Dental Erosion – An Overview with Emphasis on Chemical and Histopathological Aspects

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Key Words

Chemical mechanisms · Dental erosion diagnosis · Histopathology of erosion · Risk factors · Tooth erosion

Abstract

The quality of dental care and modern achievements in dental science depend strongly on understanding the properties of teeth and the basic principles and mechanisms involved in their interaction with surrounding media. Erosion is a disorder to which such properties as structural features of tooth, physiological properties of saliva, and extrinsic and intrinsic acidic sources and habits contribute, and all must be carefully considered. The degree of saturation in the surrounding solution, which is determined by pH and calcium and phosphate concentrations, is the driving force for dissolution of dental hard tissue. In relation to caries, with the calcium and phosphate concentrations in plaque fluid, the 'critical pH' below which enamel dissolves is about 5.5. For erosion, the critical pH is lower in products (e.g. yoghurt) containing more calcium and phosphate than plaque fluid and higher when the concentrations are lower. Dental erosion starts by initial softening of the enamel surface followed by loss of volume with a softened layer persisting at the surface of the remaining tissue. Dentine erosion is not clearly understood, so further in vivo studies, including histopathological aspects, are needed. Clinical reports show that exposure to acids combined with an insufficient salivary flow rate results in enhanced dissolution. The effects of these and other interactions result in a permanent ion/substance exchange and reorganisation within the tooth material or at its interface, thus altering its strength and structure. The rate and severity of erosion are determined by the susceptibility of the dental tissues towards dissolution. Because enamel contains less soluble mineral than dentine, it tends to erode more slowly. The chemical mechanisms of erosion are also summarised in this review. Special attention is given to the microscopic and macroscopic histopathology of erosion.

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In the last decade, tooth erosion and the associated mechanical wear (erosive tooth wear) have drawn increasing attention as a risk factor for tooth damage. There is some evidence that the prevalence of erosion is growing steadily [Jaeggi and Lussi, 2006], even though it is difficult to compare and judge the outcome reported in the studies because of different indices and examiners, as well as different samples under investigation. However, the prevalence (expressed as >1 tooth with involved dentine) seems to be over 30% [Jaeggi and Lussi, 2006].

Erosive demineralisation of the tooth crown is characterised by initial softening of the enamel surface, which varies depending on the immersion time and the acids under study. The thickness of this softened layer was reported to be between 0.2 and 3 μm [Amaechi and Higham, 2001; Eisenburger et al., 2001; Wiegand et al., 2007; Cheng et al., 2009; Voronets and Lussi, 2009]. This process is followed by continuous layer-by-layer dissolution of the enamel crystals, leading to a permanent loss of tooth volume with a softened layer at the surface of the remaining
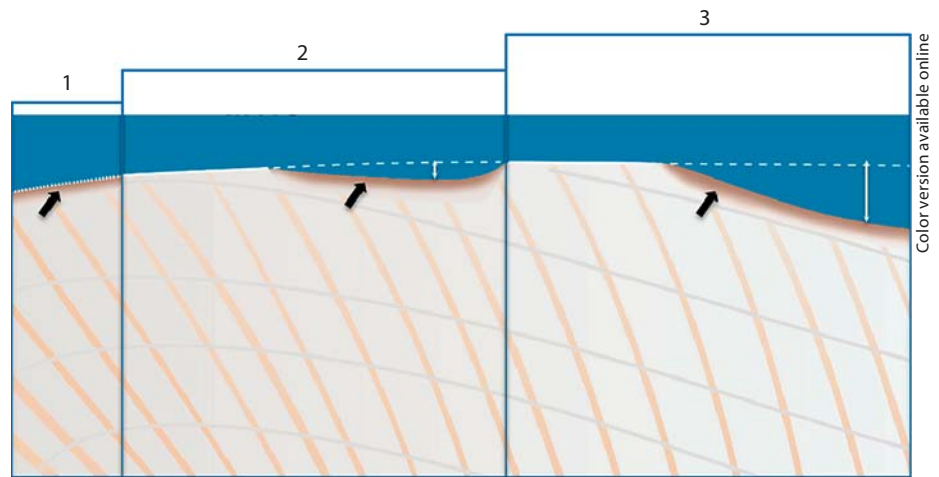
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Fig. 1. Different stages in the continuous erosion process: (1) softening of the enamel (no material loss; black arrow); (2) partial material loss (erosive tooth wear; white arrow) and softening of the underlying surface (black arrow), and (3) significant loss of material (white arrow).

tissue. In advanced stages, dentine becomes increasingly exposed (fig. 1).

The responsible acids stem from intrinsic (such as eating disorders or gastric reflux) or extrinsic sources. One important extrinsic factor in erosive tooth wear is the high consumption of acidic drinks and food. In recent years, the total amount and frequency of consumption of acid-containing products have increased because of changes in lifestyles [Packer, 2009].

Even though the prevention of the excessive use of soft drinks is an issue in oral health education, it is impossible to avoid potentially erosive agents from contacting the teeth during the entire lifetime. Therefore, to prevent erosion, emphasis should be placed on early diagnosis and adequate preventive strategies.

