Influence of Fluoride on the Prevention of Erosive Lesions – a Review

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Summary: This review describes various forms of fluoride applications for reducing dental erosive lesions induced by acidic substances. Fluoride admixtures to acidic solutions in a concentration excluding toxicologically side-effects seem unable to arrest erosive lesions. By contrast, topical fluoride applications of toothpastes, oral rinses, gels or varnishes appear to be an effective agent for reducing demineralization by erosion. In particular, high-concentrated fluoride applications are able to increase abrasion resistance and decrease the development of erosions in enamel and dentin.

Key words: fluoride, erosion, abrasion

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INTRODUCTION

While caries has declined in developed countries (Petersson and Bratthall, 1996), other dental lesions such as erosions are becoming increasingly important (Nunn et al, 2003).

Dental erosion (erosive tooth wear) is defined as the loss of tooth substance by chemical processes not involving bacteria (Zipkin and McClure, 1949) caused by a variety of extrinsic and intrinsic factors. Extrinsic factors causing dental erosion are diet, environment medication and lifestyle. It is well documented that most acidic food and drinks have the potential to cause dental erosion. Furthermore, the total acid level, acid type, concentration of phosphate, calcium, fluoride as well as frequency and time of consumption have a modifying effect on the development of erosion induced by dietary components (Behrendt et al, 2002; Grenby et al, 1989; Larsen and Nyvad, 1999; Lussi et al, 1993; Sovari et al, 1989; Thomas, 1957; West et al, 2000). Environmental factors such as exposure to acid fumes, low pH-medication as well as lifestyle factors like lactovegetarian diet or drug abuse also correlate with higher risk of erosive diseases (Duxbury, 1993; Linkosalo and Markkanen, 1985; Miller, 1907; Petersen and Gormsen, 1991; Ten Bruggen Cate, 1986; Tuominen and Tuominen, 1991; Zero, 1996).

Anorexia nervosa, bulimia nervosa and gastrointestinal diseases with frequent regurgitation of gastric contents are intrinsic factors that may cause dental erosion (Järvinen et al, 1988; Milosevic and Slade, 1989; Tylenda et al, 1991). It is also assumed that behavioral and biological factors, such as tooth position, soft tissue anatomy and quality of dental hard tissues may exert an influence on the development of erosions. Moreover, salivary factors like composition, buffer capacity and flow rate are able to modify the development and progression of dental erosion. It has also been suggested that the presence of both acquired pellicle and microbiological plaque on tooth surfaces may impair the formation of erosive lesions (Amaechi et al, 1999; Gudmundsson et al, 1995; Hannig and Balz, 1999; Johansson et al, 2002; Meurman and Ten Cate, 1996).

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The acidic attack leads to demineralization and softening of the tooth surface. As a sequela, the tooth surface is more susceptible to mechanical influences, such as abrasion and attrition (Sognnaes, 1963). Initially, the enamel demineralizes and dissolves without clinically detectable softening. Loss of substance reaching the dentin can occur in combination with abrasive processes.

The prevention of caries through the use of fluoride has played an important role in dentistry for more than half a century. Today, there is evidence that fluoride mainly works topically by promoting remineralization and impeding the demineralization of tooth hard tissue. The mode of action of fluoride and its pharmacodynamic and pharmacokinetic properties has been intensively studied (Marthaler, 1990).

This article provides a survey of the effects of fluoride on prevention, development and progression of erosive demineralization in human enamel and dentin.

PATHOLOGY OF EROSIVE LESIONS

Erosion has been described as: "The physical result of a pathologic chronic loss of dental hard tissue that is chemically etched away from the tooth surface." (Ten Cate and Imfeld, 1996). This chemical attack may be induced by acid substances and/or chelators without bacterial development, and is accompanied by a demineralization and softening of the tooth surface. The demineralized surface is more prone to abrasion through mechanical friction by lip, cheek, tongue, food or toothbrushing. Solutions with a very low pH value may cause erosion. Demineralization is pronounced if the attack is of a long duration and repeated over time.

In previous *in vitro* and *in situ* studies it was proved that the contact of enamel and dentin with acidic solutions results in a decrease of microhardness (Attin et al, 1997; Lussi and Hellwig, 2001; Sorvari et al, 1994). The susceptibility of eroded enamel to abrasion can be correlated with Vickers microhardness values, thereby suggesting an over proportional increase of abrasion with decreasing hardness values (Attin et al, 1997).

In patients with severe dental erosion the enamel is often totally detached from the tooth, resulting in a vulnerable surface prone to further erosion and abrasion. Meurman et al (1991) could show that erosive acids can expose inner dentin structures by significant enlargement of the dentinal tubules, which may explain the painful sensitivity of teeth affected by erosions.

Erosive lesions show a characteristically histological pattern. The demineralized zone of the erosive lesion is comparable to the softened superficial layer observed in very early stages of incipient caries lesions (Arends and Christoffersen, 1986). The pseudo-intact surface layer, which is typical for established initial carious lesions, can not be detected by investigation with a polarizing microscope (Attin et al, 1997).

