Prevention of erosive tooth wear: targeting nutritional and patient-related risks factors

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Key points

- Highlights the importance of early diagnosis of erosive tooth wear.
- Explores the risks factors involved in the aetiology of erosive tooth wear, both related to nutrition and to the patients.
- Describes preventive measures for patients at risk of erosive tooth wear.

This article provides an overview of the nutritional and patient-related risk factors involved in the aetiology of erosive tooth wear (ETW) and the preventive strategies to counteract them. The first step is to diagnose clinical signs of ETW and to recognise causal factors. Low pH and high buffer capacity of foods/drinks are the major risk factors, while the calcium concentration is the main protective factor. Reduction of frequency of consumption and contact time of erosive foods/drinks with the teeth, use of straws appropriately positioned and consumption of dairy products are advisable. Oral hygiene has a role in the development of ETW, however, postponing toothbrushing is not clinically advisable. In cases of drug abuse, chronic alcoholism, GERD or bulimia, the patient must be referred to a doctor. Immediately after vomiting, patients might be advised to rinse the mouth. Saliva has an important protective role and patients with reduced salivary flow can benefit from the use of chewing gum. Recent studies have focused on improving the protective capacity of the acquired pellicle as well as on the role of protease inhibitors on dentine erosion. However, the degree of evidence for these preventive measures is low. Clinical trials are necessary before these measures can be recommended.

Introduction

While dental erosion includes only the cases of softening and surface loss caused by extended exposure to acids only (chemical process), erosive tooth wear (ETW) is a chemical-mechanical process that results in a cumulative loss of dental hard tissue.[1] Despite the fact that its ultimate causative factors are non-bacterial acids and mechanical abrasive forces, ETW is a multifactorial condition and the progression of the lesions are driven by a complex interplay between nutritional and patient-related factors.[2] Appropriate preventive management of ETW aims at reducing or stopping the progression of the lesions. This can only be effectively achieved if the early clinical signs are diagnosed and appropriate preventive measures to fight the risk factors are installed in due time.[3]

This review presents an overview of the current literature regarding the risk factors involved in the aetiology of ETW and the preventive strategies that must be adopted to counteract their effect.

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Nutritional factors

The increased prevalence of ETW over the years has often been attributed to the nutritional factors such as regular consumption of fruit juices and soft drinks.[4] A systematic review showed that the frequent consumption of soft drinks, fruit juices and acidic candies significantly increases the prevalence of tooth erosion (OR values of 1.61, 1.20 and 2.24, respectively) among 8–19-year-old children and adolescents living in different countries.[5] Soft drink is of special relevance as the increase of its consumption (200% among Brazilian households) was significant from the 1980s to the 2000s.[6]

Potential erosive foods and drinks contain one or more types of weak acids in their composition, which are responsible for their low pH and high buffer capacity. Furthermore, the concentrations of soluble calcium and phosphate are usually low, which makes them undersaturated in respect to tooth apatite. The degree of saturation determines the driving force for tooth dissolution. Supersaturated solutions do not induce demineralisation, while undersaturated solutions in respect to apatite provoke surface demineralisation.[7] Some acids may have chelating property, such as citric acid, however, chelating properties are mostly important for high pH solutions, which may be considered clinically irrelevant when nutritional factors are considered.[8]

There are other physical factors related to the occurrence of tooth erosion that will not be discussed here: 1) viscosity, for example, may modulate ion exchange and clearance of the dissolved products;[9] 2) high temperature values increase the dissolution rate;[10] and 3) the motion may increase the dissipation of ions on the tooth surface.[11] The physical motion is

related to patients' drinking habits and will be further discussed in this chapter.

Acid type, buffering capacity and undissociated acid

The most common acids found in foods and drinks are phosphoric, citric, lactic, tartaric, acetic, ascorbic and maleic acids. Hannig *et al.*[12] compared different acids under standardised pH values (2, 2.3, 3) in a short-erosive challenge (5 min). At pH 2.0, lactic acid presented the highest erosive potential; while at pH 3, acetic acid was the most erosive acid. For all pH values, phosphoric acid was shown to have the lowest erosive potential. Beyer *et al.*[13] also compared the erosive potential of different acids. They showed that maleic, tartaric and citric acids were the least erosive acids, while phosphoric, ascorbic and lactic acids were the most erosive ones. However, in the latter study the acids were compared under different concentrations and pH values. Therefore, it is very difficult to isolate only one chemical factor when predicting the erosive potential of different acids.