In this field, an increasing body of experimental in vitro and in situ research is published, accompanied by discussion about methodology and study design. Many approaches stem from cariology and are validated for the respective experimental needs. Diagnosis, aetiology, pathology and histology of dental erosion, however, differ so basically from caries that specific considerations regarding the methodology in erosion research are necessary.

The aim of this overview is to give some insight into the histopathology of erosion and to discuss important protective and risk factors.

Diagnosis

In routine dental practice, a specific tool detecting dental erosion and its progression is still lacking. Therefore, clinical appearance remains the most important feature to diagnose tooth erosion for dental professionals [Lussi et al., 2006]. It is difficult to diagnose erosion in the early stages. Typical signs of enamel erosion are the appearance of smooth, silky-glazed, sometimes dull enamel in the absence of perikymata, but with intact enamel along the gingival margin. The initial features of erosion on occlusal and incisal surfaces are the same. Further progression of occlusal erosion leads to a rounding of the cusps and restorations rising above the level of the adjacent tooth surfaces (fig. 2a). In severe cases, the entire occlusal morphology disappears (fig. 2b). Furthermore, loss of enamel can lead to dentine exposure with reactionary dentine formation by odontoblasts. The exposed dentine surface may become sensitive to cold and warm foods and to tactile stimuli. In the more advanced stages, e.g. when dentine is exposed, agents which render dentine visible can be used.

Erosion has to be distinguished from attrition (tooth wear induced by tooth-tooth contact) and abrasion (tooth wear caused by interaction between teeth and other materials). It is not always easy to differentiate these wear lesions, because they frequently occur simultaneously with different proportional effects [Nunn et al., 1996]. As the tooth enamel layer demineralises, it becomes more susceptible to abrasion and attrition.

Risk Factors and Aetiology

Tooth erosion is a multifactorial condition (fig. 3) and has a complex aetiology. Every factor plays a role in inducing or preventing erosion. Over time, the interaction of all these factors may lead to either progression or indeed protection of the surface.

Fig. 2. Initial (**a**) and advanced (**b**) occlusal erosive tooth wear. The amalgam and composite fillings rising above the level of the adjacent tooth surface are clear signs of erosion. Note: Initial caries can be seen in the proximal area of the premolars (**b**).
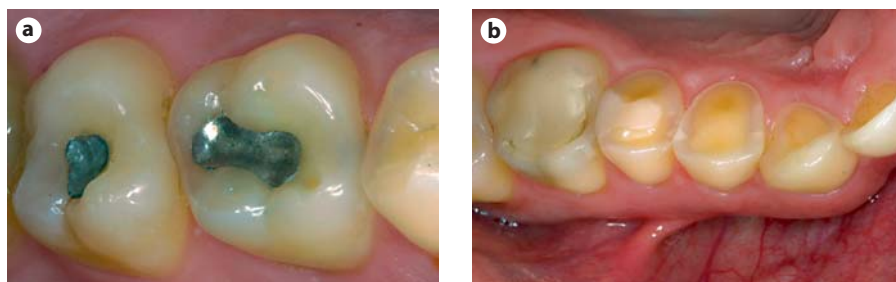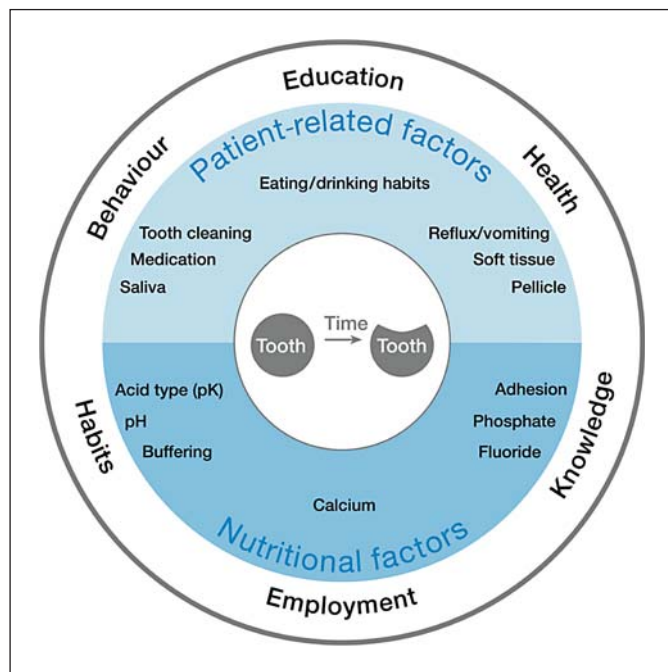
Fig. 3. Patient-related and nutritional factors affecting the development of erosion.

The most important extrinsic source of acid exposure is diet, which could include numerous components and products with complex composition and a potential for erosive damage. Apart from diet, the type of occupation and sport practised by the patient may also lead to erosive wear. Employees in the chemical industry or professional wine tasters have a higher risk of suffering erosions due to the increased acid-tooth contact [Wiegand and Attin, 2007]. Tooth erosion associated with professional athletes or excessive exercise has also been reported occasionally [Centerwall et al., 1986]. Excessive exposure to water or sport drinks with low pH, or the increased gastro-oesophageal reflux resulting from strenuous exercise may be responsible for the sport-induced erosion [Centerwall et al., 1986]. Importantly, occupation and sport can only be considered as co-factors rather than primary factors in the occurrence and development of erosion because erosion is a multifactorial process.