During an erosive attack, protons of the acidic agent attack the components of hydroxyapatite such as carbonate, phosphate and hydroxyl ions. This attack results in dissolution of the hydroxyapatite crystals with a subsequent release of calcium ions. Chelating agents like citric acid can bind the released calcium and can thereby increase the demineralization of the hydroxyapatite. Additionally, binding of salivary calcium ions may create undersaturation of saliva with respect to calcium. Due to these characteristics chelating agents are able to support loss of calcium from enamel.

Loss of the superficial enamel layer depends on the acid strength and the time of exposure to the acidic solution. After an exposure of 15 min. to an acidic beverage with a pH value of 2.84 the enamel demineralization amounted to a depth of 20 µm (Attin et al, 1997). Ultrastructural studies have shown that erosive lesions in prismatic enamel reveal characteristic demineralization patterns where either the prism cores or the interprismatic areas are dissolved, leading to a honeycomb structure (Meurman and Frank, 1991; Noack, 1989). Aprismatic enamel erodes in a highly irregular manner and is probably not as prone to erosive destruction as prismatic enamel (Meurman and ten Cate, 1996).

The demineralization of dentin is first apparent at the interface between inter- and peritubular dentin, and – with increasing exposure time – results in a hollowing and funneling of the tubules. The dentinal tubules become significantly enlarged, which explains why eroded teeth with areas of exposed dentin are more sensitive to external stimuli. Finally, the peritubular dentin is completely dissolved and the dentin shows a more porous structure (Meurman et al, 1991). The presence of a demineralized organic layer may hamper ionic diffusion influencing demineralization. Kleter et al (1994)

showed that in demineralized bovine root dentin with a layer of completely demineralized collagen, further mineral loss is increased in samples pretreated with collagenase. The demineralization rate decreased as the amount of demineralized organic matrix increased.

FLOURIDE – MODE OF ACTION

Fluoride is a well-researched agent in dentistry, although its mode of action regarding the development and progression of erosive lesions is not yet fully analyzed. In contrast, the anticarious action of fluoride has been researched in numerous studies.

The interaction between the fluoride ion and dental hard tissue is influenced by the fluoride concentrations, pH and the composition of the agents used in caries prevention.

Preeruptively, fluoride can be incorporated in enamel lattice, forming fluoride apatite or fluoridated hydroxyapatite (Luoma, 1986). This leads to a decreasing loss of mineral tissue in the event of a carious attack. Today, the interaction of fluoride with the enamel after eruption of the teeth is regarded as more important for the protection of mineral loss induced by demineralization.

Following the application of ionized fluoride, such as sodium fluoride, amine fluoride or tin fluoride a calcium fluoride-like deposition is formed on the enamel. The CaF_2-like material has been shown to persist on tooth surfaces for weeks or months under neutral conditions (Dijkman et al, 1983). Phosphate and proteins can be incorporated into the CaF_2-deposit (Christoffersen et al, 1988). The CaF_2-formation can be enhanced by increasing the fluoride concentration or exposure time and by lowering the pH of the topical agent (Saxegaard and Rølla, 1988). Using electron microscopy, Duschner et al (1997) showed that CaF_2-precipitate is not only formed on the tooth surface but also appears as a granular material in the enamel up to a depth of about 40 µm.

During a caries attack calcium fluoride-like material serves as a reservoir for the fluoride which facilitates the reprecipitation of minerals by forming fluoroapatite or fluorohydroxyapatite, thereby preventing further loss of mineral ions (Rølla et al, 1993). The stabilizing phosphate coating of the calcium fluoride globuli is dissolved at pH below 5.5, leading to the release of calcium and fluoride from the globuli (Rølla, 1988). Fluoride released from the CaF_2 -like material can be incorporated into the solid structure of the tooth resulting in a more stable and less soluble mineral (Shellis and Duckworth, 1994). Fluoride ions can also be detected in the aqueous phase surrounding the enamel crystallites (Arends and Christoffersen, 1990). Fluoride present in the liquid phase around the crystallites as well as free fluoride ions support remineralization by forming fluoride apatite or fluoridated hydroxyapatite even in the case of slightly acidic environmental conditions of the teeth (Ten Cate and Duijsters, 1983).

According to Ten Cate and Featherstone (1991) formation of fluorapatite and remineralization under acidic conditions depends on the availability of calcium, phosphate and free fluoride in the surrounding teeth. However, according to thermodynamical models remineralization at pH < 4.5 is regarded to be unlikely under physiological oral conditions and even where there is a very high fluoride concentration in the surrounding teeth (Ten Cate, 1997).

Considerable amounts of fluoride are also retained in dentin after topical application due to its porosity and water content (Ten Cate et al, 1995). CaF_2 -like material was detected in the peri- and intertubular dentin but also in intratubular regions (Laufer et al, 1981).