The acids present in food and drink are considered weak, which means that they have the capacity of releasing protons as the tooth dissolves, favouring the tooth dissolution over time. For example, citric acid contains three groups that can release hydrogen ions. Therefore, it can be dissociated in three steps (pKa_1 3.13, pKa_2 4.74 and pKa_3 6.42). Furthermore, the undissociated acid is uncharged and can easily diffuse into the tissue and suffer dissolution in the near-surface enamel, which can further contribute to their erosive potential.[7]

In other words, these weak acids have high buffer capacity at a specific pH and they can keep tooth dissolution during long periods of contact, as for example when children consume soft drinks using nursing bottles. The effect of buffer capacity seems to be pH dependent.[14] When the solution pH is equal to the acid pKa, the acid has the highest buffer capacity, which means that it will be more resistant to the neutralising effect of saliva. The most common method to know the buffer capacity is by measuring the titrable acidity (mmol OH^-/l to reach pH 5.5). However, the values are usually difficult to translate to practice.

Lussi and Carvalho[15] analysed the erosive potential of 30 substances (drinks, candies, and medicaments) on deciduous enamel. They showed that pH, titrable acidity and calcium concentration play a significant role in initial erosion of deciduous enamel. Shellis *et al.*[16] suggest that undissociated acid concentration rather than buffering per se is a major rate-controlling factor, due to the importance of undissociated acid as a readily diffusible source of H^+ ions in maintaining near-surface dissolution within the softened layer of enamel.

pH

One of the most significant factors in the erosion aetiology is the pH value of the solutions that are in direct contact with the teeth.[7] It is important to keep in mind that there is no critical pH value for erosion as there is for caries (critical pH around 5.5), which is attributed to the variable composition of the food and drink (especially in respect to the calcium and phosphate content).[2] The critical pH value depends on the tooth tissue solubility (enamel or dentine) and on the concentrations of calcium and phosphate in the solution surrounding the tooth. Yogurt is a good example of food that has low pH (around 4.0), but it is not erosive because it has high calcium concentration.

Lussi and Carvalho[2] showed that the critical pH values ranged from 3.9 to 6.5 depending on the analysed food or drink. When the pH value of the solution is lower than 3.9, it is highly erosive regardless of its calcium and phosphate concentration. For example, Coca-Cola has a pH value of 2.5, but its critical pH is 5.1, which means that it is very erosive (pH <critical pH). On the other hand, yoghurt has a pH value of 3.9 and its critical pH is also 3.9 (pH = critical pH), which means that yoghurt does not cause any mineral alteration.[2]

Shellis *et al.*[17] showed that the dissolution rate increased as the pH decreased, with the most marked effect on enamel. In respect to dentine, there is a need for further studies. Furthermore, the concentration of acid significantly affected the enamel dissolution rate at pH 2.45 and pH 3.2, but not at pH 3.9, and it did not significantly affect the dissolution rates of dentine at any pH.

Calcium, phosphate and fluoride concentration

The erosion potential of a solution can be calculated based on its degree of saturation in respect to tooth apatite, which is determined not only by the pH, but also by the calcium and phosphate concentration. On the other hand, the fluoride content is employed for the calculation of the degree of saturation with respect to fluorapatite, however, its relevant effect in the prevention of erosion is negligible.[18] The positive effect of fluoride is related to its topical application by using professional and home-care products in a high frequency.[19]

Based on this knowledge, several *in vitro* and *in situ* studies have been performed to test the effect of the supplementation of calcium and/or phosphate on the erosive potential of different beverages. Calcium supplementation in the form of calcium glycerophosphate (soft drink) or calcium lactate combined with sodium linear polyphosphate (orange juice) has shown to reduce enamel erosion.[20,21] Furthermore, the combination of calcium and phosphate also reduced the dentine erosion development *in vitro*.[21] CPC-ACP (0.2%) and NanoHA (0.25%) supplementation also have shown to reduce enamel erosion when added to beverages.[22–24]

Generally, calcium has been shown to be more effective as a supplement than phosphate,[25] which may be due to its higher proportion in apatite as well as the type of phosphate ion available in the solution. The effect of ion supplementation is dependent on the type of salt, the concentration, the pH value and the type of acid/solution. Our research group has shown that 1 mM calcium and 1 mM phosphate were unable to reduce enamel erosion by 1% citric acid,[26] but they could reduce enamel erosion by a soft drink.[27] Attin *et al.*[28] also showed reduced enamel erosion by modification of a soft drink with 1 mM calcium. Contrary to most studies that have focused on the modification of beverages, Jensdottir *et al.*[29] tested Ca-modified candies in dry mouth patients. They showed that Ca-modified candies are able to make saliva less undersaturated with respect to apatite than the control candies.

