ACTA
STOMATOLOGICA
CROATICA
www.ascro.hr

Acta Stomatol Croat. 2009;43(3):165-187.

PREGLEDNI RAD
REVIEW

Betul Kargul, Meltem Bakkal

Prevalencija, etiologija, rizični čimbenici, dijagnostika i preventivne mjere kod erozije zuba: pregled literature (I. i II. dio)

Prevalence, Etiology, Risk Factors, Diagnosis, and Preventive Strategies of Dental Erosion: Literature Review (Part l & Part II)

Stomatološki fakultet, Odjel dječje stomatologije Marmarskog sveučilišta, Istambul, Turska
Marmara University, Dental School, Department of Pediatric Dentistry, Istanbul, Turkey

Sažetak
Erozija zuba trajan je gubitak tvrdoga zubnog tkiva, a uzrokuju je kemijska sredstva (kiseline i/ili kelatori), ali u taj proces nisu uključene bakterije. Takav "kiseli napad" potpuno uništava tvrda zubna tkiva, jer progresivno omekšava površinu. Ona je nakon toga osjetljiva na djelovanje mehaničkih sila poput, primjerice, abrazije koja inače nema gotovo nikakav utjecaj na zdravo tvrdo zubno tkivo. Iako je utjecaj erozije često dominantan, kemijski i mehanički procesi su pojedinačni ili zajednički. Njezina je etiologija mnogostruka i nije u cijelosti poznata. U studijama bi se izričito trebalo odrediti što su erozija, atricija i abrazija te identificirati etiološke čimbenike. Naime, postoje različiti uzroci i etiologije erozivnih stanja. Budući da je erozivno trošenje zuba multifaktorijalno i preventivne se mjere moraju temeljiti na kemijskim i biološkim čimbenicima te na ponašanju pacijenta, jer je sve to uključeno u etiologiju i patogenezu erozije. I klinički stomatolozi sve češće shvaćaju da moraju bolje poznavati etiologiju i terapiju trošenja zuba, budući da im dolazi sve više starijih pacijenata sa svojim prirodnim zubima, ali jako istrošenima. Klinička terapija erozije trebala bi se usredotočiti na prevenciju, a tek nakon toga na moguće restorativne zahvate. Kako bi se progresija erozije prevenirala i spriječila, mora se pravodobno otkriti.

Zaprimljen: 7. kolovoza 2009.
Prihvaćen: 25. kolovoza 2009.
Adresa za dopisivanje
Prof Dr. Betul Kargul
Marmara University
Dental School
Department of Pediatric Dentistry
Güzelbahçe Büyükçiftlik Sokak
No.6 3436
Istanbul, Turkey
Tel:0212 231 91 20/109
Fax: 0 212 246 52 47
bkargul@marmara.edu.tr

Ključne riječi
zub, erozija; zub, abrazija; zub, atricija

Uvod

Zubna erozija je trajan gubitak tvrdoga zubnog tkiva u kemijskim procesima (izlaganje kiselinama i/ili kelatorima), ali u taj proces nisu uključene bakterije (1). Naime, progresivnim omekšavanjem površine (2) kiseline uništavaju tvrdo zubno tkivo. Nakon toga ono je osjetljivije na djelovanje mehaničkih sila kao što je, primjerice, abrazija (3) koja inače nema gotovo nikakav učinak na zdravo tvrdo zubno tkivo (4). Kemijski i mehanički procesi mo-

Introduction

Dental erosion is defined as irreversible loss of dental hard tissue by a chemical process (acid acid and/or chelation exposure) that does not involve bacteria (1). The acidic attack leads to an irreversible loss of dental hard tissue, which is accompanied by a progressive softening of the surface (2). This softened zone is more susceptible to mechanical forces, such as abrasion (3), which in turn have little or no effect on sound dental hard tissues (4).

gu biti pojedinačni ili zajednički, premda je učinak erozije dominantan (5). Klinički rana erozija cakline izgleda poput glatke, svilenkaste i sjajne površine (Slika 1.). Eroziji je također svojstven greben cakline koji odvaja područje defekta od marginalne gingive. U daljnjoj progresiji okluzalne erozije počinju se uočavati karakteristične izdubljene kvržice (Slika 2.), a ispuni se izdižu iznad okolne zubne strukture. Za okluzalnu eroziju svojstvene su zaobljene kvržice i udubine (6), (Slika 3.).

Prevalencija

U studijama u kojima se obrađivala prevalencija stručnjaci su se koristili različitim indeksima i mjerenjima te su često ocjenjivali opće trošenje zuba, a ne samo eroziju. Većina ih je obavljena u Europi pa treba biti oprezan u primjeni dobivenih rezultata ako je riječ o općoj populaciji i onoj u SAD-u. No, rezultati ipak daju približnu procjenu problema u općoj populaciji. Tijekom jednog istraživanja u Švicarskoj bila je pregledana 391 odrasla osoba u vlastitu domu (7) te je kod sudionika u dobi od 26 do 30 godina bilo otkriveno 7,7% vestibularnih erozivnih lezija i 29,9% okluzalnih sa zahvaćenim dentinom. Bilo je provedeno i nekoliko istraživanja prevalencije erozija na djeci. Zubna erozija uvrštena je u Velikoj Britaniji godine 1993. (8) u prvi pregled djece radi državnoga istraživanja o zdravlju njihovih zuba. Više od polovice pregledanih – njih 17.061 (u dobi od 5 i 6 godina) imalo je erozivne promjene, a kod 25% bio je zahvaćen dentin mliječne denticije. U skupini 11-godišnjaka gotovo 25% imalo je znakove erozije, a 2% zahvaćen dentin u miješanoj denticiji (9).

Caglar i suradnici ocjenjivali su prevalenciju, kliničku manifestaciju i etiologiju dentalne erozije kod djece. Pregledali su 153 dječaka i djevojčica u dobi od 11godina iz srednje klase u državnim školama u Istambulu. Zubna erozija bila je pronađena kod njih 28% (10).

Većina epidemiologije ispitivala se na djeci i adolescentima, a ne na odraslima te se vrijednost povezanosti između opsežnosti i starosti ponajprije temelji na zapažanjima kod tih mladih ljudi (11-14).

Mora se istaknuti da se etiologija u različitim zemljama različito objašnjava (15). Premda se u Velikoj Britaniji shvaća koliko je opasna zubna erozija, ne smije se zaboraviti ni na abraziju i atriciju, pa se najčešće rabi zajednički pojam - trošenje zuba (16). Stanje je manje jasno ako se uzmu u obzir patološ-

The chemical and mechanical processes can occur individually or together, although the effect of erosion is often dominant (5). Clinically, early enamel erosion appears as a smooth silky-shining glazed surface (Figure 1). Typical for erosions of the facial aspects of teeth is a ridge of enamel that separates the defect from the marginal gingiva. Further progression of occlusal erosion leads to a distinct grooving of the cusps (Figure 2) and restorations are rising above the level of the adjacent tooth surface. Occlusal erosion is characterized by rounded cusps and concavities (6), (Figure 3).

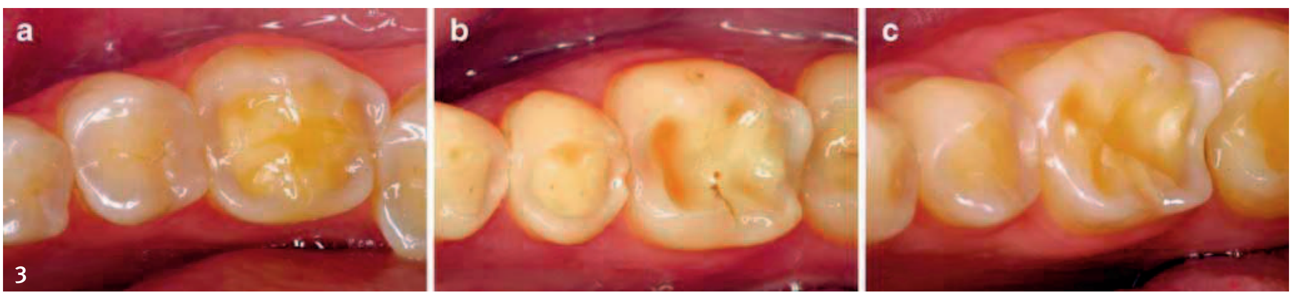
Prevalence

Prevalence studies have used different indices of measurement and often address tooth wear in general and not erosion specifically. In addition, most have been conducted in Europe, therefore, extrapolation of results to the US population must be guarded. However, these studies do give an approximation of the problem in general populations. In a Swiss study, 391 adults underwent dental examinations in their own homes (7). In subjects aged between 26 and 30 years, 7.7% had facial erosive lesions into dentin and 29.9% had occlusal tooth wear into dentin. Several prevalence studies have been conducted in children. Dental erosion was included in the examination for the first time in the 1993 National Survey of Child Dental Health conducted in the United Kingdom (8). Over half of 17,061 (5 and 6 year olds) children had erosion, 25% with dentinal involvement of the primary dentition. In the 11+ year age group, almost 25% had erosion, 2% with dentinal involvement in the mixed dentition (9). Caglar E. et al evaluated the prevalence, clinical manifestations, and etiology of dental erosion among children. A total of 153 healthy, 11-year-old children were sampled from a downtown public school in Istanbul, Turkey comprised of middle-class children. Twenty-eight percent of the children exhibited dental erosion (10).