In deeper dentin layers, increased fluoride levels act as a fluoride reservoir (Hellwig, 1992).

It is well known that demineralized enamel is able to absorb more fluoride than non-demineralized tooth surfaces (Hellwig et al, 1987). Also, more calcium fluoride-like material is formed on eroded enamel than on sound or carious enamel (Attin et al, 2000).

Where erosion occurs, fluoride is primarily applied to stop the progression of the erosion by reducing the acid solubility of the tooth surface. Imfeld (1996) assumed that highly concentrated fluoride applications appear to be more suitable for treating erosive lesions.

The caries-inhibiting effects of fluoride applications have been confirmed for a long time. However, the effect of fluoride on the progression of erosive demineralization in human enamel and dentin has been not investigated in detail. Nonetheless, it could be assumed that similar to its anticariogenic properties, fluoride might also be an adjunct in the decreasing susceptibility of dental hard tissues to dental erosion.

PREVENTION AND TREATMENT OF EROSIVE WITH FLOURIDE

On the assumption that non-carious erosive lesions are becoming more significant in dentistry, preventive and restaurative therapeutic measures to deal with these erosions are increasingly needed.

Changes in dietary or behavioral patterns, or the reduction of the erosive potential of acidic agents may be considered as one possible strategy for arresting the development of erosions. Moreover, other strategies may be aimed at increasing enamel resistance, either by improving remineralization or through a reduction in the demineralization of dental hard tissues. Furthermore, chemical and/or physical protection of the tooth surface may help to avoid development of erosive lesions. Additionally, it seems to be important to minimize the abrasive influences of surface-softened dental hard tissue.

Eventually, the question arises whether fluoride treatment is able to prevent both erosion and further progressive mineral loss induced by acidic agents. Consequently, it is interesting whether admixtures of fluoride to acidic solutions, such as beverages, or the treatment of dental hard tissues with fluoride may exert an influence on dental erosive lesions.

ADMIXTURES OF FLOURIDE TO ACIDIC SOLUTIONS

Some *in vitro* tests have demonstrated that beverages containing monocalcium phosphate, sodium phosphate or calcium lactate caused less erosion than beverages without those components (Reussner et al, 1975; Beiraghi et al, 1989).

The effect of the addition of fluoride to acidic beverages is examined in several *in vitro* tests and animal experiments. Spencer and Ellis (1950) demonstrated that an admixture of 50 ppm fluoride to grapefruit juice reduced erosive lesions in rats. Sorvari et al (1988) also showed a reducing effect of 15 ppm fluoride in soft drinks (pH 3.2) in relation to erosive formation in rats.

Other authors confirmed the findings of Sorvari et al (1988) even in cases with lower fluoride concentrations. Thus, Restarski et al (1945) demonstrated that supplementing citric acid with 1 ppm fluoride decreased the amount of erosive lesions in molar surfaces of rats. Fuks et al (1973) demonstrated that the rate of erosion in molars of hamsters given a commercial grapefruit beverage supplemented with 1.9 ppm fluoride could be reduced by up to 30% when compared to teeth of hamsters fed with pure grapefruit juice. Shabat et al (1975) reported a significant decrease in the mean erosive score by increasing the fluoride supplement in soft drinks. Cola beverages supplemented with 1.9 or 5.2 ppm fluoride produced less erosion in molars than colas supplemented with 0.2 ppm fluoride. Analysis of the teeth showed that the concentration of fluoride was significantly higher in the surface of molars of rats drinking cola supplemented with 1.9 or 5.2 ppm fluoride compared with corresponding groups drinking fluoridated (1.7 or 5 ppm) water. This concurs with the study by Zipkin and McClure (1949) who reported that the addition of up to 5 ppm fluoride to citrate solutions (pH 6.3 – 6.5) given to rats as their only drinking fluid resulted in considerable protection of their molars against erosion. It is also interesting that commercially available beverages with higher fluoride concentration showed a smaller degree of surface softening in human enamel *in vitro* (Lussi et al, 1995).

The above mentioned findings seem to disagree with the results of the *in vitro* study of Larsen and Richards (2002). They pointed out that fluoride in a concentration excluding toxicologically side-effects added to drinks did not help to reduce the development of erosion. Larsen and Richards (2002) did not support the opinion that a topical fluoride treatment of the teeth has a noticeable effect on the erosive effect of these drinks, because the acidic beverages rapidly dissolved the calcium fluoride formed on the enamel surface.

Saturation with calcium fluoride reduces the *in vitro* development of erosions induced by drinks with pH above 3 by as much as 28%. Despite a total fluoride concentration of up to 20 ppm in drinks with pH below 3, the depth of erosions was unaffected by the admixed fluorides. Moreover, it should be emphasized that for toxicological reasons fluoride in drinks in such high concentrations is not feasible (Larsen and Richards, 2002). The threshold dose for children to develop fluorosis in permanent teeth amounts to 0.02 – 0.1 mg fluoride per kilogram of body weight (Forsman, 1977; Fejerskov et al, 1996). This means that children weighing 20 kg that drink 1 litre of juice per day supplemented with 5 – 50 ppm fluoride – i.e. as proposed in the study by Spencer and Ellis (1950) and Sorvari et al (1988) – have a high risk of developing fluorosis of the permanent teeth.

