE+	N° of children
Al-Malik	3	72%	28%	131
	4	71%	29%	459
	5	65%	35%	415
Jones and Nunn	3	72%	28%	135
Luo et al.	3	95%	5%	373
	4	95%	5%	801
	5	94%	6%	775
Total	4			3071

DE- = no erosion, DE+ = any erosion

TABLE 1 Prevalence of dental erosion.

treated patients, and inadequate sample size mostly compromised the methodological quality of the retrieved studies. Data deriving from selected articles were collected in order to describe the prevalence of dental erosion in primary dentition (Fig. 3, 4).

Discussion

The significant relationship of extensive tooth wear with age in the deciduous dentition, but not in the permanent dentition, suggests that the deciduous teeth are less wear-resistant than permanent teeth, which confirms laboratory findings [Correr et al., 2007]. The main methodological difference between the studies was the tooth wear index used. Nine more or less distinct tooth wear indices were applied in 29 studies out of the 50 taken into consideration at the beginning of this study, but the common denominator was dentin exposure. We still cannot identify a specific factor influencing the prevalence of wear in these young age groups, neither can we explain the role of diet in the development of severe wear on the basis of the present data as we can also see in the article written by Kreulen et al. [2009]. Only the UK National Diet and Nutrition Survey (NDNS) of preschool children showed a relationship between the consumption of carbonated soft drinks and erosion, and

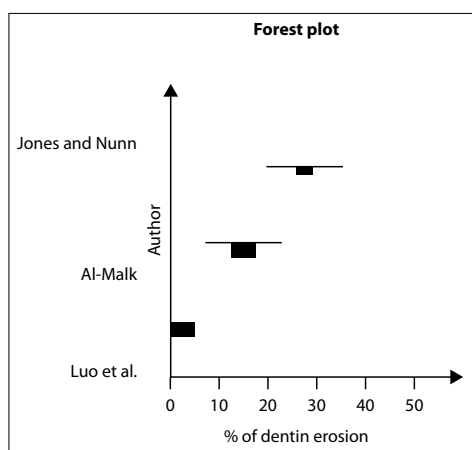


FIG. 3 Forest plot showing the prevalence of deciduous dentin exposure per study (ordered by year of publication, 1995-2002-2005). The area of the square reflects the size of the study based on the square root of N (Sample size). The horizontal line corresponds to the recalculated 95% confidence interval of each data point.

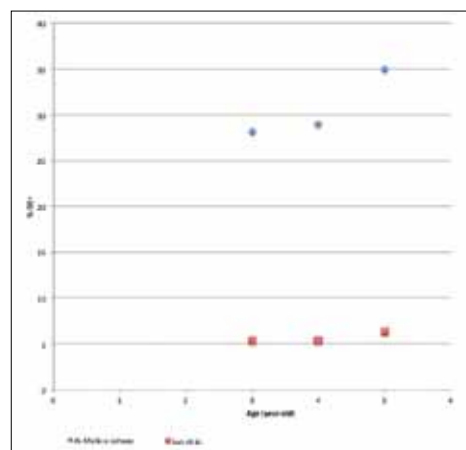


FIG. 4 Scatter plot of the adjusted prevalence of dentin exposure related to age as found in the included studies.

the NDNS of young people reported that the age-related increase of severity of DE was greater in children with the highest consumption of acidic food and drinks. The sample was drawn from 2-5 year-old children attending kindergarten schools in three different countries (China, Saudi Arabia and UK). The children were dentally examined in schools with the same procedures. Excluded from the study eligibility phase of the research were articles which considered children older than 5 years, with permanent dentition, even if just represented by the first molars, and in which a randomized cluster was not selected, but a convenience sample.

The Chi-squared test was used to underline the difference between Luo's and Al-Malik and Jones' records. The hypothesis was that Chinese children have the same prevalence of DE found in UK and SA (χ^2 3 years old=67.8; χ^2 4 years old=153.8 and χ^2 5 years old=181.8 with DF=1 and $P<0.001$ per $\chi^2>10.83$). The English and the Arabic studies have similar results about prevalence of DE, whereas in China, the sample size reveals a very low prevalence of the condition. We may suggest that different education, lifestyle and dietary habits in the Chinese population might protect deciduous teeth from DE, but there are not randomized control trials to support it. Of relevance is that in the permanent dentition, the prevalence of erosion was noted in 8% and 31% of 7 and 14-year-old UK children and in 46% of children aged 13-19 years, respectively [Mcquire et al., 2009]. These percentages show how DE affects adolescents more than children and that even if the permanent dentition has a different kind of enamel and salivary biofilm, the lesions increase irreversibly as in deciduous teeth, but slower.

Conclusion

Even if the data obtained are not as extensive as we expected at the beginning of the study, the results of this systematic review indicate that the prevalence of tooth wear into the dentin of deciduous teeth in children increases linearly with age. DE of the primary dentition is commonly encountered in children and may continue into

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 DE 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 [Taji and Seow, 2010]. Thus, children may have hypersensitivity but also pulpal lesions, if they do not undergo a strict follow-up. Other studies are required to obtain reliable data.

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