Much of the epidemiology has been investigated in children and adolescents rather than adults and so the validity of any association between severity and age is based on clinical observations seen primarily in these young people (11-14).

However, different countries interpret the etiologies in different ways (15). In the United Kingdom, although the importance of erosion is acknowledged, the impact of abrasion and attrition is also recognised, and the term tooth wear is more commonly used (16). When pathological levels of wear are considered, the situation is less clear. There are insufficient data from epidemiological studies on ke razine trošenja. Na temelju istraživanja na odraslima nema dovoljno podataka za konačan odgovor. U slučajevima za koje imamo podatke, oni sugeriraju slična teška stanja trošenja kao i u svim dobnim skupinama te bi se moglo zaključiti da ne ovise o starosti (12, 14, 17). Zato se čini – iako je fenomen trošenja zuba povezan s godinama – da teška stanja nisu povezana (18). Jasno je iz literature da su potrebna nova istraživanja u populaciji, kako bi se točno razlučilo što su to erozija, atricija i abrazija te identificirali etiološki čimebnici.

adults to be definitive. But where data are present, it suggests similar proportions of severe levels of wear are observed in each age group, and it could be argued that this was independent of age (12, 14, 17). It seems, therefore, that although tooth wear is an age-dependant phenomenon, severe tooth wear is not (18). It is clear from a review of existing epidemiological studies that more population-based studies are needed. These studies should clearly delineate erosion, attrition, and abrasion with identification of etiologic factors.



Slika 1. Klinički prikaz erodirane zubne cakline. Erozija cakline kod trideset i šestogodišnje pacijentice, uzrokovana učestalom konzumacijom cola napitaka.Erozija je dijagnosticirana kao oštri rub na griznom bridu zubne krune.

Figure 1 Clinical appearance of tooth wear in enamel. Enamel erosion in a 36-year-old female patient caused by frequent consumption of a cola soft drink. Erosion can be detected by the presence of a wedge-shaped defect, which shows a sharp margin on the coronal portion.

Slika 2. Klinički prikaz eksponiranog zubnog dentina kod dvadesetpetogodišnjeg sportaša koji učestalo konzumira zakiseljene sportske napitke

Figure 2 Clinical appearance of tooth wear in dentin in a 25-year-old athlete due to frequent consumption of acidic sport drinks

Slika 3. (a–c) Tipičan oblik uznapredovale okluzalne erozije zuba 45 i 46 kod triju pacijenata: nestaje cijela okluzalna morfologija te se vide velika područja eksponiranog dentina (19)

Figure 3 (a–c) Typical pattern of advanced occlusal erosion of teeth 45 and 46 of three different patients: The whole occlusal morphology disappears, and extensive exposed dentinal areas are visible (19)

Slika 4. Erozija kod petogodišjeg djeteta zbog česte konzumacije kiseloga voćnog soka prije spavanja (9)

Figure 4 Erosion in a 5-year-old child caused by frequent consumption of an acidic fruit juice at bedtime (9)

Etiologija

Etiologija erozije je mnogostruka i nije u cijelosti objašnjena. Mnogobrojni su i čimbenici etiologije erozivnih stanja. Međudjelovanje kemijskih i bioloških čimbenika te ponašanje pacijenata prijeko je potrebno raščlaniti ako se želi objasniti zašto se erozije javljaju kod nekih češće, a kod nekih rjeđe iako su izloženi istim količinama kiselina u prehrani (19) (Tablica 1.).

Istaknimo - najvažniji izvori kiselina su u hrani, u onoj zakiseljenoj, ili u bezalkoholnim napitcima (20), te u želucu (želučana kiselina zbog povraćanja i refluksnih tegoba) (21). Trenutačno se smatra da je često konzumiranje kisele hrane i bezalkoholnih napitaka vrlo važan čimbenik u nastanku erozivnog trošenja (2), (Slika 4.).

Etiology

The etiology of erosion is multifactorial and not fully understood. There are different predisposing factors and aetiologies of the erosive condition. The interplay of chemical, biological and behavioural factors is crucial and helps explain why some individuals exhibit more erosion than others, even if they are exposed to the same acid challenge in their diets (19), (Table 1).

The most important sources of acids are those found in the diet, such as acidic foods and drinks (20) and those originated from the stomach, like gastric acids from regurgitation and reflux disorders (21). Currently, the increased consumption of acidic foods and soft drinks is becoming an important factor for the development of erosive wear (2), (Figure 4).

Tablica 1. Rizični čimbenici za nastanak zubne erozije (49)
Table 1 Risk Factors for Dental Erosion (49)

Čimbenici rizika • Risk Factors	
konzumacija citrusnog voća - agruma (više od dva na dan) • Citrus fruits intake (more than twice daily)	konzumacija sportskih napitaka (tjedno ili češće) • Sports drinks intake (weekly or more often)
konzumacija bezalkoholnih napitaka (4-6 ili više na tjedan) • Soft drinks consumed (4-6 or more per week)	konzumacija jabučnog octa (na tjedan ili češće) • Apple vinegar intake (weekly or more often)
poremećaji u prehrani • Eating disorder	povraćanje(tjedno ili češće) • Vomiting (weekly or more often)
bruksizam • Bruxism habit	ekstezivna atricija • Excessive attrition
ukupna nestimulirana slina (≤0,1 mL/min) • Whole saliva unstimulated flow rate (≤0.1 mL/min)	simptomi ili povijest gastroesophagealnog refluksa • Symptoms or history of gastroesophageal reflux disease

Kemijski čimbenici

Sa stajališta kemije, etiologija zubne erozije može se definirati kao kronično izlaganje zuba vanjskim i unutarnjim kiselinama, među kojima su usne tekućine nezasićene u odnosu prema zubnim mineralima (22, 23). U uvjetima *in vitro* bez fizičkoga utjecaja, zubi se centripetalno demineraliziraju (Slika 5.), a takav se način gubitka tkiva u ustima ne uočava pravodobno (24).

Zubna se erozija teško otkriva, posebice u ranim fazama dok su lezije male i lako se mogu previdjeti. Unutarnje (gastro-interstinalne) i vanjske (prehrambene i iz okoliša) kiseline glavni su etiološki čimbenici zubne erozije (25, 26).

Vanjski čimbenici

Uključuju demineraliziranu kiselu hranu, kao citrusno voće (agrume) i kisele napitke (27) te neke lijekove i preparate poput šumećega vitamina C, vitamina C u tabletama za žvakanje i napitke sa željezom. Jedan od čimbenika je i kiselina u zraku. Pri-

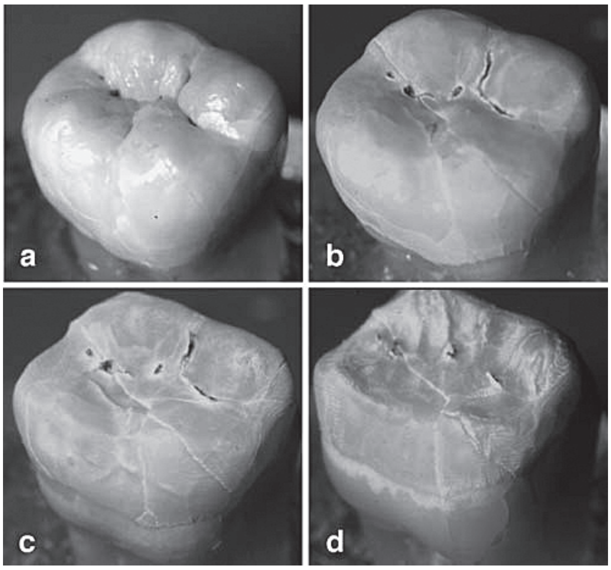
Chemical factors

From the chemical view, the aetiology of dental erosion can be defined as the chronic exposure of the teeth to extrinsic or intrinsic acids under the condition that the oral fluids are undersaturated with respect to tooth mineral (22, 23). Under in vitro conditions without physical impact, teeth demineralise centripetally (Figure 5), a feature of substance loss which is normally not observed in the mouth (24).

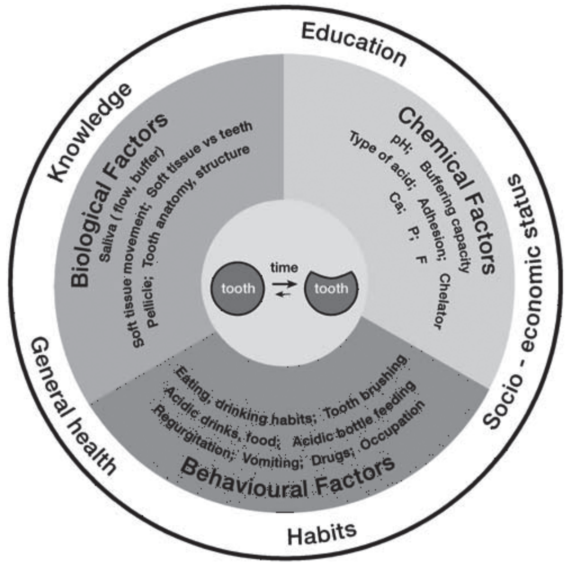
Dental erosion can be difficult to detect, especially in the early stages when lesions are subtle and can be easily overlooked. Acids of intrinsic (gastrointestinal) and extrinsic (dietary and environmental) origins are the main etiologic factors for dental erosion (25, 26).