In conclusion, it seems unlikely that supplementing highly acidic drinks with fluoride in a concentration excluding toxicologically side-effects provides a preventive effect against erosion in human enamel.

TOPICAL APPLICATION OF FLOURIDES

Toothpastes

Several *in vitro* studies have investigated the effect of the topical application of fluorides, such as toothpastes, rinsing solutions and varnishes, on human enamel and dentin erosion (Gedalia et al, 1992; Munoz et al, 1999).

Rytömaa et al (1989) reported that some oral care products exhibit a low pH value. On one hand, oral care products with low pH can aid erosive damage; on the other hand a low pH enhances the chemical stability of some fluoride components. Furthermore, a lower pH favors the incorporation of fluoride into enamel crystals and the precipitation of calcium fluoride on the tooth surface. This deposited fluoride fraction is able to enhance remineralization after an erosive attack.

In vitro, an increase of hardness of intact enamel after topical application of fluoridated toothpaste has been reported (Munoz et al, 1999). Enamel treatment with fluoridated toothpastes reduces the drop in enamel hardness induced by acidic drink exposure (Munoz et al, 1999). Profilometric measurements by Davis and Winter (1977) revealed that a single application of a fluoridated toothpaste (0.8% sodium monofluorophosphate in calcium carbonate) administrated before an erosive attack reveals a protective effect of about 20% in enamel. Lussi and Jaeggi (2001) reported that even a fluoride-free toothpaste produced a significant reduction of hardness and thus indicated an erosive potential.

Recently, it was demonstrated that the salivary pellicle formed on enamel provides protection against erosive destruction to the underlaying enamel (Hannig and Balz, 1999). Toothbrushing before consumption of erosive food and beverages can lead to the loss of the acquired pellicle and can therefore increase the risk of erosive progression.

Nevertheless, treatment of already existing erosive lesions with fluoridated toothpaste also increases the hardness of erosively softened enamel (Munoz et al, 1999). These findings were confirmed by Bartlett et al (1994) who reported less tooth wear by toothbrushing with fluoridated toothpaste (1234 ppm) than with non-fluoridated toothpaste.

An *in vitro*-study by Ganss et al (2001) showed that fluoridated toothpaste (0.15%) reduced erosion induced mineral loss in both enamel and dentin. However, application of fluoridated toothpaste was less effective compared to intensive additional fluoridation with fluoridated toothpaste and fluoride solution and gel. It is important to note that, when using toothbrushing as a vehicle for fluoridation, at least 60 minutes should elapse before toothbrushing of the softened enamel surface (Attin et al, 2001).

Fluoride Gels/Rinses/Varnishes

Fluoridated solutions also appear to have an inhibiting effect on the progression of erosion.

Salivary remineralization as a natural reparative pathway is a well documented defense mechanism against demineralization. Mouthwashes containing ionic fluoride together with calcium and phosphate ions from saliva are able to enhance remineralization of enamel (White, 1987).

Despite acidic pH values, fluoridated mouth rinses and gels are able to increase the hardness of enamel as demonstrated by Lussi and Jaeggi (2001) and Attin et al (1999). An investigation by Jones et al (2002) found that a reduced depth of erosive demineralization of enamel occurred with the increased frequency of treatment with acidulated phosphate fluoride (1.23%). Treatment of enamel erosions with an acidified fluoride gel results in a higher abrasion resistance compared to gels which are either unfluoridated or neutral (Attin et al, 1999). However, pretreatment of enamel with carbamide peroxide bleaching solution before fluoridation does not induce a higher fluoride uptake or a better resistance against erosion in enamel (Burgmaier et al, 2002).

Additionally, fluorides admixed to carbamide peroxide gels are also not able to reduce the susceptibility of enamel to erosion (Attin et al, 2003)

In situ, microhardness of the enamel surface was determined following exposure to an acidic beverage and after mastication of Cheddar cheese with and without a Meridol® (0.025% F^-) prerinse. Thus, an increase in microhardness could be reported, and this effect was increased by the additional use of a fluoridated oral rinse. However, initial hardness of intact enamel surface could not be

re-established using these measures (Gedalia et al, 1992; Gedalia et al, 1996).

Amaechi and Higham (2001) reported that long-term application of a solution containing 0.022 ppm fluoride on enamel specimens remineralized early enamel erosions, which were produced through immersion in orange juice. Sorvari et al (1994) demonstrated a reduction of the erosive capacity of a cola beverage after treatment with a fluoride solution (1.2%) for 48 hours or a fluoride varnish (2.26%) for 24 hours. Larsen and Richards (2002) suggested that the amounts of calcium fluoride formed during a 48-hour continuous fluoride exposure seemed to be clinically unrealistic since administration of fluoride rinses may not exceed 2 min. *in vivo*. Furthermore, they pointed out that acidic beverages running over the teeth will rapidly dissolve accessible calcium fluoride and remove the remaining traces of a previously applied topical fluoride treatment.