On the other hand, the ion supplementation can cause alterations in the taste that may be unacceptable to the consumers and in the solution stability, which can alter the expiration date of the product. Furthermore, there is a lack of information about eventual systemic effects of the supplementation. Some positive results have been found in laboratorial studies, but, to date, no clinical data is available to support the modification of food or drink.

Patient-related factors

Eating/drinking habits

ETW is a multifactorial condition in which the high frequency of dietary acids consumption plays an important role, increasing its risk of development.[5] However, patients may be unaware of their acid intake and case history taking is not always effective to determine

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eating and drinking habits.[30] Therefore, the first step regarding preventive measures for this condition is to identify the frequency, amount and time of the day when erosive products are consumed using dietary and behaviour records over four days, including weekdays and weekends.[3,30] A previous study revealed that four or more dietary acid intakes per day in patients with other risk factors for erosion resulted in a higher risk of ETW.[31] To achieve appropriate prevention, the reduction of frequency of consumption and contact time of potentially harmful food and drink with the teeth are desirable. Patients must avoid the retention of dietary acids in the mouth before swallowing, not swishing them around the teeth or sipping over an extended period.[3] The use of straws positioned toward the back of the mouth is also recommended when drinking, to ensure that the flow does not make contact with the tooth surfaces.[32] On the other hand, clinical evidence shows that erosive lesions can rapidly affect incisors when there is excessive intake of acidic drinks using straws positioned into the labial vestibule.[33] Finally, the intake of dairy products at the end of meals and at night might be introduced into eating habits.[30]

Lifestyle

Vegetarian and vegan diets can be part of certain types of lifestyles and are associated to various health benefits, such as reduction in body weight, lower incidence of the metabolic syndrome, diabetes and cardiovascular diseases.[34,35] Other reasons for using these diets are animal rights and animal welfare, or social and environmental concerns related to sustainability.[36,37] A vegetarian diet does not include meat (including fowl), seafood and products containing these foods, but can incorporate dairy products (lacto-vegetarianism) or eggs (ovo-vegetarianism).[38] In vegan diets, besides meat, seafood and their products, the consumption of all types of flesh, dairy, and egg foods and sometimes even honey is also avoided.[38] The literature shows few studies regarding the prevalence of erosion in individuals with vegetarian or vegan diets and the results are contradictory.[39–45] Most studies have methodological limitations due to their small sample size. In general, no association was found between consumption of erosive foods and drinks with the prevalence of erosive tooth wear in vegetarian or vegan persons.[40,42,45] On the other hand, other studies found that a vegetarian diet increases the risk for ETW development.[43,44] The recommended preventive measures for these groups may focus on the way they ingest acidic foods, managing acid intake and exposure. Therefore, it seems advisable to avoid sipping, holding or swishing acidic drinks in the mouth and to avoid acidic foods last thing at night.[30] In addition, the use of high-concentration, acidic formulations and polyvalent fluoride sources should be stimulated.[46] In the case of lacto-vegetarianism, it is also recommended to finish meals with dairy products.[30]

There are also unhealthy lifestyles such as use of illicit drugs and alcohol abuse, which are often speculated to increase the risk for erosion. However, only few studies deal with these issues and they have weak evidence with some potential bias.[47] Anyway, there seems to be a tendency of higher risk of ETW in individuals with chronic alcoholism.[47] For these patients, the most important recommendation is to treat the alcohol and illicit drug use disorders.