Another aetiological factor is stomach acid, which enters the mouth as a consequence of chronic vomiting or reflux. Reflux is the involuntary movement of gastric contents from the stomach into the mouth due to some abnormality in the gastro-intestinal tract. In general, acidic gastric contents entering the mouth might erode the teeth if they have acted on the dental hard tissues regularly over some time. It has to be kept in mind that reflux may occur without symptoms. Therefore, patients with severe erosion should be examined for gastro-oesophageal reflux disease.

Patient-related risk factors have also to be taken into account and are discussed below with emphasis on physicochemical factors.

The manner in which dietary acids are introduced into the mouth (sipping, sucking, with/without drinking straw) determines the duration and the localisation of the acid attack [Millward et al., 1997; Edwards et al., 1998; Johansson et al., 2004]. In addition, the mechanical abrasion caused by the tongue was considered to be a factor contributing to erosion [Holst and Lange, 1939].

The frequency and duration of acid attacks are closely associated with erosion and, therefore, are very important factors for the adoption of prophylactic measures [Järvinen et al., 1991; Lussi and Schaffner, 2000; O'Sullivan and Curzon, 2000; Johansson et al., 2002]. Contact of the teeth with acids during the night can lead to erosion because of the reduced production of saliva. Thus, for example, apart from carious lesions, there can be massive erosive tooth structure destruction due to consumption of acidic, sweet drinks, which some infants drink from their bottles continuously during the night.

Clinical experience demonstrates the importance of saliva, as erosion can be severe in patients with impaired salivary flow. Several salivary protective mechanisms come into play during an erosive challenge, such as dilution and clearance of an erosive agent from the mouth, neutralisation and buffering of acids, and formation of the acquired pellicle [Zero and Lussi, 2000].

The acquired pellicle, composed of glycoproteins, proteins, lipids and several enzymes [Hannig et al., 2005], is also considered to be an important factor. This film is assumed to protect against erosion by acting as a diffusion barrier or a selectively permeable membrane preventing direct contact between acids and the tooth surface, and it has been shown that at least its basal structure survives relatively severe acid exposures [Hannig and Balz, 1999]. In vitro experiments have indeed demonstrated protective effects after relatively mild acid challenges [Amaechi et al., 1999; Wetton et al., 2006; Wiegand et al., 2008], but to a lesser extent under more severe conditions [Hara et al., 2006a; Cheaib and Lussi, 2011]. In no case, however, was the protection against erosive dissolution complete. A recent in situ study has shown that consumption of soft drinks for only 20 s led to a decrease in surface microhardness even though pellicle structures survived on the tooth surface [Hannig et al., 2009].

In summary, it is obvious that saliva is important for protection against erosive demineralisation, but further research is necessary to identify saliva risk factors.

Tests of the stimulated and unstimulated flow rates as well as of the buffering capacity of saliva may provide some information about the susceptibility of an individual to dental erosion [Lussi and Schaffner, 2000; Holbrook et al., 2009]. However, these properties are only two aspects of a multifactorial condition. Sialometric evaluations should be carried out at a fixed time point or within a limited time interval in the morning in order to avoid intra-individual variation due to the circadian cycle. Studies have shown that sour foodstuff has a strong influence on the anticipatory salivary flow [Christensen and Navazesh, 1984; Lee and Linden, 1992], which can be significantly increased compared to the normal unstimulated flow rate [Engelen et al., 2003]. Besides the acidity of a foodstuff, low temperature and mechanical stimulation will further enhance salivary flow [Dawes et al., 2000]. Hypersalivation also occurs prior to vomiting as a response from the 'vomiting centre' of the brain [Lee and Feldman, 1998] and is frequently seen in individuals suffering from anorexia, bulimia nervosa, rumination or chronic alcoholism. It is suggested that this could reduce the erosion caused by acids of gastric origin. On the oth-

er hand, patients with symptoms of gastro-oesophageal reflux should not expect salivary output to increase before gastric juice reflux since this is an involuntary response that is not coordinated by the autonomic nervous system [Hara et al., 2006b]. Therefore, there might be insufficient time for saliva to act before erosion occurs.

Millward et al. [1997] monitored the pH at the tooth surface in healthy volunteers after drinking 1% citric acid. They observed that the pH recovered to pH >5.5 within 2 min at a site adjacent to the palatal surface of the upper central incisor and within 4–5 min at another palatal surface on the upper first molar. Thus, the clearance rate of erosive agents may be influenced by the anatomy of the teeth and soft tissues, by the movement of the tongue and vestibular mucosa as well as by the swallowing pattern.

Histology, Ultrastructure and Related Physical Aspects

The normal histology of the dental hard tissue has been extensively studied [Berkovitz et al., 1989]. In the following, only a few aspects which could be of importance in erosion experiments are mentioned.