Other authors demonstrated an increase of abrasion resistance of eroded dentin specimens after immersion in fluoridated solution for one minute (Attin et al, 1998). They reported a significantly better protection against further demineralization through an acidic drink with a high-concentration (2000 ppm) rinse in contrast to a low-concentration (250 ppm) rinse. Susceptibility to abrasion of the eroded dentin specimens treated with high-concentration solution did not differ significantly from the uneroded specimens. Ganss et al (2001) also proved that formation of erosive lesions could be influenced by fluoride applications. The authors reported a decrease of erosive mineral loss of enamel and dentin using intensive fluoridation after erosive attacks by citric acid (pH 2.3). Topical application of toothpaste (0.15% F^-, 3 x 5 min/daily) in combination with a fluoride rinse (0.025% F^-, 3 x 5 min/daily) and a fluoride gel (1.25% F^-, 1x5 min/on alternate days) reduced the progression of erosion in enamel. The different amount of enamel mineral loss between the fluoridated groups and the control group corresponded with the thickness of the calcium fluoride-like layer. Ganss et al (2001) suggested that the CaF_2 -like layer provided some additional mineral to be dissolved during an acid attack before the underlying enamel is attacked. Futhermore, they speculated that frequent and intensive fluoride applications lead to formation of stable CaF_2 -like deposit due to adsorption of salivary phosphates and proteins.

New tests revealed the effects of titanium tetrafluoride on artificial enamel lesion *in vitro*.

Titanium tetrafluoride (0.32%F) was found to be more effective than other fluoride agents (Duraphat 2.26% F^-, Elmex 1.25% F^-) in preventing artificial enamel lesion formation (Tezel et al, 2002). Moreover, the application of titanium tetrafluoride resulted in the formation of an acid resistant layer on enamel (Büyükylmaz et al, 1997). Therefore, studies of the effect of low-concentrated titanium tetrafluoride applications on erosive lesions appear to be interesting.

In contrast to enamel, intensive fluoridation of dentin was almost completely effective for inhibiting the progression of erosions (Ganss et al, 2001). The erosive loss of dentin was high at the beginning and continued with somewhat lower dissolution rates. Previous studies had proved that the presence of an organic layer on demineralized dentin decreases demineralization (Kleter et al, 1994). This layer may not only function as a diffusion barrier but also exhibits buffering properties and serves as a polar membrane that modifies the diffusion process. Ganss et al (2001) speculated that a sufficiently thick organic surface layer with buffering properties is able to prevent mineral loss of dentin in the presence of high-fluoride concentrations.

SEM studies have revealed that the dentinal tubules are enlarged as a result of an erosive attack (Meurman et al, 1991). It is therefore conceivable that penetration of fluoride into dentin is facilitated in eroded dentin.

CONCLUSION

The results of Ganss et al (2001) and Attin et al (1998, 1999) suggest that the prolonged application of highly concentrated fluoride agents has a positive effect on the prevention and progression of erosion. Modification of erosive products by the addition of fluoride seems to be of inferior importance for reducing the demineralizing capacity of these products.

Despite increasing abrasion resistance through the use of fluoridated toothpaste, patients should abstain from toothbrushing prior to erosive attacks, because very meticulous toothbrushing resulted in the loss of the acquired pellicle. This aspect seems to be important since the acquired pellicle may act as a barrier against erosive lesion and may therefore protect the underlying enamel surface against demineralization (Hannig and Balz, 1999; Meurman and Frank, 1991b).

Patients should therefore be advised to avoid toothbrushing for at least one hour after consumption of acidic beverages so as to minimize tooth substance loss through toothbrush abrasion. This delay should allow for rehardening of the softened eroded surface supported by saliva.

Fluoride application using oral rinses, gels or varnishes seems to be recommendable before or after erosive attacks to increase abrasion resistance and decrease erosive progression. In particular, erosion reaching the dentin can be treated more effectively than enamel through the use of highly-concentrated topical fluoride agents (Ganss et al, 2001).

It should also be added that hypersensitivity caused by erosive defects can be beneficially treated by the use of fluoride varnishes (Addy and Dowell, 1983).