Oral hygiene

The main form of abrasion in the oral cavity is toothbrushing, consisting of three-body wear, in which the toothbrush and the teeth are moving surfaces separated by an intervening slurry of dentifrice, with abrasive particles.[48] Normal toothbrushing habits with toothpastes in accordance with the International Organisation for Standardisation (ISO) standard will benefit oral health with minimal harmful effects on enamel and dentine.[49] In addition, toothbrushing without dentifrice has a minor impact on dental tissues.[50–52] However, under, over or abusive toothbrushing, when combined with erosion, may cause significant wear.[49] Erosive demineralisation of enamel results in surface softening, which accounts for the increased susceptibility to mechanical wear.[53,54] In dentine, there are some studies showing that the superficial layer of demineralised collagenous matrix is resistant to clinically relevant toothbrushing forces.[55,56] There are indications that the impact of toothbrushing on eroded dentine is less pronounced compared to eroded enamel, because the exposed collagen might act as a barrier against mechanical impacts.[56] However, *in vivo*, the demineralised organic matrix may be degraded by proteolytic enzymes, increasing the effect of abrasion.[48]

Several *in vitro* and *in situ* studies have shown that increasing waiting periods before brushing after erosive challenges probably enhances the abrasion resistance of enamel, due to the remineralisation of the surface.[53,57–59] However, this topic is a matter of debate, because recently it has been suggested that proper remineralisation does not occur in erosive lesions.[2] Scanning electron microscopy observation showed an amorphous mineral deposition on top of enamel prisms, after two hours of *in vitro* remineralisation.[60] This is not an ideal form of mineralisation, since the regrowth of the partly dissolved crystal is desired.[8] In none of the referred studies did eroded enamel present similar characteristics compared to uneroded ones, after treatment with saliva.[53,57–59] Therefore, a clinically significant remineralising by saliva effect might not have been reached. Accordingly, another study showed that even waiting periods of two or four hours presented no protective effect against enamel erosion.[61] In addition, an epidemiological study conducted in seven European countries with 3,187 young adults showed signs of moderate to severe ETW in approximately 29% of the patients and the delay of toothbrushing did not significantly affect the degree of ETW.[62] This is in-line with a recent case-control study showing that toothbrushing within ten minutes of acid intake was not associated with ETW after adjustment for dietary factors.[63]

Alternatively, a study showed that patients suffering from severe erosion could benefit from brushing their teeth immediately before rather than after an erosive attack, to avoid abrasion of softened tissue. It should be noted, however, that the interval between the erosive challenges was four hours.[64] Indeed, this measure is not a consensus since brushing before erosion with fluoride-free toothpaste diminished enamel loss only by 12% when compared to brushing immediately after erosion.

Postponing brushing after an erosive attack seems not to be a useful preventive measure.[2] It is important to bear in mind that active ingredients present in the dentifrices, such as fluoride, can counteract the mechanical deleterious effect, reducing the progression of ETW.[46]

Reflux

Before describing the measures to prevent erosion in individuals with reflux it is important to understand this condition. Gastroesophageal reflux corresponds to brief episodes of oesophageal peristalsis followed by saliva effect aimed to remove and neutralise gastric contents entering the oesophagus. These episodes can be a result of reduced lower oesophageal sphincter pressure and change in oesophageal motility, reduced salivary flow rate or increase in abdominal pressure.[65] Gastroesophageal reflux is a physiological phenomenon, different from the gastroesophageal reflux disease

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(GERD), in which the gastric contents entering the oesophagus promote pH fall below four during a certain time.[66] GERD can be symptomatic (heartburn, regurgitation, belching, chronic cough, chronic hoarseness, globus and dysphagia) with oesophageal mucosa breaks; silent, when mucosa breaks without symptoms is observed; and non-erosive reflux disease that shows typical symptoms with no mucosal alteration.[67] In patients with GERD, erosion is more common and more severe compared to healthy controls.[47] In an epidemiological study from seven European countries, including the UK, with 3,187 young adults, reflux symptoms were identified as a risk factor in the development of ETW.[62] In a middle-aged Finnish cohort, daily symptoms of GERD (OR 3.8; CI 1.2–12.0) was included among the strongest risk indicators for severe erosive wear.[68]