Human dental enamel is a highly mineralised tissue mainly composed of a non-stoichiometric form of hydroxyapatite (see below). Enamel has a mostly prismatic structure. These enamel prisms, often described as keyhole shaped, are bundles of elongated crystallites. The crystallite width and thickness seem to be about 50–70 and 20–25 nm, respectively [Nikiforuk, 1985], but the length is difficult to measure and is sometimes considered to be indeterminate [Orams et al., 1976]. From isolated crystals, lengths around 100 μm were measured, and from these observations it was concluded that the crystals can extend through the entire thickness of the enamel [Daculsi et al., 1984]. The crystals are densely packed and the mineral content is around 87 vol%, assuming that the mineral has the composition of hydroxyapatite [Nikiforuk, 1985]; higher values for the apatite content of enamel (96 vol%) have been calculated on the basis of the mineral composition [Elliott, 1997]. Other constituents of enamel are water (11 vol%) and organic material (2 vol%) [Nikiforuk, 1985]. For the density of the tissue, values ranging from 2.89 to 3.12 $g \cdot cm^{-3}$ have been published [Nikiforuk, 1985; Elliott, 1997]. The values for enamel hardness depend on the measuring technique and cannot be considered as constant [Collys et al., 1992]. The values obtained using a Knoop diamond are inverse-

ly related to the load used. For sound enamel, Knoop hardness numbers of 431 and 339 at loads of 50 and 200 g, respectively, were reported [Collys et al., 1992]. Within a sound tooth, Knoop hardness of enamel may vary, e.g. between 280 and 390 using a load of 100 g [Lussi, unpubl. data].

To some extent, the chemical composition and physical properties of enamel change with depth [Weatherell et al., 1974; Meredith et al., 1996], which might be worth noting in sensitive experiments. The density and hardness of the tissue tend to decrease with increasing distance from the surface [Weatherell and Robinson, 1973; Meredith et al., 1996; He and Swain, 2009], and solubility increases [Theuns et al., 1986]. The mineral content reaches a maximum in areas where the enamel is thickest and decreases to the cervical region [Theuns et al., 1983].

Dentine is different from enamel in structure and composition, and is more soluble than enamel but probably not as soluble as it has been suggested in the past [Shellis et al., 2010]. Its mineral content is much lower (47 vol%), whereas the organic content is much higher (33 vol%). The organic portion consists mainly of collagen type I, which constitutes about 90 wt% of the organic fraction. Other components are non-collagenous phosphoproteins and glycoproteins as well as proteogycans and lipids. Dentine is a relatively moist tissue containing about 21 vol% of water [Nikiforuk, 1985]. The hardness of dentine is much lower than that of enamel [Meredith et al., 1996], but there are also factors that influence the outcome of hardness measurements, in particular the elastic properties and shrinkage due to drying [Herkstroter et al., 1989]. Further, the peritubular dentine is harder than the intertubular areas [Kinney et al., 1996].

The tissue is penetrated by a number of dentinal tubules extending from the pulp to the enamel-dentine or cementum-dentine junction. The tubules are surrounded by peritubular dentine, which represents the most mineralised portion of the tissue with a mineral content ~40% higher than in the intertubular regions [Frank and Nalbandian, 1989], and its organic content seems to be minimal. The intertubular dentine consists mainly of calcified collagen with the mineral crystals located within and between the collagen fibrils. The mineral consists of imperfect hydroxyapatite in the form of thin hexagonal plate-like crystals, around 3.4 nm thick, 14 nm wide and 25 nm long. The crystals are generally oriented with their c-axis parallel to the collagen fibrils, though divergent arrangements frequently occur [Takuma et al., 1986]. Peri- and intertubular crystals are of similar shape. Besides calcium and phosphate, other constituents and trace ele-

ments also occur in different proportions to enamel [Weatherell and Robinson, 1973].

Dentine shows structural differences in relation to depth, with the most relevant being the number and diameter of tubules in the context of erosion research. The number of tubules ranges from 45,000 to 65,000/mm² close to the pulp and from 29,500 to 35,000/mm² near the enamel-dentine junction. Tubule diameters range from 2 to 3 μm at the pulpal side and from 0.5 to 0.9 μm at the dentino-enamel junction [Frank and Nalbandian, 1989], which is attributed to differences in peritubular dentine thickness.

A further aspect for methodologies in erosion research might be the presence of the smear layer after preparation. This is formed when dentine or enamel is prepared and consists of an amorphous surface layer of tissue debris that, in the case of dentine, may plug the tubules [Pashley et al., 1988]. The presence or absence of the smear layer modifies the permeability of dentine, which could be relevant in experiments with short-term erosion, even if most of the smear layer has previously been removed after application of 6% citric acid for 30 s [Pashley et al., 1981].