REFERENCES

1. Addy M, Dowell P. Dentine hypersensitivity – a review. Clinical and *in vitro* evaluation of treatment agents. J Clin Periodontol 1983;10:351-363.
2. Amaechi BT, Higham SM. *In vitro* remineralization of eroded enamel lesions by saliva. J Dent 2001;29:371-376.
3. Amaechi BT, Higham SM, Edgar WM, Milosevic A. Thickness of acquired salivary pellicle as a determinant of the sites of dental erosion. J Dent Res 1999;78:1821-1828.
4. Arends J, Christoffersen J. The nature of early caries lesions in enamel. J Dent Res 1986;65:2-11.
5. Arends J, Christoffersen J. Nature and role of loosely bound fluoride in dental caries. J Dent Res 1990;69:601-605.
6. Attin T, Hilgers RD, Hellwig E. Einfluss von Muzin auf die Entstehung von Schmelzerosionen. Deutsch Zahnärztl Z 1996;51:241-245.
7. Attin T, Koidl U, Buchalla W, Schaller HG, Kielbassa AM, Hellwig E. Correlation of microhardness and wear in differently eroded bovine dental enamel. Arch Oral Biol 1997;42: 243-250.
8. Attin T, Zirkel C, Hellwig E. Brushing abrasion of eroded dentin after application of sodium fluoride solutions. Caries Res 1998;32:344-350.
9. Attin T, Deifuss H, Hellwig E. Influence of acidified fluoride gel on abrasion resistance of eroded enamel. Caries Res 1999;33:135-139.
10. Attin T, Dumont B, Buchalla W. Fluoride uptake in carious, eroded and sound enamel after application of a 2000 ppm fluoride solution. Dtsch Zahnärztl Z 2000;55:455-460.
11. Attin T, Knöfel S, Buchalla W, Tütüncü R. *In situ* evaluation of different remineralization periods to decrease brushing abrasion of demineralized enamel. Caries Res 2001;35: 216-222.
12. Attin T, Kocabiyik M, Buchalla W, Hannig C, Becker K. Susceptibility of enamel surfaces to demineralization after application of fluoridated carbamide peroxide gels. Caries Res 2003;37:93-99.
13. Bartlett DW, Smith BG, Wilson RF. Comparison of the effect of fluoride and non-fluoride toothpaste on teeth wear *in vitro* and the influence of enamel fluoride concentration and hardness of enamel. Br Dent J 1994;176:346-348.
14. Behrendt A, Oberste V, Wetzel WE. Fluoride concentration and pH of iced teas products. Caries Res 2002;36:405-410.
15. Beiraghi S, Atkins S, Rosen S, Wilson S, Odom J, Beck M. Effect of calcium lactate in erosion and S. mutans in rats when added to Coca-Cola. Pediatr Dent 1989;11:312-315.
16. Burgmaier GM, Schulze IM, Attin T. Fluoride uptake and development of artificial erosions in bleached and fluoridated enamel *in vitro*. J Oral Rehabil 2002;29:799-804.
17. Büyükylmaz T, Øgaard B, Rølla G. The resistance of titanium tetrafluoride-treated human enamel to strong hydrochloric acid. Eur J Oral Sci 1997;105:473-477.
18. Christoffersen J, Christoffersen MR, Kibalczyc W, Perdok WG. Kinetics of dissolution and growth of calcium fluoride and effects of phosphate. Acta Odontol Scand 1988;46:325-336.
19. Davis WB, Winter PJ. Dietary erosion of adult dentine and enamel. Protection with fluoride toothpaste. Br Dent J 1977; 143:116-119.
20. Dijkman AG, de Boer P, Arends J. *In vivo* investigation on the fluoride content in human enamel after topical applications. Caries Res 1983;17:392-402.
21. Duschner H, Götz H, Øgaard B. Fluoride induced precipitates on enamel surface and subsurface areas visualized by electron microscopy and confocal laser scanning microscopy. Eur J Oral Sci 1997;105:466-472.
22. Duxbury AJ. Ecstasy – dental implications. Br Dent J 1993; 10:175-38.
23. Fejerskov O, Richards A, Baelum V. Dose-response and dental fluorosis. In: Fejerskov O, Ekstrand J, Burt BA. Fluoride in Dentistry. Copenhagen: Munksgaard 1996;153-166.
24. Forsman B. Early supply of fluoride and enamel fluorosis. Scand J Dent Res 1977;85:22.
25. Fuks A, Anaise J, Westreich V, Gedalia I. Effect of fluoride supplementation of a citrus beverage on the erosion rate of molars of hamsters. J Dent Res 1973;52:1149.
26. Ganss C, Klimek J, Schäffer U, Spall T. Effectiveness of two fluoridation measures on erosion progression in human enamel and dentine *in vitro*. Caries Res 2001;35:325-330.
27. Gedalia I, Davidov I, Lewinstein I, Shapira L. Effect of hard cheese exposure, with and without fluoride prerinse, on the rehardening of softened human enamel. Caries Res 1992; 26:290-292.
28. Gedalia I, Braustein E, Lewinstein I, Shapira L, Ever-Hadani P, Sela M. Fluoride and hard cheese exposure on etched enamel in neck-irradiated patients *in situ*. J Dent 1996;24: 365-368.
29. Grenby TH, Phillips A, Desai T, Mistry M. Laboratory studies of the dental properties of soft drinks. Br J Nutr 1989;62: 451-464.
30. Gudmundsson K, Kristleifson G, Theodors A, Holbrook WP. Tooth erosion, gastroesophageal reflux, and salivary buffer capacity. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79:185-189.
31. Hannig M, Balz M. Influence of *in vivo* formed salivary pellicle on enamel erosion. Caries Res 1999;33:372-379.
32. Hellwig E, Klimek J, Wagner H. The influence of plaque on reaction mechanism of MFP and NaF *in vivo*. J Dent Res 1987;66:46-49.