When a dental professional suspects the presence of GERD, it is important to refer the patient to a gastroenterologist, who will manage and treat the disease by lifestyle modifications and non-pharmaceutical therapies, medication or surgery.[65] This is particularly important, since it was observed that significant oligosymptomatic GERD occurs in the majority of patients with ETW.[69] Lifestyle modification includes avoiding reflux-provoking foods (such as wine, citric, acid, vinegar, fatty foods, tomato, peppermint, coffee, black tea, carbonated drinks, chocolate) and meals before bedtime, decreasing alcohol consumption, stimulating weight loss and the use of chewing gum.[30,65] Chewing gum is able to increase salivary flow rate and swallowing frequency, improving the saliva's protective effect against dental erosion and also promoting the clearance of gastric acids in the oesophagus.[53,70]

Vomiting

Bulimic individuals show binge eating and self-induced vomiting episodes (more than twice a week) to avoid body weight gain.[65] During vomiting, the intrinsic acid present in the stomach, which is highly erosive, makes contact with the tooth surfaces. However, vomiting can be only considered a risk factor for developing erosion when it is frequent over an extended period of time.[3,47] Recently, it was reported that patients with eating disorders had more risk of erosion (OR = 12.4), which is increased when vomiting is self-induced (OR = 19.6).[71] Dental professionals are often the first ones to identify and diagnose bulimia by detecting ETW and they must refer the patient to psychological or psychiatric treatment for a permanent reduction of the exposure to intrinsic acids. In addition, immediately after vomiting patients might be advised to avoid toothbrushing but instead to rinse the mouth with water, milk, sodium bicarbonate solution or preferably fluoride containing mouth rinse.[30]

Vomiting can also occur during the first trimester of pregnancy, therefore in this period the women might follow the recommendations presented above to diminish the risk of ETW.

Medication

The frequent or long-term use of medications with low pH and high titratable acidity in the form of chewable tablets or effervescent drinks is potentially erosive (for example, preparations containing acetylic salicylic acid or vitamin C). In addition, some medications can indirectly enhance the risk of dental erosion development from other erosive agents by reducing the salivary flow rate and/or buffer capacity of the saliva (for example, tranquilisers, antihistaminics, antiemetics and antiparkinsonian medicaments).[3,47,72] Li *et al.*[73] conducted a meta-analysis study to review dietary factors associated with dental erosion and found an association between vitamin C consumption and erosion, with an odds ratio of 1.16. The association between asthma medication in the form of inhalation aerosols and dental erosion presents contradictory results.[72] Dugmore and Rock[74] in a random sample of 1,753 twelve-year-old children observed 16.8% of asthma prevalence and no association was found between asthma and tooth erosion. The authors reported that the majority of medicaments prescribed for the treatment of asthma were not potentially erosive and that these medicaments would not be able to spread to the labial surfaces of anterior teeth after inhalation. On the other hand, Al-Dlaigan *et al.*[75] compared the prevalence of dental erosion in children with asthma in relation to those presenting significant tooth erosion but with no history of asthma, and with healthy children. The results showed that children with asthma had a higher prevalence of erosion than the control group. In addition to conflicting results, the association between medication intake and the occurrence of dental erosion shows weak evidence with some potential bias, because there are few data from valid controlled studies and most available results come from *in vitro* studies and case reports.[47] Nevertheless, health professionals need to know the erosive potential of liquid oral medicines, chewable tablets and effervescent vitamins with prolonged use, and must recommend a safe intake form without direct contact with the teeth whenever this option exists.[72]

Occupation

Some types of work expose individuals to acids, potentially increasing the risk of dental erosion development. Sulphuric acid is frequently found in battery, galvanising and plating factories. The risk for ETW and the severity of erosion increases with increasing concentration of the acid or the acidic fumes, increasing exposure time and duration of employment and shortening the distance between the worker and the acid source. There are already occupational safety measures that must be followed, ensuring also the prevention of ETW. Therefore, workers must use personal protective equipment (respiratory masks) and the factories must adhere to the threshold limit values recommended by occupational health legislations.[76]

Sommeliers are also at risk for erosion development due to the chemical characteristics of the wine (acid content, low pH and low concentration of calcium ions) and because of the tasting habits (wine is kept and swilled in the mouth for a prolonged time). However, there are few studies investigating the association between professional wine tasting and erosive tooth wear.[47]

The literature also reports the occurrence of erosion in competitive swimmers.[77] This issue can be easily prevented by well-buffered and pH-controlled chlorinated swimming pool water.[47]