Extrinsic factors

Extrinsic factors include demineralizing acidic foods-such as citrus fruits and acidic beverages (27) -and some medicines- such as effervescent vitamin C preparations, chewable vitamin C tablets, and iron tonics. Another extrinsic cause of erosion



Slika 5. Učinak nakon stalnog izlaganja trećeg molara u 10-postotnoj limunskoj kiselini. Očit je amorfan centripetalan oblik gubitka tkiva (a-intaktan zub, b-gubitak tkiva nakon 4 sata, c- nakon 8 sati i d – nakon 12 sati) (24)
Figure 5 Effect of the continuous exposure of a human third molar to 10% citric acid. The amorphous, centripetal tissue loss is obvious (a- unaffected tooth, b- tissue loss after 4, c- 8, and d- 12 h immersion time) (24)



Slika 6 Međudjelovanje različitih čimbenika na razvoj zubne erozije (2)
Figure 6 Interactions of the different factors for the development of dental erosion (2)

je nego što su bile prihvaćene mjere zaštite na radu, izlaganje kiselinama u zraku bilo je svakodnevno i uobičajeno u kemijskoj i metalskoj industriji. Danas se to može povezati i sa slobodnim aktivnostima, primjerice čestim plivanjem u bazenima s kloriranom vodom (28).

Unutarnji čimbenici

Uključuju često povraćanje zbog psihičkih poremećaja kao što su anoreksija i bulimija (29), ili vraćanje želučanog sadržaja zato što postoji neki poremećaj u gastro-interstinalnom traktu. Želučana kiselina ima mnogo niži pH od kritičnog pH-otapanja cakline, što znači da nakon dužeg razdoblja refluks u usnu šupljinu može prouzročiti velike gubitke zubnih struktura. Stomatolozi moraju posumnjati na eroziju induciranu refluksom ako se kod pacijenta pojave simptomi refluksne bolesti ili oblik erozije koji bi mogao upozoravati na unutarnje izlaganje kiselini (28). Jedan od dodatnih čimbenika za zubnu eroziju jest i loš protok sline, što naravno rezultira nedovoljnim ispiranjem i puferiranjem tih kiselina na zubnu strukturu (30). Na Slici 6. željelo se predstaviti multifaktorijalne čimbenike i etiologiju erozivnog stanja (2).

Ima dokaza da kisela hrana i napitci itekako utječu na početak procesa, ali pH-vrijednost tih namir-

is acids in the air breathed. Before effective occupational health measures were adopted, exposure to acids was common in the chemical and metal industries. Nowadays, exposure to extrinsic acid can also be associated with leisure activities such as frequent swimming in chlorinated pool water (28).

Intrinsic factors

Intrinsic causes of erosion include recurrent vomiting as a result of psychological disorders, e.g., in anorexia and bulimia (29), or regurgitation of gastric contents because of some abnormality in the gastrointestinal tract. The pH of stomach acid is much lower than the critical pH of enamel dissolution; therefore, reflux of stomach contents into the oral cavity over an extended period of time can cause severe loss of tooth structure. Dentists must maintain a high degree of suspicion for reflux-induced erosion whenever a patient displays symptoms of acid reflux disease or a pattern of erosion that suggests an intrinsic source of acid exposure (28). One important additional factor in dental erosion is low salivary flow, which, naturally, results in inadequate rinsing and buffering of demineralizing acids on tooth surfaces (30). Figure 6 is an attempt to reveal the multifactorial predisposing factors and aetiologies of the erosive condition (2).

nica ipak nije dovoljan prognostički pokazatelj da uzrokuje eroziju, kao što drugi procesi modificiraju erozivne procese. Ti su čimbenici kemijski (pH-vrijednosti, adhezija i kelacijske sposobnosti, kalcijski, fosfatni i fluoridni sadržaj) i biološki (protok i sastav sline, puferski kapacitet, stvaranje pelikule, sastav i anatomija zuba te mekih tkiva), a ne smijemo zaboraviti ni ponašanje (navike kod jela i pića, način života te pretjerano konzumiranje kiselina) (20).

Caglar i suradnici ustanovili su da je među djecom koja su pila narančin sok njih 32% imalo znakove erozije, a među onom koja su radije odabrala gazirana pića bilo je 40%. Od djece koja su jela voćne jogurte 36% je imalo erozije. Kod djece koja su profesionalno plivala u bazenima 60% je imalo znakove erozije. Multipla regresijska analiza nije upućivala na povezanost između erozije i mogućih navedenih izvora erozije (10).

Ponašanje i navike

Tijekom tzv. erozivnog napada i nakon njega u modificiranju trošenja zuba vrlo je važno obratiti pozornost na ponašanje. Kiseline koje unosimo u usnu šupljinu ponajprije djeluju na one zube s kojima dolaze u doticaj te možda i tijekom njihova daljnjeg odvođenja. U ovom je desetljeću način života znatno drugačiji, a promijenila se i količina te učestalost konzumiranja kisele hrane i pića. Tako je potrošnja bezalkoholnih pića u SAD-u u posljednjih dvadeset godina porasla za 300% (31, 32). Istraživanja kod djece i odraslih upućuju na to da je s erozijom i njezinom progresijom povezan broj obroka na dan, ako postoje i ostali rizični čimbenici (33, 34).

Nekoliko istraživanja *in vitro* i *in situ* pokazalo je da erozivni učinak kiselih napitaka i jela nije isključivo vezan s njihovom pH-vrijednosti, nego je snažan i utjecaj sadržaja minerala, titrabilna kiselost (puferski kapacitet) i sposobnost keliranja kalcija (35). Nizak stupanj zasićenosti u odnosu prema caklini ili dentinu rezultira inicijalnim površinskim lezijama koje slijede lokalno povećanje pH-vrijednosti te povećani mineralni sadržaj u tekućem sloju odmah uz površinu zuba. To što se pića i hrana pojačanim puferiranjem sline odupiru promjenama pH-vrijednosti, možda je vrlo važno za početak erozije kod djece. Može se reći da voćni jogurti nemaju erozivni potencijal (36).

Kargul i suradnici ističu da voćni jogurt *in vitro* ima najveći puferski kapacitet. Može se tvrditi da nije moguće inducirati eroziju cakline s bilo kojom vrstom jogurta (37).

There is evidence that acidic foodstuffs and beverages play a role in the development of erosion. However, the pH of a dietary substance alone is not predictive of its potential to cause erosion as other factors modify the erosive process. These factors are chemical (pKa values, adhesion and chelating properties, calcium, phosphate and fluoride content), behavioural (eating and drinking habits, life style, excessive consumption of acids) and biological (flow rate, buffering capacity, composition of saliva, pellicle formation, tooth composition, dental and soft tissue anatomy) (20).

Caglar E. et al evaluated the children who consumed orange juice, 32% showed erosion, while 40% who consumed carbonated beverages showed erosion. Of children who consumed fruit yogurt, 36% showed erosion. Of children who swam professionally in swimming pools, 60% showed erosion. Multiple regression analysis revealed no relationship between dental erosion and related erosive sources (10).

Behavioral factors

During and after an erosive challenge, behavioural factors play a role in modifying the extent of tooth wear. The manner that dietary acids are introduced into the mouth will affect which teeth are contacted by the erosive challenge and possibly the clearance pattern.

As lifestyles have changed through the decades, the total amount and frequency of consumption of acidic foods and drinks have also changed. Soft drink consumption in the USA increased by 300% in 20 years (31, 32). Studies in children and adults have shown that this number of servings per day is associated with the presence and the progression of erosion when other risk factors exist (33, 34).

Several in vitro and in situ studies show that the erosive potential of an acidic drink or foodstuff is not exclusively dependent on its pH value but is also strongly influenced by its mineral content, its titratable acidity ('the buffering capacity') and by the calcium-chelation properties (35). A low degree of undersaturation with respect to enamel or dentine leads to a very initial surface demineralization which is followed by a local rise in pH and increased mineral content in the liquid surface layer adjacent to the tooth surface. The capability of drinks and foods to resist pH changes brought about by salivary buffering may play an important role in the dental erosion process in children. It could be stated that fruit yogurt has no erosive potential (36).