33. Hellwig E. Fluoride retention in dentin after topical application of aminefluoride. J Dent Res 1992;71:1558-1560.
34. Imfeld T. Prevention of progression of dental erosion by professional and individual prophylactic measures. Eur J Oral Sci 1996;104:215-220.
35. Järvinen VK. Meurman JH, Hyavärinen H, Rytömaa I, Murtomaa H. Dental erosion and upper gatrointestinal disorders. Oral Surg Oral Med Oral Pathol 1988;65:298-303.
36. Johansson AK, Lingstrom P, Birkhed D. Comparison of factors potentially related to the occurrence of dental erosion in high- and low-erosion groups. Eur J Oral Sci 2002;110:204-211.
37. Jones L, Lekkas D, Hunt D, McIntyre J, Rafir W. Studies on dental erosion: An *in vitro-in vivo* model of endogenous dental erosion-its application to testing protection by fluoride gel application. Aust Dent J 2002;47:304-308.
38. Kleter GA, Damen JJ, Everts V, Niehof J, Ten Cate JM. The influence of the organic matrix on demineralization of bovine root dentin *in vitro*. J Dent Res 1994;73:1523-1529.
39. Larsen MJ, Nyvad B. Enamel erosion by some soft drinks and orange juices relative to their pH, buffering effect and contents of calcium phosphate. Caries Res 1999;33:81-87.
40. Larsen MJ. Prevention by means of fluoride of enamel erosion as caused by soft drinks and orange juice. Caries Res 2001; 35:229-234.
41. Larsen MJ, Richards A. Fluoride is unable to reduce dental erosion from soft drinks. Caries Res 2002;36:75-80.
42. Laufer B, Mayer I, Gedalia I, Deutsch D, Kaufman HW, Tal M. Fluoride-uptake and fluoride-residual of fluoride-treated human root dentine *in vitro* determined by chemical, scanning electron microscopy and X-ray diffraction analyses. Arch Oral Biol 1981;26:159-163.
43. Linkosalo E, Markkanen H. Dental erosions in relation to lactovegetarian diet. Scand J Dent Res 1985;93:436-441.
44. Luoma H. Fluoride, magnesium and dental caries. Current aspects. Proc Finn Dent Soc 1986;82:290-297.
45. Lussi A, Jaeggi T, Schärer S. The influence of different factors on *in vitro* enamel erosion. Caries Res 1993;27:387-393.
46. Lussi A, Jaeggi T, Jaeggi-Schärer S. Prediction of the erosive potential of some beverages. Caries Res 1995;29:349-354.
47. Lussi A, Hellwig E. Erosive potential of oral care products. Caries Res 2001;35:52-56.
48. Lussi A, Jaeggi T. The erosive potential of various oral care products compared to foodstuffs and beverages. Schweiz Monatsschr Zahnmed 2001;111:274-281.
49. Marthaler TM. Cariostatic efficacy of the combined use of fluorides. J Dent Res 1990;69:797-800.
50. Meurman JH, Drysdale T, Frank RM. Experimental erosion of dentin. Scand J Dent Res 1991;99:457-462.
51. Meurman JH, Frank RM. Progression and surface ultrastructure of *in vitro* caused erosive lesions in human and bovine enamel. Caries Res 1991a;25:81-87.
52. Meurman JH, Frank RM. Scanning electron microscopic study of the effect of salivary pellicle on enamel erosion. Caries Res 1991b;25:1-6.
53. Meurman JH, ten Cate JM. Pathogenesis and modifying factors of dental erosion. Eur J Oral Sci 1996;104:199-206.
54. Miller WD. Experiments and observations on the wasting of tooth tissue variously designated as erosion, abrasion, chemical abrasious denudation, etc. Dent Cosmos 1907; 49:1-23.
55. Milosevic A, Slade PD. The orodental status of anorexics and bulemics. Br Dent J 1989;167:66-70.
56. Munoz CA, Feller R, Haglund A, Triol CW, Winston AE. Strengthening of tooth enamel by a remineralizing toothpaste after exposure to an acidic soft drink. J Clin Dent 1999;10: 17-21.
57. Noack MJ. REM-Untersuchungen an Erosionen der Zahnhartsubstanzen *in vivo*. Dtsch Zahnärztl Z 1989;44:517-520.
58. Nunn JH, Gordon PH, Morris AJ, Pine CM, Walker A. Dental erosion – changing prevalence? A review of British National children's surveys. Int J Paediatr Dent 2003;13:98-105.
59. Petersen PE, Gormsen C. Oral conditions among German battery factory workers. Community Dent Oral Epidemiol 1991; 19:104-106.
60. Petersson HG, Bratthall D. The caries decline: a review of reviews. Eur J Oral Sci 1996;104:436-443.
61. Restarski JS, Gortner RA, McCay CM. Effect of acid beverages containing fluorides upon the teeth of rats and puppies. J Am Dent Assoc 1945;32:668-675.
62. Reussner GH, Coccodrilli G Jr, Thiessen R Jr. Effects of phosphates in acid-containing beverages on tooth erosion. J Dent Res 1975;54:365-370.
63. Rølla G. On the role of calcium fluoride in the cariostatic mechanism of fluoride. Acta Odontol Scand 1988;46: 341-345.
64. Rølla G, Øgaard B, Cruz Rde A. Topical application of fluorides on teeth. New concepts of mechanisms of interaction. J Clin Periodontol 1993;20:105-108.
65. Rytömaa I, Meurman JH, Franssila S, Torkka H. Oral hygiene products may cause dental erosion. Proc Finn Dent Soc 1989;85:161-166.
66. Saxegaard E, Rølla G. Fluoride acquisition on and in human enamel during topical application *in vitro*. Scand J Dent Res 1988;96:523-535.
67. Shabat E, Anaise J, Westreich V, Gedalia I . Erosion and fluoride content in molar surfaces of rats that drank a cola beverage with and without fluoride. J Dent Res 1975;54:426.
68. Shellis RP, Duckworth RM. Studies on the cariostatic mechanisms of fluoride. Int Dent J 1994;44:263-273.
69. Sognnaes RF. Dental hard tissue destruction with special reference to idiopathic erosions. In: Sognnaes RF (Ed.), Mechanisms of hard tissue destruction. Washington: Association of the advancement of science 1963;91-153.
70. Sorvari R, Kiviranta I, Luoma H. Erosive effects of a sport drink mixture with and without addition of fluoride and magnesium on the molar teeth of rats. Scand J Dent Res 1988; 96:226-231.
71. Sorvari R, Meurman JH, Alakuijala P, Frank RM. Effect of fluoride varnish and solution on enamel erosion *in vitro*. Caries Res 1994;28:227-232.
72. Sorvari R: Effects of various sports drink modifications on dental caries and erosion in rats with controlled eating and drinking pattern. Proc Finn Dent Soc 1989;85:13-20.
73. Spencer IJ, Ellis LN. The effect of fluoride and grapefruit juice on the etching of teeth. J Nutr 1950;42:107-115.
74. Ten Bruggen Cate HJ. Dental erosion in industry. Br J Ind Med 1968;25:249-266.
75. Ten Cate JM, Duijsters PP. Influence of fluoride in solution on tooth demineralization. II. Microradiographic data. Caries Res 1983;17:513-519.
76. Ten Cate JM, Featherstone JDB. Mechanistic aspects of the interactions between fluoride and dental enamel. Crit Rev Oral Biol Med 1991;2:283-296.