Saliva

Among the patient-related factors, saliva is probably the most important one for the protection against ETW, for many reasons: (1) it is the main contributor to the formation of the acquired pellicle; (2) it acts directly on the erosive agents, diluting, clearing and buffering the acids; (3) being supersaturated with respect to the tooth mineral, it reduces the rate of demineralisation and enhances remineralisation, providing calcium and phosphate to enamel and dentine; (4) many proteins present in saliva and acquired pellicle are able to modulate the rate of ETW.[78,79] It is important to highlight that both the proteins and the ions present in saliva, in the right proportions, are important to protect against ETW.[80] The salivary components play a crucial role in the development of dental erosion. Enamel specimens challenged with citric acid mixed with saliva collected from patients with severe erosive

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lesions suffered more demineralisation when compared to specimens challenged with citric acid mixed with saliva collected from patients without erosive lesions.[81] An interesting study investigated the relationship between saliva parameters and early erosion of hydroxyapatite discs *in situ*. Multivariate analysis revealed a significant role for sodium, urea, total protein, albumin, pH and flow of unstimulated saliva and for sodium, potassium, urea, and phosphorus of stimulated saliva in the erosion of the hydroxyapatite discs.[82]

The salivary flow rate is the best indicator of the protective ability of saliva, since it directly influences the above-mentioned salivary parameters.[83] Higher salivary flow accelerates the clearing of the acids and also increases organic and inorganic components of saliva, thus creating a favourable environment for the prevention of initial erosive attacks. The main buffer of saliva is hydrogen carbonate. Its concentration increased from 5 mmol/l in unstimulated whole saliva up to 60 mmol/l in stimulated whole saliva.[79] The salivary flow can be stimulated both mechanically (by mastication)[84] and chemically (for example, by dripping citric acid droplets to the tongue[85]). Moreover, the proteomic profile of saliva changes following stimulation by different tastes.[86]

The average unstimulated salivary flow rate is around 0.3–0.4 ml/min. Unstimulated flow rates lower than 0.1 ml/min are considered evidence of hyposalivation.[87] The main causes of decreased flow rate are therapeutic drugs, especially when many drugs are combined,[88,89] as well as Sjögren's syndrome[90,91] and radiation treatment for head and neck cancer,[92–94] even though these two latter conditions are rare.

Many clinical trials show an association between reduced salivary flow and increased prevalence of ETW.[68,95–98] There is some controversy in the literature regarding impaired stimulated salivary flow or buffering capacity as they have been shown to be associated with a higher prevalence of erosive tooth wear in patients with GERD.[97,99–102] The cause for the reduction in the stimulated salivary flow in GERD patients is not completely understood,[97] but could possibly be related to the use of proton pump inhibitors that have been associated with xerostomia (sensation of dry mouth).[103] In addition, hyposalivation (OR 3.8, CI 1.2–11.8) was one of the strongest risk indicators for severe erosive wear in a recent study that evaluated the influence of self-reported intrinsic factors, such as, long-term alcoholism, long-term heavy use of alcohol and multiple pregnancies on erosive tooth wear in a middle-aged cohort sample (N = 1,962).[68] Moreover, unstimulated flow rate was found to be significantly lower in adolescents with ETW, while the chloride concentration was significantly higher.[104] In another study that evaluated the effect of salivary factors on ETW, unstimulated salivary buffering capacity and urea concentration in salivary samples of participants aged 16–20 years with no ETW were significantly higher than in those with ETW, while stimulated salivary total protein was significantly higher in the group with ETW in those aged 46–50 years.[105] In a study where the prevalence of wear and stimulated parotid saliva parameters were compared between bulimic and non-bulimic volunteers, the bulimic ones presented a higher prevalence of wear and lower levels of pH, bicarbonates and phosphates in saliva compared with controls, but no significant difference in urea was found.[106]

Suggested preventive measures for patients with hyposalivation and high risk of erosion include the use of chewing gums[53,107–109] and the consumption of hard cheese[110] to increase the salivary flow, as well as the use of saliva substitutes supersaturated with respect to hydroxyapatite.[111] High-viscous saliva substitutes should be preferred, while those with low pH or containing citric acid should be avoided.[112] Mouth rinsing (with water or commercial mouthwashes) after an acidic challenge is able to increase salivary pH and theoretically has the potential to reduce ETW.[113] In more severe cases, such as patients with xerostomia due to radiation on head and neck cancer or diseases such as Sjögren's syndrome, pilocarpine is still the best performing sialogogue.[114] However, the degree of evidence for these preventive measures is low, since it comes from *in vitro* and *in situ* studies. Clinical trials are necessary before these measures can be recommended with a higher degree of confidence.