Chemical Aspects of Erosion

Teeth are composed of a calcium-deficient carbonated hydroxyapatite containing some fluoride. The molar Ca/P ratio is ~1.61 for enamel minerals (cf. 1.67 for hydroxyapatite). A simplified formula of tooth mineral composition is $Ca_{10-x}Na_n(PO_4)_{6-y}(CO_3)_z(OH)_{2-u}F_u$, which is different from stoichiometric hydroxyapatite with the formula $Ca_{10}(PO_4)_6(OH)_2$. The substitutions, especially carbonate, in the mineral crystal lattice weaken the enamel structure [Featherstone et al., 1983; Featherstone and Lussi, 2006]. As a consequence, the mineral in enamel and dentine is somewhat more acid soluble than hydroxyapatite, which in turn is more soluble than fluorapatite with the formula $Ca_{10}(PO_4)_6F_2$. Tooth mineral also includes lower concentrations of sodium, magnesium, chloride, potassium and various trace elements [Weatherell and Robinson, 1973; Elliott, 1997]. The concentration of fluoride is normally in the order of 0.01% dry weight, but can vary substantially.

The critical pH is the pH at which a solution is just saturated with respect to a specified solid, e.g. enamel mineral. If the pH of the solution is less than the critical pH, the solution is undersaturated and can dissolve the solid, while the solution is supersaturated if the pH is

above the critical pH, and thus more mineral may precipitate. The critical pH depends both on the solubility of the solid of interest and on the concentrations (or more correctly on the activities) of the relevant mineral constituents of the solution. In case of tooth mineral, the principal relevant constituents are calcium, phosphate and to a lesser extent the fluoride activity since they determine the degree of saturation in the solution, which is the driving force for dissolution and precipitation.

In relation to caries, the relevant fluid is the plaque fluid. In resting plaque, the calcium and phosphate concentrations in the fluid are around 3.5 and 13.2 mmol/l, respectively. Fermenting plaque contains 8.2 mmol/l of calcium and 13.5 mmol/l of phosphate [ten Cate et al., 2008]. On the basis of these average concentrations, the critical pH for plaque fluid may be 5.5–5.7. The concentrations of calcium and phosphate are rather constant for a given person but may differ between people, which explains in part interindividual differences in the critical pH value.

By definition, dental erosion is the dissolution of tooth mineral in the absence of plaque. Therefore, the critical pH determined from the composition of plaque fluid is not a guide to whether erosion can occur. The solution adjacent to tooth mineral may have higher concentrations of calcium and phosphate than plaque fluid and will therefore not dissolve tooth mineral even at pH values lower than the critical pH for caries. For example, yoghurt has a pH around 4, a calcium concentration of up to 42.5 mmol/l, phosphate content of up to 49.8 mmol/l and as it is supersaturated with respect to enamel it has no erosive impact [Lussi and Jaeggi, 2006]. Similarly, orange juice (pH 4) supplemented with calcium (42.9 mmol/l) and phosphate (31.2 mmol/l) did not erode enamel after immersion for 7 days [Larsen and Nyvad, 1999].

When the liquid surrounding the tooth surface is only slightly undersaturated with respect to the mineral, initial surface demineralisation occurs followed by a local pH rise and increased concentrations of ions in the liquid surface layer adjacent to the tooth mineral. As a result, this layer becomes saturated and does not demineralise enamel further. Consequently, no softening can be measured with most modern methods. An increase in agitation (e.g. when a patient is swishing the drink in the mouth) will enhance the dissolution process as the semistatic layer of solution (the Nernst layer) immediately adjacent to the surface region of tooth mineral will be constantly replaced without reaching saturation level.

In summary, the concentrations of calcium and phosphate ions in a drink or in food play a key role in tooth erosion since, together with the pH value, they determine the degree of saturation with respect to enamel or dentine. In other words, there is no defined critical pH for erosion as there is for caries.

Apart from the degree of saturation, the ability of an acidic solution to dissolve enamel or dentine depends on its buffering capacity, which is related to the undissociated acid concentration in drinks and food. Consequently, the driving force for demineralisation at the site of dissolution is maintained [Gray, 1962; Featherstone and Rodgers, 1981]. The greater the buffering capacity of the drink, the longer will it take for saliva to neutralise the acid, and the more mineral may be dissolved before dissolution ceases.

Histopathological Aspects of the Erosive Process

The histology of erosion is strikingly different from that of caries, and the histology of enamel erosion is basically different from dentine erosion. Considering the specific morphology of erosion of dental hard tissues and the effects of treatment strategies, the histological appearance is particularly important for the appropriate choice and use of measuring methods as well as for the interpretation of study results.

Erosive demineralisation of enamel is a centripetal process starting with the partial loss of surface mineral causing an increase in roughness [Nekrashevych and Stösser, 2003]. If the acid impact continues, bulk mineral loss occurs while the remaining surface still exhibits partial demineralisation (fig. 4a). The surface structure of eroded enamel corresponds more or less to a typical etching pattern [Meurman and Frank, 1991; Eisenburger et al., 2004]. On cross sections, a thin, loosely structured layer is present at the surface, but this can vary in structure and extent, depending on the stages of erosion and treatment strategies [Schlueter et al., 2009; Wiegand et al., 2009] (fig. 4b).