77. Ten Cate JM, Buijs MJ, Damen JJ. pH-cycling of enamel and dentin lesions in the presence of low concentration of fluoride. Eur J Oral Sci 1995;103:362-367.
78. Ten Cate JM, Imfeld T. Dental erosion, summary. Eur J Oral Sci 1996;104:241-244.
79. Ten Cate JM. Review on fluoride, with special emphasis on calcium fluoride mechanisms in caries prevention. Eur J Oral Sci 1997;105:461-465.
80. Tezel H, Ergucu Z, Onal B. Effects of topical fluoride agents on artificial enamel lesion formation *in vitro*. Quintessence Int 2002;33:347-352.
81. Thomas AE. Further observations on the influence of citrus fruit juices on human teeth. N Y State Dent 1957;23: 424-430.
82. Tuominen M, Tuominen R. Tooth surface loss and associated factors among factory workers in Finland and Tanzania. Community Dent Health 1992;9:143-150.
83. Tylenda CA, Roberts MW, Elin RJ, Li SH, Altemus M. Bulemia nervosa: its effect on salivary chemistry. J Am Dent Assoc 1991;122:37-41.
84. West NX, Hughes JA, Addy M. Erosion of dentin and enamel *in vitro* by dietary acids: the effect of temperature, acid character, concentration and exposure time. J Oral Rehabil 2000; 27:875-880.
85. White DJ. Reactivity of fluoride dentifrices with artificial caries. I. Effects of early lesions: F uptake, surface hardening and remineralization. Caries Res 1987;21:126-140.
86. Zero DT. Etiology of dental erosion – extrinsic factors. Eur J Oral Sci 1996;104:162-177.
87. Zipkin J, McClure FJ. Salivary citrate and dental erosion. J Dent Res 1949;28:613-626.