Kargul B. et al. suggested that, in vitro, fruit yogurt has the greatest buffering capacity. It can be

Najčešći izvor kiselina je u bezalkoholnim napitcima poput *cole*. Njezina kariogenost je također veća od mlijeka i šećera (38). U usporedbi s karijesom čini se da je erozija jače povezana s bezalkoholnim pićima. Razorna snaga pića uglavnom je u njihovim pH-vrijednostima i puferskom kapacitetu. Tako su inicijalne vrijednosti pH-vrijednosti i puferskog kapaciteta nekih napitaka određene u nekim dosadašnjim istraživanjima. Gazirana pića imala su niži pH od voćnih sokova. Puferski kapacitet bio je sljedeći: voćni sokovi>gazirana pića s voćnom osnovom< gazirana pića bez voćne osnove (39,40)

Četkanje zuba dobar je način održavanja oralne higijene, ali je nakon toga postupka gubitak tvrdog tkiva znatno veći nego nakon same erozije (41, 42). Paste za izbjeljivanje također pojačano troše zdravu caklinu, kako erodiranog tako i zdravog dentina (43, 44).

Biološki čimbenici

Slina, stečena pelikula, zubna struktura i smještaj u odnosu prema mekim tkivima i jeziku u neposrednoj su vezi s razvojem erozije. Vrlo važan parametar je slina. Tijekom erozivnog napada djeluje nekoliko njezinih zaštitnih mehanizama: ona razrjeđuje i otplavlja erozivna sredstva iz usta, neutralizira i puferira kiselinu te usporava otapanje cakline preko zajedničkih ionskih učinaka kalcija i fosfata (45).

Stečena pelikula iz sline proteinski je sloj koji se brzo stvara na površini zuba nakon što je mehanički uklonjen četkanjem i zubnim pastama te drugim profilaktičnim postupcima ili kemijskim otapanjem. Taj organski sloj postaje vidljiv na zubnoj površini nakon samo nekoliko minuta izlaganja oralnom okolišu (46). U ranoj fazi stvaranja pelikule može se također uočiti enzimatska aktivnost (47). Smatra se da pelikula raste dok ne postigne ravnotežu između proteinske izgradnje i razgradnje - obično su to dva sata (48).

Stečena pelikula može štititi od erozije tako da djeluje kao difuzijska prepreka ili polupropusna selektivna membrana koja sprječava neposredan doticaj kiselina i površine zuba te tako smanjuje stupanj otapanja hidroksilapatita (48).

Postavljanje dijagnoze

Vrlo je teško postaviti dijagnozu u ranim fazama erozije jer nema gotovo nikakvih znakova, a simp-

stated that it is not possible to induce erosion on enamel with any type of yogurt (37).

The most frequent source of the acids is soft drinks like cola. It is also indicated that the cariogenicity of cola is higher than that of milk and sucrose (38). Compared with caries, dental erosion seems to have much stronger relationship with soft drinks. The erosive potential of drinks is mainly represented by their pH and the buffering capacity. In previous reports, the initial pH values of some soft drinks and their buffering capacities were determined. Carbonated drinks had lower pH than fruit juices. The buffering capacities are in the following order: fruit juices>fruit-based carbonated drinks>non-fruit-based carbonated drinks (39, 40).

Toothbrushing is a way to keep a good oral hygiene. Hard tissue loss after erosion and toothbrushing is significantly greater than erosion alone (41, 42). Whitening dentifrice also leads to significantly greater wear of sound enamel and of both eroded and sound dentine (43, 44).

Biological factors

Biological factors such as saliva, acquired pellicle, tooth structure and positioning in relation to soft tissues and tongue are related to dental erosion development.

A very important biological parameter is saliva. Several salivary protective mechanisms come into play during an erosive challenge: dilution and clearance of an erosive agent from the mouth, neutralisation and buffering of acids, and slowing down the rate of enamel dissolution through the common ion effect by salivary calcium and phosphate (45).

The salivary acquired pellicle is a protein-based layer which is rapidly formed on dental surfaces after its removal by tooth brushing with dentifrice, other prophylaxis, measures or chemical dissolution. This organic layer becomes detectable on dental surfaces after few minutes of exposure to the oral environment (46). Enzymatic activity is also detectable at early stages of pellicle formation (47). It is suggested that it grows until reaching an equilibrium between protein adsorption and desorption within 2 h (48).

The acquired pellicle may protect against erosion by acting as a diffusion barrier or a perm-selective membrane preventing the direct contact between the acids and the tooth surface, reducing the dissolution rate of hydroxyapatite (48).

Diagnosis

Diagnosis of early forms of erosion is difficult, as it is accompanied by few signs and fewer if any tomi su malobrojni i rijetki. U Tablici 2. je protokol za procjenu stanja i postupke s pacijentima koji gube površine zuba, s obzirom na dosadašnje spoznaje o uzrocima erozije i imajući na umu često preklapanje s atricijom i abrazijom (9).

U svakodnevnoj stomatološkoj praksi nema uređaja za otkrivanje erozije i mjerenje njezina pojačavanja. Zato je u postavljanju dijagnoze najvažnija klinička slika. To je vrlo važno za ranu fazu (2). Prvi znakovi erozivnog trošenja vide se kao promjene optičkih svojstava cakline – ona postaje glatka svilen-

symptoms. Given the current state of knowledge of causes of erosion and keeping in mind the frequent concurrence of attrition and abrasion, a protocol for assessment and management of patients who present with tooth surface loss is presented in Table 2 (9).

There is no device available in routine dental practice for the specific detection of dental erosion and its progression. Therefore, clinical appearance is the most important feature for dental professionals to diagnose this condition. This is of particular importance in the early stage of dental erosion

Tablica 2. Dijagnostički postupak za zubnu eroziju (49)
Table 2 Diagnostic Protocol for Dental Erosion(49)

I. Prikupiti podatke za povijest bolesti - provjeriti sljedeće: • I. Obtain historical data. Check for following items:	
medicinsku povijest bolesti • Medical History • pojačano povraćanje, rumination • Excessive vomiting, rumination • poremećaj u prehrani • Eating disorder • gastroesophagealni refluks • Gastroesophageal reflux disease • simptomi refluksa • Symptoms of reflux • česta uporaba antacida • Frequent use of antacids • alkoholizam • Alcoholism • autoimune bolesti (Sjogrenov sindrom) • Autoimmune disease (Sjogren's) • radijacijska terapija glave i vrata • Radiation tx of head and neck • suhoća usta, suhoća očiju • Oral dryness, eye dryness • lijekovi koji uzrokuju smanjenu salivaciju • Medications that cause salivary hypofunction • lijekovi koji su kiseli • Medications that are acidic	**stomatološku povijest bolesti • Dental History** • povijest bruksizma (škrgutanje ili stiskanje) zvukovi škripanja tijekom sna, opazio ih je partner?. jutarnji zamor ili bol žvačnih mišića? • History of bruxism (grinding or clenching) -Grinding bruxism sounds during sleep noted by bed partner? -Morning masticatory muscle fatigue or pain? • korištenje udlage (nagrizne ploče) • Use of occlusal guard
povijest prehrane • Dietary History • učestalost kisele hrane i pića • Acidic food and beverage frequency • način konzumacije (cuclanje, gutanje?) • Method of ingestion (swish, swallow?)	**metode oralne higijene • Oral Hygiene Methods** • učestalost i vrste četkanja • Toothbrushing method and frequency • vrsta zubne paste (abrazivna?) • Type of dentifrice (abrasive?) • korištenje tekućina za ispiranje usta • Use of mouthrinses • korištenje topikalnih fluorida • Use of topical fluorides
profesionana/rekreacijska povijest • Occupational/ Recreational History • redoviti plivač? • Regular swimmer? • kušač vina? • Wine-tasting? • opasnosti u radnoj okolini? • Environmental work hazards?	
II. Obaviti fizičku procjenu – posvetiti pozornost sljedećem: • II. Perform physical assessment. Observe for following features:	
pregledu glave i vrata • Head and Neck Examination • napeti mišići (bruksizam?) • Tender muscles (bruxism?) • hipertrofija maseteričnog mišića (bruksizam?) • Masseteric muscle hypertrophy (bruxism?) • povećane doušne žlijezde (autoimune bolesti, anoreksija, alkoholizam) • Enlarged parotid glands (autoimmune disease, anorexia, alcoholism) • facijalni znakovi alkoholizma: -crvenila, otekline na licu -spider-angiomi na koži • Facial signs of alcoholism: -Flushing, puffiness on face -Spider angiomas on skin	**iintra-oralnom pregledu • Intra-oral Examination** • znakovi salivarne disfukcije • Signs of salivary hypofunction:: -upala sluznice • Mucosal inflammation -suhoća sluznice • Mucosal dryness -nemogućnost istiskivanja sline iz izvodnog kanala • Unable to express saliva from gland ducts • sjajne fasete ili trošenja na ispunima (bruksizam?) • Shiny facets or wear on restorations (bruxism?) • smještaj i stupanj trošenja zuba (dokumentirati slikama, modelima, radiogramima) • Location and degree of tooth wear (document with photos, models, radiographs
općem stanju • General Survey • pothranjenost (anoreksija) • Underweight (anorexia)	**procjeni salivarne funkcije • Salivary function assessment** • količina protoka • Flow rate • pH, puferski kapacitet (u istraživanju) • pH, buffer capacity (in research)

kasto sjajna i staklasta. Ako se gubitak tkiva nastavi, počet će i promjene u osnovnoj morfologiji zuba.. Na glatkim površinama konveksne se površine izravnavaju ili se razvijaju konkaviteti čija širina prelazi njihovu dubinu. Lezije su locirane koronarno od caklinsko-cementnog spoja (CEJ-a) i uz gingivni rub nema zdrave cakline. Na okluzalnim i incizalnim površinama kvržice se zaobljuju i postaju tupe te se počinje udubljavati incizalni brid, a ispuni se izdižu u odnosu prema okolnoj zubnoj površini. Kod uznapredovalih slučajeva nestaje cjelokupna okluzalna morfologija (24).