Acquired pellicle

The acquired pellicle is a thin acellular film formed on the tooth surface by selective adsorption of proteins mainly, but also containing lipids and glycoproteins. Most of the proteins present in the acquired pellicle originate from the salivary glands.[115–117] It is a physical barrier that acts as a permeable membrane, preventing the direct contact between the acids and the tooth surface, thus reducing the dissolution of hydroxyapatite and protecting the tooth against erosive attacks.[118]

The initial phase of pellicle formation occurs within seconds of enamel exposure to saliva.[119] During this phase, precursor proteins that have a high affinity by hydroxyapatite (statherin, histatins, acidic proline-rich proteins, mucins, amylase, cystatins, lysozyme and lactoferrin) selectively adhere to the tooth surface, forming a protein layer 10–20 nm thick.[116] During the second stage of pellicle formation, its thickness increases rapidly (100–1000 nm) due to the adsorption of protein aggregates.[120]

The efficiency of the acquired pellicle to protect against erosive attacks depends on its thickness. The pellicle is thickest on the lingual surfaces of the lower teeth[121] and thinnest on the palatal surfaces of maxillary anterior teeth.[122] ETW shows a typical distribution pattern between the dental arches, being the palatal and occlusal surfaces the most commonly affected.[123] This means that the most affected dental surfaces are the ones presenting the thinnest pellicles. Recently, we showed that the protein composition of the acquired enamel pellicle varies according to its location in the dental arches. For example, cystatins were not identified in the acquired pellicle collected from the posterior teeth. This can be also related to the site-specificity of dental erosion.[124]

Regarding the degree of protection conferred by pellicles formed at different times, pellicles formed over 24 hours or 7 days have no significant difference in the ability to protect against erosion.[119] Moreover, the degree of protection provided by pellicles formed *in situ* for three minutes or two hours is similar.[125] This means that the greatest protection against erosion seems to be provided by the electron dense basal pellicle.[126] This is in-line with the removal of the globular outer layers of the acquired enamel pellicle after the consumption of orange juice, while the basal pellicle is not affected.[127] Considering the partial acid resistance of the acquired pellicle and the fact that toothbrushing can remove parts of the acquired pellicle,[128] patients at high risk of ETW should be instructed to reduce the frequency of toothbrushing and to use dentifrice with low abrasiveness to avoid damaging of the acquired pellicle.[129]

As mentioned above, part of the pellicle remains on the tooth surface, even after severe erosive challenges,[127] which suggests a partial acid resistance of the *in vivo* formed pellicle.[130] This prompted us to investigate which proteins within the acquired enamel pellicle are resistant to removal by acids. Once

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identified, the enrichment of the acquired pellicle with these proteins, a procedure called 'acquired pellicle engineering', could have good potential to protect against ETW. For this purpose, 2-hour *in vivo* formed acquired enamel pellicles were treated with 1% citric acid (pH 2.5) for ten seconds. The remaining acquired enamel pellicles were collected and the acid-resistant proteins were identified by mass spectrometry. Cystatin-B, lysozyme C and salivary acidic proline rich phosphoprotein ½ were increased 20.7, 2.8 and 2.5-fold after treatment with citric acid in comparison to deionised water. These results indicate that these proteins, especially cystatin-B are potentially protective against ETW.[131] However, the cost of human recombinant cystatins is prohibitive for this purpose and alternative homologues are desired. In this sense, we cloned and recombinantly expressed a cystatin derived from sugarcane (called CaneCPI-5). It was observed during the initial tests that this protein strongly adhered to the quartz cuvette.[132] This, in conjunction with the fact that its salivary homologue cystatin-B is highly increased in the acquired enamel pellicle after exposure to citric acid, prompted us to perform experiments that showed that CaneCPI-5 strongly binds to dental enamel and protects against initial enamel erosion *in vitro*. The best protective effect when this protein is added in a rinse solution applied for two hours is achieved at the concentration of 0.1 mg/ml and increasing concentrations do not have an additive effect.[132] Despite these promising results, they should be confirmed using protocols that more closely resemble the clinical condition and, later on, in clinical trials. In addition, other vehicles to deliver CaneCPI-5 to the acquired enamel pellicle should be evaluated.