The partial loss of mineral at the surface results in a loss of hardness (softening), which progresses with continuing acid impacts and makes eroded enamel surfaces vulnerable to physical impacts [Attin et al., 1997; Jaeggi and Lussi, 1999; Voronets et al., 2008]. After prolonged or severe erosion has caused loss of volume from the tooth surface, a softened layer persists at the surface of the remaining tissue (fig. 1). The thickness and microhardness of this softened layer will reach a steady state and will not change further during further loss of tissue from the tooth surface. With respect to sound enamel, the order of

Fig. 4. SEM images of cross sections from enamel specimens eroded with citric acid (pH 2.3, 6 × 5 min/day, then stored in a mineral salt solution; 10 days). Samples were fractured, embedded and polished. **a** Distinct bulk tissue loss with a thin, loosely structured, partially demineralised layer at the eroded surface. **b** Surface layer from **a** at higher magnification. Fluffy surface structure followed by a denser area before the sound tissue is reached.
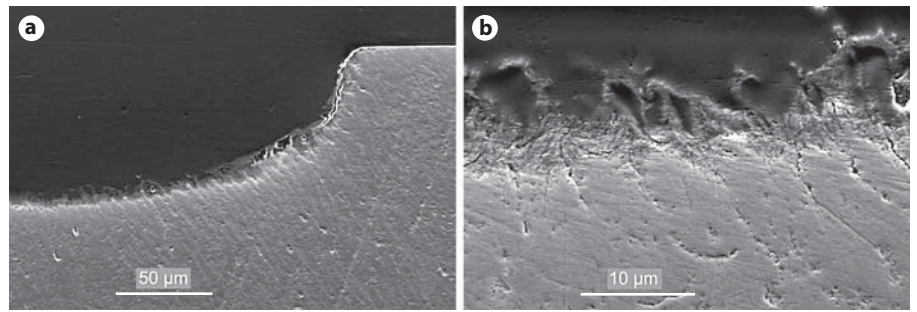
Fig. 5. SEM images of a cross section from a dentine specimen eroded with citric acid (pH 2.3) for 120 min, then fractured and critical point dried. **a** Overview: images **b–d** derived from the marked area. **b** Decalcified organic matrix, with enlarged tubules and fibrous intertubular structure. **c** The intertubular areas appear more calcified but the peritubular dentin is still missing and tubules appear enlarged. **d** Mineralised intertubular dentine, first signs of peritubular dentine (arrows) and decrease in tubule diameter with depth.
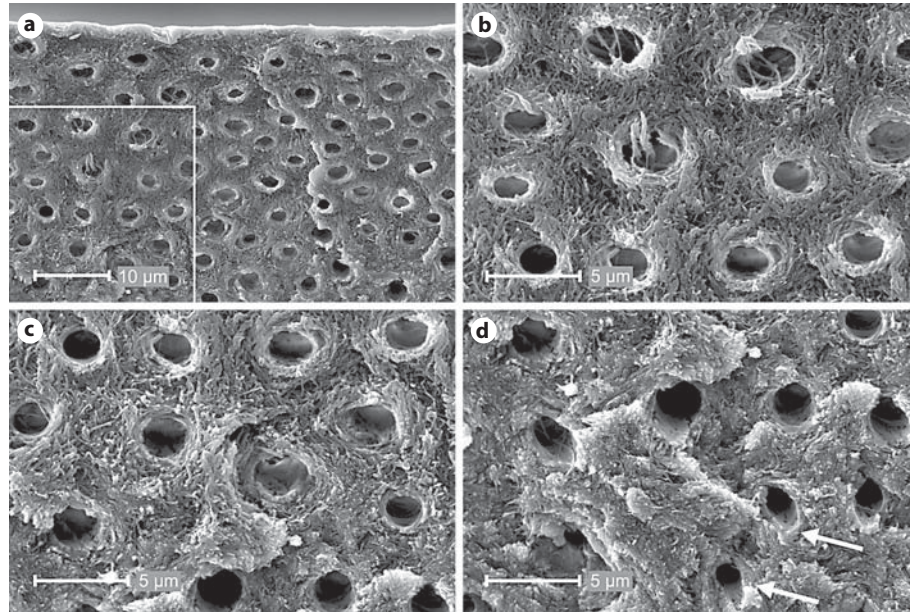
values obtained using a Knoop diamond (Knoop hardness number) depends on the load applied. Importantly, it should be considered that the relationship of the Knoop hardness number between sound and eroded enamel is not proportional. Using loads of 50, 100, 145 and 200 g gives a reduction in hardness after etching of 75, 68, 57 and 52% compared to the sound tissue [Collys et al., 1992]. Ultrasonication can remove the outer, more demineralised part of the softened layer, but the sound enamel is not reached [Eisenburger et al., 2004]. Similarly, toothbrush abrasion removes the outer softened tissue, but the abraded surface retains an appearance similar to an etching pattern [Rios et al., 2008].