Stručnjaci se koriste mnogobrojnim metodama i mjerama kako bi otkrili eroziju, uključujući kliničke indekse (49), analizu slike (50), kontaktnu profilometriju (51), ultrasonifikaciju (52), određivanje propusnosti jodida (53), skening-elektroničku mikroskopiju (54), određivanje površinske mikrotvrdoće (51), transverznu mikroradiografiju (TMR) (55) i kvantitativnu svjetlom induciranu fluorescenciju (QLF) (56). Većina tih tehnika ima velike nedostatke zbog destruktivnog djelovanja na ispitivano tkivo i nemogućnosti da se upotrebljava *in vivo* ili neprecizne kvalifikacije erozije. Može se reći da nema idealne, premda se QLF-om ipak možemo koristiti i *in vitro* i *in vivo* (57).

Vrijednost dosadašnjih dijagnostičkih kriterija dentalne erozije dosad nije sustavno proučena, iako je postignut dogovor o definiciji, no svi se mogu kritizirati, posebice nakon što su predloženi novi indeksi (24). Svi ti novi indeksi za kliničku dijagnostiku erozivnog trošenja zuba su uglavnom modifikacija i kombinacija onih koje su predložili Eccles (58) te Smith i Knight (59). Najčešće citirani primjeri indeksa erozije razvijenih tijekom posljednjih dvadeset godina su (60, 61):

- The Smith and Knight Tooth Wear Index (TWI) (1984.) (59)
- The Eccle's Index (1979) (58)
- UK National Survey of Children's Dental Health Index (1999./2003.) (62)
- Erosion Index according to Lussi (1996.) (63)
- Modified scoring system of Linkosalo and Markkanen (1985.) (64)
- Aine Index 1993. (65)
- The Larsen and Westergaard Index (2000.) (66)
- The O'Sullivan Index (2000.) (67)

Nedavno je postavljeno pitanje koliko su pouzdani i dobri sadašnji dijagnostički kriteriji i podaci o eroziji. Indeksi korišteni u posljednjih dvadeset godina ne mogu se usporediti, nema "zlatnog" standarda - ni studije o ocjeni vrijednosti nisu pomogle

(2). The early signs of erosive tooth wear appear as changes of the optical properties of enamel resulting in a smooth silky–shining glazed surface. When the tissue loss continues, changes in the original morphology occur. On smooth surfaces, convex areas flatten or concavities develop, the width of which clearly exceeds the depth. Lesions are located coronal from the enamel–cementum junction (CEJ) with an intact enamel rim along the gingival margin. On occlusal and incisal surfaces, rounding and cupping of the cusps and grooving of the incisal edges occur, and restorations may rise above the level of the adjacent tooth surfaces. In advanced cases, the whole occlusal morphology disappears (24).

Many methods of erosion detection and measurement have been utilised including clinical indices (49), image analysis (50), contacting profilometry (51), ultrasonification (52), iodide permeability (53), scanning electron microscopy (54), surface microhardness (51), transverse microradiography (TMR) (55) and quantitative light-induced fluorescence (QLF) (56). However, most techniques have inherent disadvantages, such as a destructive nature and the inability to be used in vivo, or inaccurate quantification of erosion; no ideal technique has been established. QLF can, however, be used both in vivo and in vitro (57).

The validity of current diagnostic criteria for dental erosion has not been systematically studied, even though there is consensus about their definition. Nevertheless, there is enough support for a criticism of current diagnostic criteria particularly in the light of the development of a new index (24).

On this basis, a lot of indexes for the clinical diagnosis of erosive tooth wear have been proposed, which are more or less modifications of combinations of the index published by Eccles (58) or Smith and Knight (59). The most cited examples of Erosion Indexes developed during the last 20 years are (60, 61).

- The Smith and Knight Tooth Wear Index (TWI) (1984) (59)
- The Eccle's Index (1979) (58)
- UK National Survey of Children's Dental Health Index (1999/2003) (62)
- Erosion Index according to Lussi (1996) (63)
- Modified scoring system of Linkosalo and Markkanen (1985) (64)
- Aine Index 1993 (65)
- The Larsen and Westergaard Index (2000) (66)
- The O'Sullivan Index (2000) (67)

Recently, the question has arisen how reliable and valid current diagnostic criteria and data on eroidentificirati indeks koji bi mogao biti standard za procjenu zubne erozije.

Zato je osmišljen Basic Erosive Wear Examination (BEWE) kako bi se omogućilo jednostavno bodovanje koje se može uporabljati s dijagnostičkim kriterijima svih dosadađnjih indeksa, no rezultati se moraju prenijeti u jedinicu BEWE sume. BEWE je temeljna struktura za razvoj međunarodno prihvaćenog, standardiziranog i provjerenog indeksa. To treba potaknuti kliničare, studente i stomatologe opće prakse da više pozornosti posvete erozivnom trošenju, što bi trebalo poboljšati zdravstvenu zaštitu pacijenata (68).

Potrebno je odgovoriti na sljedeća pitanja:

- Koji su indeksi erozije najčešći u znanstvenoj literaturi?
- Koje bi kriterije kvalitete trebao imati indeks?
- Kakve su razlike između pojedinačnih i indeksa erozije baziranih na populaciji ?

U razvoj međunarodnih indeksa treba uložiti još mnogo truda kako bi se zubna erozija mogla pouzdano procijeniti. Trenutačne preporuke Svjetske zdravstvene organizacije (WHO-a) i Europske unije te zdrastvenih ustanova i stomatoloških udruga trebale bi se iskoristiti za razvoj i raspravu o konceptu zubne erozije i jedinstvenoga indeksa erozije (61).

Preventivne mjere

Budući da je erozivno trošenje zuba multifaktorijalno, preventivne se mjere moraju primijeniti uzimajući u obzir kemijske i biološke čimbenike te ponašanja pacijenata uključena u etiologiju i patogenezu erozije (Slika 6.) (2).

Preventivne mjere za čimbenike ponašanja

Na pojavu i progresiju zubne erozije odlučujuće utječe način ponašanja (69). Ako često jedemo mnogo kiselih jela, to povećava opasnost od nastanka zubne erozije. I oralna higijena može utjecati na progresiju takvih lezija. Abrazivni postupci, poput četkanja, uklanjaju oslabljenu i demineraliziranu površinu tvrdih struktura zuba. Tako četkanje nakon erozivnog napada (70, 71) i vrsta zubne paste mogu djelovati na progresiju trošenja zuba (72).

sion are. The indexes developed and used during the last 20 years are not comparable; a gold standard does not exist, and validation studies have not had the effect of identifying an index that could be used as a standard for assessing tooth erosion.

The Basic Erosive Wear Examination (BEWE) has therefore been designed to provide a simple scoring system that can be used with the diagnostic criteria of all existing indices aiming to transfer their results into one unit which is the BEWE score sum. The BEWE is a basic structure to initiate the development of an internationally accepted, standardised and validated index. It needs to encourage clinicians, students and GDPs to pay more attention to erosive wear and hence will be beneficial for patient care (68).

The following questions should be answered to advance the definition and assessment of tooth erosion:

- Which erosion indexes are mostly used in the scientific literature?
- Which quality criteria should indexes possess?
- What differences exist between individual- and population-based erosion indexes?

Further efforts have to be made in the development of an internationally agreed index which is able to assess dental erosion with as much reliability and validity as possible. Current recommendations of the WHO and the European Union and health task forces within countries and Dental Associations should be used to develop and to discuss the concept of tooth erosion and the development of a unified erosion index (61).

Preventive Strategies

As erosive tooth wear is a multifactorial condition, preventive strategies have to be applied which account for chemical, biological and behavioral factors involved in the etiology and pathogenesis of erosion (Figure 6) (2).

Preventive Strategies for Behavioral Factors

Behavioral factors have a decisive influence on the appearance and progression of dental erosion (69). The frequent and excessive consumption of acids is associated with an increased risk for dental erosion. Oral hygiene measures might also influence the progression of erosive lesions. Abrasive procedures, such as toothbrushing, are known to remove the fragile surface of demineralized dental hard tissues. In this way, the moment of toothbrushing after an erosive attack (70, 71) as well as the kind of toothbrush and toothpaste used might influence the progression of dental wear (72).