In fact, the incorporation of proteins into the pellicle layer can affect its ability to protect against erosion. Patients suffering from erosion have half the amount of proteins within the acquired pellicle formed on splint blocks when compared to normal controls.[133] Besides cystatins, other salivary proteins seem to play a role in the ability of the acquired pellicle to protect against erosion. Statherin, a calcium-binding protein, is 35% less abundant in patients suffering from erosion than in controls.[133] This protein strongly binds to hydroxyapatite through its N-terminus[134] and it has been shown that statherin-like peptides containing at last 15 N-terminal residues or more are required for protection against demineralisation.[135] In addition, mucins seem also to be able to inhibit erosive demineralisation,[136] especially when combined with casein,[137] as well as ovalbumin.[138]

'Acquired pellicle engineering' procedures are quite promising in preventing erosive tooth wear. Many proteins and peptides, alone or in combination, have already been tested but so far all the studies were performed *in vitro*. Protocols that more closely resemble the clinical condition, as well as clinical trials, are required before these procedures can be employed clinically.

Dentine erosion: the role of host protease inhibitors

In dentine, the use of host protease inhibitors to preserve the demineralised organic matrix after an erosive attack has a protective role against further mineral loss,[139] which has been highlighted in recent reviews.[140–143] The host protease inhibitors tested, in different vehicles such as rinse solutions or gels, are chlorhexidine,[144,145] iron,[146] epigallocatechin gallate,[144,145,147,148] sodium fluoride,[144,148,149] anacardic acid[148] and proanthocyanidin.[150] Despite the promising results obtained in *in situ* studies, clinical trials are necessary to confirm these findings and to establish protocols of use of these inhibitors.

Concluding remarks

The preventive management of ETW and dental erosion is quite complex, since a plethora of factors, some related to nutrition and others related to the patient, drive the progression of the lesions. The preventive management depends on the early recognition of the first signs of the lesions to ensure that appropriate strategies to fight the risk factors can be implemented in due time.

The nutritional factors are associated to the composition of the food and drink. In general, low pH and high buffer capacity are the major risk factors, while the calcium concentration is the main protective factor. However, in the face of the multifactorial aetiology of the lesions, the characteristics of the food and drink must be investigated in conjunction with patient-related factors, by using dietary and behaviour records over some days. Reduction of the frequency of consumption and contact time of potentially erosive foods and drinks with the teeth are important, as well as the use of straws positioned toward the back of the mouth. It is also advisable to avoid the consumption of erosive foods and drinks last thing at night, replacing them with dairy products, which could also be consumed at the end of meals. In the case of unhealthy life styles, such as drug abuse and chronic alcoholism as well as in the presence of medical conditions that increase the risk of ETW, such as GERD and bulimia, the patient must be referred to a doctor for proper causal treatment. Immediately after vomiting, patients might be advised to rinse the mouth with water, milk, sodium bicarbonate solution or preferably fluoride containing mouth rinse. Some liquid oral medicines, chewable tablets and effervescent vitamins with prolonged use are potentially erosive and health professionals must recommend a safe intake form without direct contact with the teeth whenever possible. Workers exposed to acid or acidic fumes must use personal protective equipment (respiratory masks) and the factories must respect occupational health legislation. Patients with reduced salivary flow caused by different conditions (use of certain therapeutic drugs, Sjögren's syndrome and radiation treatment for head and neck cancer) can benefit from the use of chewing gum and the consumption of hard cheese. In addition, the use of high-viscous, supersaturated with respect to hydroxyapatite saliva substitutes has been proposed. Furthermore, patients at high-risk of ETW must brush with fluoridated dentifrice and might also benefit from the use of high-concentration, acidic and polyvalent fluoride formulations, as well as the use of formulations containing protease inhibitors in case of dentine lesions. Delaying toothbrushing after erosive challenges does not prevent ETW.

However, the degree of evidence for these preventive measures is low, since it comes from *in vitro* and *in situ* studies. Clinical trials are necessary before these measures can be recommended with a higher degree of confidence.

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