In initial caries lesions, remineralisation of the remaining enamel subsurface structure can take place. In case of erosion, mineral gain takes place in the partly demineralised surface enamel. Being a fixed term in cariology, 'remineralisation' should be therefore reconsidered in the context of erosion. The extent to which mineral is deposited in situ or in vivo is not very well known. From intra-oral exposure to saliva, only limited mineral precipitation can be expected. Scanning electron microscopy (SEM) studies have shown that after immersion of etched enamel into the oral environment for 1 h, the etching pattern is still clearly visible [Rios et al., 2008], and softened enamel was not capable of rehardening after hours in situ [Lippert et al., 2004]. Even after weeks in situ, neither significant deposition of mineral salts was found at etched enamel surfaces [Garberoglio and Cozzani, 1979; Allin et al., 1985] nor does exposure to the oral environment lead to a relevant increase in microhardness [Collys et al., 1991, 1993]. Enamel will therefore be still vulnerable to abrasive forces such as tooth brushing even hours after softening.
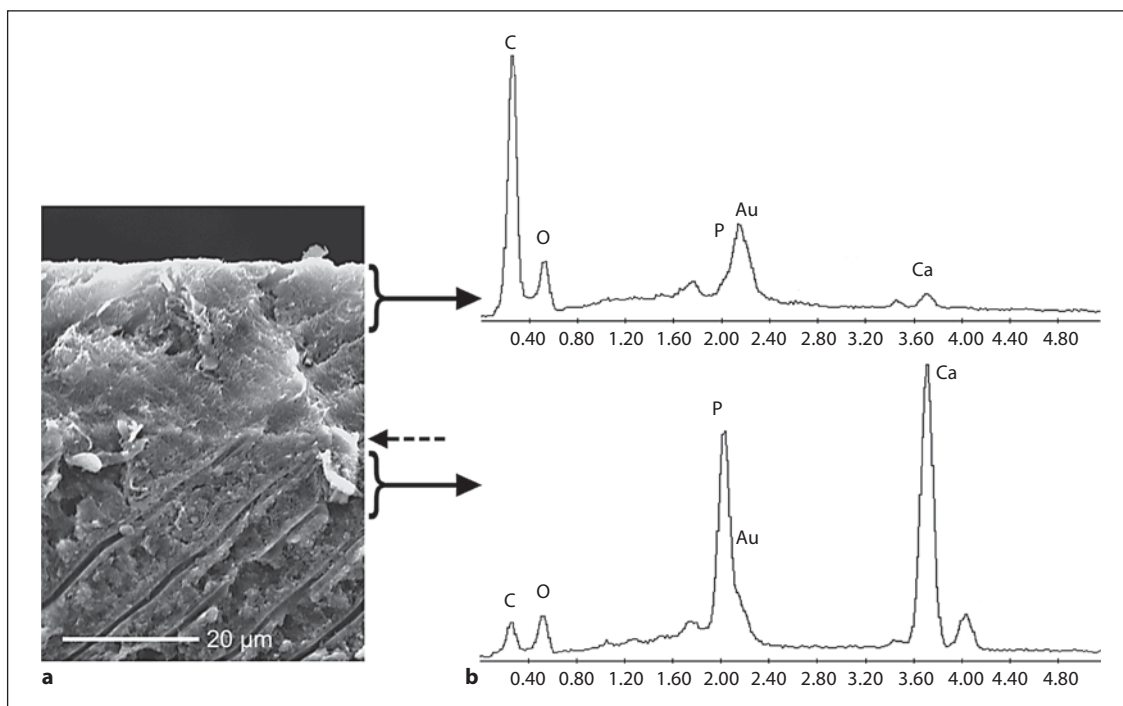
Fig. 6. a SEM image of a cross section of a dentine specimen subjected to erosion with 0.5 M citric acid (pH 2.3; 6 × 5 min/day, storage in a mineral salt solution; 10 days). The dotted arrow indicates the clear-cut boundary between the demineralised and the mineralised tissue. **b** Energy-dispersive X-ray spectra (based on the same scale) of the demineralised and mineralised regions of the tissue. The relative heights of the Ca and C peaks in the two spectra reflect the relative mineral contents.

From these findings, the question of whether or not to include storage periods in natural/artificial saliva in experimental models arises.

When mineral salt solutions or preparations of active agents are applied, the partly demineralised surface can gain mineral due to the precipitation of various salts. Particularly after application of solutions containing polyvalent metal cations, e.g. tin or titanium fluorides, distinct coatings were found [Ellingsen, 1985; Büyükyilmaz et al., 1997; Ganss et al., 2008; Wiegand et al., 2009]. This effect might interfere with measuring methods and need to be considered when interpreting study results.

When dentine is exposed to acids at clinically relevant strength and concentration, the mineral component readily dissolves while the organic portion is retained. Even after a 30-second immersion in low pH citric acid, a thin zone of a dense, fibrous collagen network becomes present [Breschi et al., 2002]. This zone increases with increasing erosion time and, depending on the demineralisation agent and exposure time, a fully demineralised zone develops (fig. 5a, b), beneath which partially demineralised dentine is found (fig. 5c, d), and
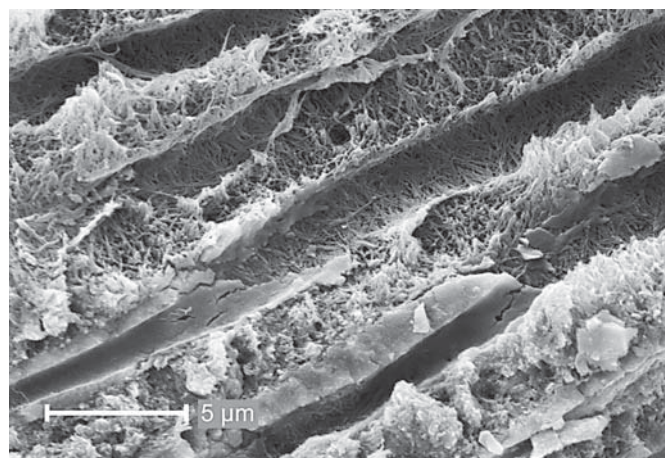

Fig. 7. SEM image of a cross section of a dentine specimen subjected to erosion in hydrochloric acid (pH 1.6; 6 × 2 min/day, storage in a mineral salt solution; 9 days), then fractured and critical point dried. The tubules are cut longitudinally. Sharp border between the fully demineralised and the fully mineralised tissue.