Mjere za što manju izloženost kiselinama

Kako bi se prevenirala zubna erozija, najvažnije je smanjiti izloženost kiselinama. Vanjski izvori su uglavnom kiseline u hrani, ali tu se ubraja i način života (primjerice različite droge) i boravak u kiselom okolišu tijekom rada. Da bi se smanjila opasnost od erozija potaknutih hranom, pacijente bi trebalo upozoravati neka ne jedu kisele međuobroke kako bi omogućili slini da očvrsne erodiranu zubnu površinu. Treba ih upozoriti i na nezdrave načine života, kao što su uzimanje droga, alkohol i laktovegetarijanska prehrana, jer mogu povećati rizik od erozije (69). Zbog kiseloga okoliša najugroženiji su radnici s baterijama, u punionicima i oni u procesu galvanizacije, jer je tamo jako djelovanje sumporne ili klorovodične kiseline. Osobna zaštitna oprema (maske za disanje) i oprez kako se ne bi prešle granične vrijednosti koje su preporučili stručnjaci zaštite na radu, smatraju se važnim preventivnim mjerama za smanjivanje erozije u radnim okružjima (73). Unutarnji čimbenici uključuju poremećaje povezane sa želučanom kiselinom u usnoj šupljini, a tu ubrajamo povraćanje i gastroezofagealni refluks. Te tegobe zahtijevaju kauzalnu terapiju (opći lijekovi, psihoterapija).

Mjere za smanjivanje mehaničkog učinka

Iz istraživanja *obavljenih in vitro*(74) i *in situ* (75, 76) može se zaključiti da mehanički stres na erodiranim površinama uzrokuje uglavnom četkanje zuba, ali i atricija zbog dodira zub-na-zub, trenja jezika ili abrazije okolnoga mekog tkiva pod kliničkim uvjetima. U dosadašnjim istraživanjima (77, 78, 79) istaknuto je da električne i ručne četkice zbog različitog pritiska variraju u mogućnostima da uklone krhku površinu demineralizirane cakline i dentina. Za razliku od toga, abraziju tijekom četkanja uzrokuju većinom zubne paste (80). Njhov abrazivni učinak određuje se prema veličini i količini abrazivnih zrnaca, pH-vrijednosti, puferskom kapacitetu i koncentraciji fluorida (80, 81). Smatra se da zubne paste s fluoridima ne samo da smanjuju erozivnu demineralizaciju, nego i abraziju erodiranih površina (82, 42). Zato bi se pacijenti s erozivnim lezijama trebali koristiti zubnim pastama s fluoridima i niskim stupnjem abrazivnosti.

Preventivne mjere za biološke čimbenike

Na razvoj erozije mogu utjecati biološki čimbenici, kvaliteta zubne strukture, svojstva sline, smje-

Measures to Reduce the Acid Exposure

Of major importance for the prevention of dental erosion is the reduction of the acid exposure. Extrinsic acid sources of erosion are mainly dietary acids, but also lifestyle factors (e.g. drugs) or occupational acid exposure. To decrease the risk of dietary induced erosive lesions, patients should be advised to refuse from acidic snacks between the principal meals to allow the saliva to reharden eroded tooth surfaces. In addition to the dietary acids, patients should be aware of unhealthy lifestyle factors, such as consumption of drugs, alcohol abuse and lacto-vegetarian diet, which might increase the risk for erosion (69).With regard to environmental acid exposure, an increased risk for dental erosion is reported for battery, charging and galvanizing workers, which are commonly exposed to sulphuric or hydrochloric acid. Personal protective equipments (respiratory masks) and adherence to threshold limit values recommended by occupational health legislations are considered as important preventive strategies to decrease occupational erosion (73). The intrinsic etiologic factors of erosion include disorders that are associated with the presence of gastric acid in the oral cavity, such as vomiting or gastroesophageal reflux. These disorders require a causal therapy (general medicine, psychological therapy) for a permanent reduction of the intrinsic acid exposure.

Measures to Reduce the Mechanical Impact

From in vitro (74) and in situ studies (75, 76) it is concluded that the mechanical stress of eroded surfaces may be mainly induced by toothbrushing but also by attrition due to tooth-tooth-contact, tongue friction or abrasion of surrounding soft tissues under clinical conditions. Previous studies (77, 78, 79) have shown that powered and manual toothbrushes as well as manual toothbrushes applied with different brushing loads vary in their ability to remove the fragile surface of demineralized enamel and dentin. In contrast, toothbrushing abrasion is mainly influenced by the toothpaste used (80). The abrasivity of the toothpaste is determined by the size and amount of abrasives, pH, buffering capacity and fluoride concentration (80, 81). Fluoridated toothpastes might not only reduce the erosive demineralization, but also reduce the abrasion of eroded tissues (82, 42). Therefore, patients with erosive lesions should use fluoridated toothpastes with low abrasivity for their oral hygiene measures.

Preventive Strategies for Biological Factors

With regard to biological factors, the quality of dental tissues, properties of saliva, tooth position and štaj zuba i anatomija mekih tkiva (83). Čini se da je slina najvažnija u smanjivanju trošenja cakline i dentina u erozivnim/abrazivnim napadima zbog njezina puferskog kapaciteta i svojstva remineralizacije te stvaranja zaštitne pelikule na površini tvrdih zubnih tkiva (84, 85). Kserostomija ili hiposalivacija dosta je česta kod pacijenata tijekom radijacijske terapije glave i vrata, ali i kod onih s bolesnim žlijezdama slinovnicama (Sjögrenov sindrom) ili može nastati jatrogeno kao posljedica nekih sistemskih lijekova. Rios i suradnici (86) istaknuli su da slina stimulirana korištenjem žvakaćih guma bez šećera pomaže remineralizaciju kod erozivnog/abrazivnog fenomena.

Taj se učinak može pojačati ispiranjem mlijekom ili konzumacijom sira. Te su namirnice vrlo zanimljive, jer sadržavaju više kalcija i fosfata negoli voda ili slina te mogu postati izvor tih tvari za remineralizaciju (87). Slina je također odgovorna za stečene pelikule – one su fizička zapreka koja štiti zub od erozivnog napada (83, 87). Na taj se način sprječava doticaj kiselina i površine zuba te smanjuje topljenje hidroksilapatita. Zaštita zubne površine uz pomoć stečene pelikule potanko je opisana u literaturi i dokazana u mnogobrojnim istraživanjima (88, 89).

Preventivne mjere za kemijske učinke

Utjecaj terapije fluoridima na progresiju erozije cakline i dentina analiziran je u nekoliko studija (90, 91). Stvaranje sloja sličnog kalcijevu fluoridu - CaF_2 te njegov zaštitni učinak ovise o pH-vrijednosti, koncentraciji fluora (F) i vrsti korištenih fluoridnih soli (92). Preparati s visokom koncentracijom fluorida, kao što su tekućine za ispiranje usta, gelovi i lakovi, povećavaju otpornost na abraziju i smajuju razvoj caklinske i dentinske erozije *in vitro* i *in situ* (90, 93).

Za razliku od preparata s visokim udjelom fluorida, zubne paste s 1000 ppm fluora vrlo malo i ograničeno utječu na abraziju erodiranog dentina i cakline, gotovo poput onih nefluoridiranih (88). U jednoj nedavnoj studiji istaknuto je da je pasta s 5000 ppm fluorida imala isti učinak kao i ona od 1100 ppm na erodirani te na erodirani i abradirani dentin (42). Posljednji podaci upućuju na to da fluoridi mogu zaštititi zubnu strukturu od teške erozije kod pH-vrijednosti 2,35 i 3,0 (94, 95). Hove i suradnici (96) istaknuli su da intenzivna aplikacija fluorida dobro štiti caklinu od erozije u uvjetima

anatomy of the soft tissues might affect the development of dental erosion (83). Saliva seems to play an important role in minimizing enamel and dentin wear in erosive/abrasive attacks due to its buffering and remineralizing capacities as well as the ability to form a protective pellicle layer on dental hard tissues (84, 85). Xerostomia or hyposalivation is a condition frequently observed in patients undergoing radiation therapy on the head and neck region, but is also common in patients suffering from diseases of the salivary glands (Sjögren syndrome) or can be induced by several systemic medications. Rios, et al. (86) showed that saliva stimulated by the use of sugar-free chewing gum promoted a remineralizing action in the erosive/abrasive phenomena.

This remineralizing effect might be increased by rinsing with milk or eating cheese, which are of interest as they contain higher levels of calcium and phosphate than water or saliva and, therefore, may act as donor of calcium and phosphate for remineralization (87). Saliva is also responsible for the formation of the acquired pellicle, which is a physical barrier that protects the tooth against erosive attacks (83, 87). This selective barrier prevents the direct contact between acids and the tooth surface, thus reducing the dissolution of hydroxyapatite. Protection of the tooth surface by the acquired pellicle is well established in the literature and has been demonstrated by several studies (88, 89).

Preventive Strategies for Chemical Factors

The impact of fluoride treatment on the progression of enamel and dentin erosion has been analyzed in several studies (90, 91). The formation of the CaF_2-like layer and its protective effect on demineralization depend on the pH, F concentration and type of F salt of the agent (92). High-concentrated fluoride agents, such as oral rinses, gels or varnishes, have been demonstrated to increase abrasion resistance and decrease the development of enamel and dentin erosion in vitro and in situ (90, 93).

In contrast to the application of highly fluoridated agents, a 1,000 ppm F dentifrice was shown to have a limited beneficial effect compared to non-fluoridated dentifrices on abrasion of eroded dentin and enamel (88). However, a recent in situ study showed that a 5,000 ppm F dentifrice had the same effect as a 1,100 ppm F dentifrice on eroded and eroded and abraded dentin (42). Recent evidence indicates that fluoride can protect tooth structure from severe erosion at pH 2.35 and pH 3.0 (94, 95). Hove et al.(96) observed that intensive application of fluoride provided significant protection against enamel erosion stimuliranoga želučanog refluksa kod pH-vrijednosti 2,0, a Willumsen i njegovi kolege (97) uočili su da fluoridi nisu zaštitili caklinu kod pH-vrijednosti 1,2. Ostali podaci pokazuju da topikalna aplikacija fluorida može zaštititi caklinu i dentin od kombinacije abrazije četkanjem i erozije kod pH-vrijednosti od oko 3,0 (98, 99), ali ne i od atricije cakline i dentina (100).

Alternativna preventivna metoda mogla bi uključivati primjenu sredstava za podmazivanje na mjestu trošenja. Primjerice, Kaidonis i suradnici (101) istaknuli su sljedeća sredstva za podmazivanje: prah kalcijeva fluorida (CaF) te mješavinu kalcijeva fluorida i maslinova ulja – oba su na mjestu doticaja smanjila trošenje cakline u usporedbi sa suhim stanjem (bez dodatnog sredstva za podmazivanje).

Nedavno su se ispitivala i ostala sredstva poput tetrafluorida (TiF_4, ZrF_4, HfF_4 u koncentracijama između 0, i 10%, te pH 1-2), a posebice se istraživalo na koji način titanijev tetrafluorid sprječava eroziju. Nekoliko istraživanja *in vitro* s otopinom TiF_4 dokazalo je inhibitorni učinak na nastanak zubne erozije (92, 102), što se ne pripisuje samo fluoridu nego i titaniju. Nedavno je završena studija o progresiji caklinske erozije u kojoj se uspoređuje eksperimentalna 4-postotna otopina TiF_4 i komercijalni lak (NaF). Eksperimentalni premaz s TiF_4 bio je bolji u zaštiti negoli komercijalni premaz (NaF), a otopina TiF_4 nije utjecala na smanjivanje trošenja cakline (103). Nažalost, preparati s TiF_4 imaju jako kiselu pH-vrijednost (pH 1-2), što pacijentima ne dopušta samoaplikaciju.

Jedna od preventivnih mjera može biti i smanjenje erozivnog potencijala kiselih napitaka dodavanjem iona (kalcija, fosfata i fluorida). Tako ako dodamo kalcij, smanjuje se erozivna snaga čistih kiselina i kiselih napitaka, posebice kod erozije cakline (104-106).

Larsen i Nyvad (107) te Larsen i Richards (108) istaknuli su da mješavine s dodatkom fluorida u koncentracijama koje ne potiču toksične popratne pojave, ne mogu smanjiti erozivne lezije. U njihovim studijama kaže se da dodatak niskih koncentracija kalcija, fosfata i fluorida nije smanjio erozivnost otopina s pH-vrijednostima manjima od 4,0 (6).

Fluoridi su ključni za održavanje oralnoga zdravlja, posebice u prevenciji oštećenja tvrdih zubnih tkiva (109). Zbog toga se u preparatima za poboljšanje oralnog zdravlja istraživalo nekoliko spojeva fluora, kao NaF, SnF2 ili TiF (110). Otkriveno je da su vrlo djelotvorni preparati fluora s kositrom, baš kao što se i mislilo već prije pet desetljeća (111).

under conditions simulating gastric reflux at pH 2.0, but Willumsen et al. (97) noted that fluoride did not protect enamel against erosion at pH 1.2. Other reports have indicated that topical fluoride can protect enamel and dentine against a combination of toothbrush abrasion and erosion at around pH 3.0 (98, 99), but not against attritional wear between opposing enamel and dentine surfaces (100).

An alternative preventive method could involve application of a lubricating agent at the wear interface. For example, Kaidonis et al. (101) showed that lubrication at the wear interface, provided by calcium fluoride powder (CaF) and CaF/ olive oil slurry, reduced enamel wear compared with dry (no additional) lubrication.

More recently, other agents, such as tetrafluorides (TiF_4, ZrF_4, HfF_4 in a concentration between 0.4 to 10%, pH 1-2), especially titanium tetrafluoride, have been investigated for erosion prevention. With regard to TiF_4 solution, several in vitro studies have shown an inhibitory effect on dental erosion (96, 102), which is attributed not only to the effect of fluoride, but also to the action of titanium. Recently, an in vitro study comparing the efficacy of a 4% TiF_4 solution, an experimental 4% TiF_4 varnish and commercial NaF varnishes on the progression of enamel erosion was performed. The experimental TiF_4 varnish showed the best protective effect when compared to commercial NaF varnishes, while TiF_4 solution was not effective to reduce the enamel wear (103). However, TiF_4 agents have a very acidic pH (pH 1-2), which does not allow for patient self-application.

One preventive strategy might be the reduction of the erosive potential of acidic beverages by ions supplementation (calcium, phosphate and fluoride). The addition of calcium has been shown to reduce the erosive potential of pure acids and acidic drinks, especially on enamel erosion (104-106).

Larsen and Nyvad (107) and Larsen and Richards (108) showed that fluoride admixtures in a concentration excluding toxicological side effects seem unable to reduce erosive lesions. The supplementation of low levels of calcium, phosphate and fluoride was not effective in decreasing the erosive potential of solutions with a pH below 4.0 in the above-mentioned studies (6).

Fluorides are a key component in oral health promotion, especially in the prevention of damage of the oral hard tissues (109). Due to such properties, several fluoride compounds, such as NaF, SnF2 or TiF (110), have been investigated for use in prod-

Osim što ima antimikrobna svojstva, kositar štiti od demineralizacije zahvaljujući sposobnosti da reagira (112) i preoblikuje zubnu površinu (113), a rezultat je veća otpornost na karijes. Doista, u mnogobrojnim su se studijama istraživala svojstva kositra i potvrdio njegov učinak u zaštiti od karijesa, pa rezultati obećavaju. U eksperimentalnim modelima karijesa, topivost cakline je smanjena poslije demineralizacije mliječnom kiselinom za oko 80% samo nakon primjene 0,2-postotne otopine SnF2 (114). U drugom istraživanju terapija sa SnF2 rezultirala je znatno manjom topivošću cakline negoli terapija natrijevim fluoridom (115). Premda se preparati kositrenog fluorida promoviraju kao lijek izbora za smanjenje topivosti cakline, rjeđe se upotrebljavaju (barem u Europi) (2), zbog ograničene stabilnosti SnF2 u zubnim pastama ili loše formule, posebice kad je riječ o okusu. Zbog toga se radije odabiru alternativni fluoridi poput natrijeva- ili amin-fluorida (AmF) te natrijeva monofluorofosfata (116). U posljednjih nekoliko godina kositreni je fluorid ponovno u središtu pozornosti.

Već preporučene mjere u prevenciji erozije četkanjem uključuju i zaštitni plašt od smole na zubima (117) ili topikalnu fluoridaciju, što povećava otpornost zubne strukture na erozivno trošenje (118). Nije moguće uvijek ukloniti uzroke trošenja zuba te je potrebno razviti i druge preventivne mjere (117).

Xylitol može stvarati komplekse s kalcijem, prodrijeti u demineraliziranu caklinu i umiješati se u prijenos otopljenih iona iz lezije u demineralizirajuću tekućinu te tako smanjiti difuzijski koeficijent kalcija i fosfata iz lezije u otopinu (119, 120).

Kiselim se otopinama alternativno mogu dodati metalni ioni poput željeza koji im, čini se, smanjuju erozivnu snagu (121). Željezo može sudjelovati u remineralizaciji cakline, u nukleaciji apatita, zamjeni za kalcij u apatitu i inhibiciji demineralizacije (122). Važno je istaknuti da su istraživanja u kojima se analizira učinak dodavanja željeza u bezalkoholna pića s visokim koncentracijama iona, pokazala da ioni mogu imati toksične učinke (123). Zbog svega toga prijeko su potrebna daljnja istraživanja koja će uzeti u obzir sve te čimbenike, posebice promjene okusa, stabilnost otopine i sistemski učinak na pacijente. Potrebno je obaviti i istraživanja o utjecaju modificiranih bezalkoholnih pića na eroziju dentina.

ucts for oral health. In particular, stannous fluoride containing preparations have proved to be very effective, where their therapeutic benefits were established as early as five decades ago (111).

In addition to its antimicrobial properties, the efficacy of tin in protection against demineralisation is attributed to its ability to react with (112) and to modify the tooth surface (113), and this effect is likely to result in a greater resistance to decay. Indeed, several studies have investigated tin's properties of conferring protection against caries, and the results have been very promising. In a caries model, the solubility of enamel, with demineralisation by lactic acid, was reduced by approximately 80% with the application of a 0.2% SnF2 solution (114). In another study, SnF2 treatment resulted in a greater reduction of enamel solubility compared with NaF treatment (115).