Fig. 8. SEM images of replicas from the palatal surface of the left upper canine from a 30-year-old male with severe chronic vomiting. **a** Enamel with distinct signs of erosive demineralisation similar to an etching pattern. **b** Exposed dentine with open tubules surrounded by peritubular dentine.
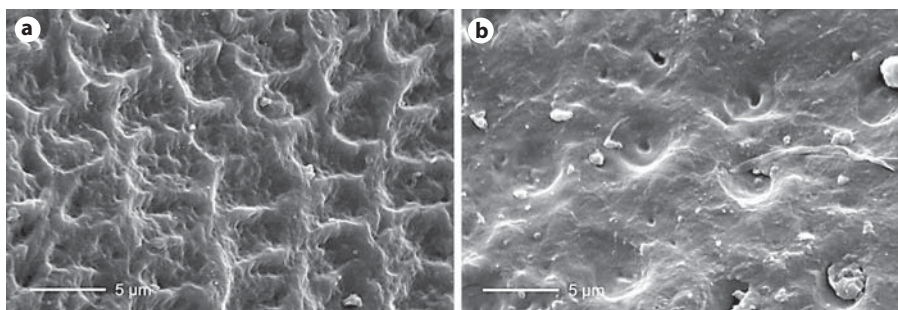
finally sound dentine (fig. 6) is reached [Kinney et al., 1995]. The partially demineralised zone, however, is not always present (fig. 6, 7). Initially, both peri- and intertubular dentine recede at similar rates, but after the first minute, the peritubular dentine is further dissolved while the intertubular region appears more stable [Kinney et al., 1995].

With continuing acid exposure, the demineralisation rate decreases. When the demineralised matrix has reached a certain thickness, the mineral loss decreases markedly [Hara et al., 2005; Ganss et al., 2009b]. This might be partly due to buffering properties of collagen, which lead to a lower pH decrease at the demineralisation front. In addition, dissolved mineral will be prevented from immediate removal by the surrounding liquid phase, thus increasing the state of saturation. Vice versa, the removal of the organic matrix increases the dissolution rate [Kleter et al., 1994; Hara et al., 2005].

Compared to an uneroded reference area, the surface of the retained organic portion represents the original sample surface as long as it is kept hydrated, but is prone to rapid shrinkage at ambient air [Ganss et al., 2007]. It is very stable against mechanical impacts. Brushing abrasion experiments have shown that not only the organic covering as such, but also its structure, is preserved at brushing forces of 2 N [Ganss et al., 2007]. Even at much higher loads, the structure is not removed [Ganss et al., 2009a]. These findings are consistent with the organic material having a considerable tensile strength. Completely demineralised coronal dentine has a tensile strength of 9–17 MPa, depending on the shear direction relative to the direction of the tubules [Miguez et al., 2004], but even values of 26–32 MPa are reported [Sano et al., 1994]. The stiffness of the decalcified dentine reversibly increases with dehydration, whether by immersion in acetone or alcohol, or by air drying, and irreversibly increases after treatment with glutaraldehyde [Maciel et al., 1996]. The organic matrix, however, is degraded by specific and unspecific proteolytic enzymes [Kleter et al., 1994; Ganss et al., 2004; Tonami and Ericson, 2005; Schlueter et al., 2010] or by sodium hypochlorite [Hara et al., 2005].

Knowledge about the histological structure of erosive lesions developing in vivo is of importance for experimental designs and in particular for interpreting study outcomes. Only very little information, however, is available on this matter.

In enamel, loss of lustre is a diagnostic criterion of early erosion indicating an increase in roughness. Indeed, in a patient with a severe eating disorder and with chronic vomiting, SEM images of replicas showed a structure similar to an etching pattern (fig. 8a). Other enamel erosive lesions appear much smoother. For dentine, the question arises if and to what extent organic surface material is also present in vivo (fig. 8b). From clinical experience, in vivo dentine lesions are relatively hard when scratched with a probe, which is in stark contrast to experimental samples exhibiting a resilient and soft surface. In other cases, the surface of erosive lesions appears smooth with traces of microwear. The surface structure of in vivo lesions is caused by the manifold physical and chemical impacts and appears smoother or more etched depending on the main causative factor as well as on lesion activity.

In conclusion, the complex aetiology and histology of erosive lesions require a thorough consideration of study designs. In particular for dentine, further research is necessary to develop suitable methodologies. In this context, an important issue to consider is the role of the organic matrix.

Disclosure Statement

The authors declare that they have no conflicts of interest in relation to this paper.

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