Although stannous fluoride-containing preparations have been promoted as the agent of choice for reducing enamel solubility, it has been less frequently used (at least in Europe) (2), which may be due to the limited stability of SnF2 in dentifrice or the poor formulation flexibility, particularly as a result of its flavour. Therefore, alternative fluorides, such as NaF, amine fluoride (AmF) or sodium monofluorophosphate have been preferred (116). In the last few years, stannous fluoride has again become the focus of research.

Previously suggested strategies for preventing erosive tooth wear by toothbrushing include the application of a protective resin coating on teeth (117) or of topical fluoride, which increases the resistance of tooth structure against erosive wear (118). It is not always practical to eliminate the causes of tooth wear in individuals, so it is desirable to develop other effective preventive strategies to manage tooth wear (117).

Xylitol might form complexes with calcium, penetrate into demineralized enamel and interfere with the transport of dissolved ions from the lesion to the demineralizing solution by lowering the diffusion coefficient of calcium and phosphate ions from the lesion into the solution (119, 120).

Alternatively, acidic solutions can be supplemented with metal ions, such as iron, which seems to decrease the erosive potential of acidic solutions (121). Iron can participate in the remineralization of human enamel, in the nucleation of apatite, substitution of calcium in apatite and inhibition of demineralization (122). It is important to highlight that studies analyzing the effect of iron supplementation to soft drinks

Aplikacija kalcija

Istraživanja o zubnom karijesu pokazuju da bi veća koncentracija kalcija u slini i plaku mogla povećati unos fluorida i njihovo zadržavanje te pojačati učinak u procesima demineralizacije i remineralizacije. Pokušavajući spriječiti zubnu eroziju razumno bi bilo povećati koncentraciju kalcija u slini, jer bi mogao pomoći u odlaganju fluorida na zubno tkivo i stvaranju zaliha CaF_2(6).

Trenutačno postoji samo nekoliko studija o utjecaju na eroziju zubnih pasta obogaćenih kalcijem. Lennon i suradnici (124) analizirali su djelovanje paste s kazein/kalcijevim fosfatom (Topcalom) na eroziju cakline *in vitro*. Topcal i kombinacija Topcala s 250 ppm fluorida samo su neznatno štitili od erozije i bili su znatno manje učinkoviti od visoko fluoridiranog amin-fluoridnoga gela.

Rees i suradnici (125) te Piekarz i njegovi kolege (126) ustanovili su da je Tooth Mousse (CPP-ACP: kazein fosfopeptid–amorfni kalcijev fosfat) (GC Asia Pty. Ltd. Japan) jako smanjio eroziju cakline uzrokovanu limunskom kiselinom (126) te kiselim sportskim napitcima (127). Ranjitkar i suradnici (128) izvijestili su da je atricijsko trošenje dentina *in vitro* gotovo uklonjeno nakon stalne primjene TM-a u usporedbi s klorovodičnim kiselim sredstvom za podmazivanje (pH 3,0) te s deioniziranim vodenim sredstvom za podmazivanje (pH 6,1). Naizmjenična primjena TM-a također je smanjila trošenje dentina u kiselom i gotovo neutralnom okolišu, no moraju se istaknuti njegova svojstva podmazivanja i remineralizacije kad je riječ o smanjivanju erozivnog trošenja dentina (128). Učinak TM-a u smanjivanju erozivnog trošenja zuba zbog četkanja nije se ocjenjivao.

U nedavnoj studiji *in vitro* gotovo je uklonjeno atricijsko trošenje dentina i to kontinuiranom primjenom paste s antikariogenim sadržajem u obliku kazein fosfopeptid–amorfnog kalcijeva fosfata (CPP–ACP-a) i nanokompleksa (128). Naizmjenična primjena paste s CPP–ACP-om također je smanjila trošenje dentina kako u kiseloj tako i u neutralnoj okolini, te se moraju istaknuti svojstva podmazivanja i remineralizacije u smanjivanju ero-

or acid solutions, used high concentrations of iron, which might exhibit toxic effects (123).

Therefore, further research taking into account these factors is requires with special emphasis on the consequences of taste alterations, stability of the solution and systemic effects for the patients. Studies evaluating the effects of soft drink modification on dentin erosion should also be performed.

Calcium Application

Studies involving dental caries suggest that increased salivary and plaque calcium concentrations might enhance fluoride uptake and retention and, thus, increase the action of fluoride in the de- and remineralization process. Regarding dental erosion, it is reasonable to increase the salivary calcium concentration, which might enhance fluoride deposition on the dental tissues by formation of a CaF_2-like reservoir (6).

There are currently only few studies about the effect of calcium-rich toothpastes on dental erosion. Lennon, et al. (124) analyzed the effect of a casein/calcium phosphate-containing tooth cream (Topacal) on enamel erosion in vitro. Topacal or a combination of Topacal and a 250 ppm fluoride solution provided only little protection against erosion and were significantly less effective than a highly fluoridated amine fluoride gel.

Rees, et al. (125) and Piekarz, et al. (126) found that Tooth Mousse (CPP-ACP: Casein phosphopeptide - amorphous calcium phosphate) (GC Asia Pty. Ltd. Japan), significantly reduced enamel erosion caused by citric acid (126) and an acidic sports drink (127).

Ranjitkar et al. (128) reported that attritional wear of dentine was almost eliminated in vitro with continuous application of TM compared with hydrochloric acid lubricant (pH 3.0) and deionized water lubricant (pH 6.1). Furthermore, intermittent application of TM also reduced dentine wear in both acidic and near neutral environments, highlighting its lubricating and remineralizing properties in reducing erosive dentine wear (128). However, the effectiveness of TM in reducing erosive tooth wear involving toothbrush abrasion has not been assessed.

In a recent in vitro study, attritional wear of dentine was almost eliminated with continuous application of a paste containing an anticariogenic remineralizing agent in the form of casein phosphopeptide–amorphous calcium phosphate (CPP–ACP) nanocomplexes (128). Furthermore, intermittent application of CPP–ACP paste also reduced dentine wear at both acidic and near neutral environments, high lighting its lubricating and remineralizing properties in rezivnog trošenja dentina (128). To podupire spoznaja da CPP–ACP može smanjiti zubnu eroziju uzrokovanu limunskom kiselinom (125), bijelim vinom (126) i sportskim napitcima (127) te erozivno trošenje zbog abrazije četkanjem (129). Nakon tih analiza opravdano je istraživanje učinka CPP–ACP-a na trošenje cakline zbog kombinacije atricije i erozije (129, 130).

CPP–ACP kao antikariogeno sredstvo potvrđen je *in vitro*, ali i *in situ* (131, 132). Tako je spriječio demineralizaciju i poticao remineralizaciju potpovršinske karijesne lezije u caklini i dentinu (133). CPP–ACP održava razinu zasićenosti kalcija i fosfata na površini zuba i zalihe neutralnih ionskih parova (CaHPO4 0) koji inhibiraju demineralizaciju i potiču stvaranje kristala hidroksilapatita u karijesnoj leziji (134). Može se otkriti i u sastavu plaka i na površini bakterijskih stanica tri sata nakon primjene tekućina za ispiranje usta sa CPP–ACP-om ili žvakaćim gumama (131). Na koji način CPP–ACP smanjuje erozivno trošenje, još se ne zna. Nalaz da TM povećava tvrdoću cakline erodirane *colom* (135) upućuje na to da njegov učinak na eroziju vjerojatno uključuje i proces remineralizacije. Za razliku od remineralizacije karijesne lezije, erodirana će površina prije biti popravljena odlaganjem minerala u poroznu zonu, negoli rastom kristala (136). Hipoteza je u skladu sa spoznajama da se na caklini nakon izlaganja sportskim napitcima sa CPP–ACP-om stvaraju zrnate strukture (137).

Zbog premalo podataka zasad nije moguće dati konačan zaključak o učinkovitosti preparata bogatih kalcijem, kad je riječ o zubnoj eroziji. Potrebna su daljnja istraživanja kako bi se ispitao preventivni učinak otopina kalcija i pasta bogatih kalcijem na eroziju cakline i dentina (6).

Primjena lasera

Posljednjih je godina sve veće zanimanje za istraživanje zaštitnog učinka lasera na demineralizaciju cakline i dentina. Proučavalo se nekoliko vrsta, kao ruby, CO_2, Nd:YAG i argonski, s različitim operativnim modovima i izlaznom energijom. Terapija laserom potiče nekoliko kemijskih promjena na zubnoj površini, uključujući smanjenje karbonata i promjene hidroksilapatita u fluorapatit ako se primjenjuju zajedno sa sredstvima fluorida (138). Otopljena površina cakline može potaknuti rast kristala

ducing erosive dentine wear (128). These findings are supported by observations that CPP–ACP can reduce dental erosion by citric acid (125), white wine (126) and sports drinks (127), as well as erosive tooth wear involving toothbrush abrasion (129). Given these findings, an investigation of the effect of CPP–ACP on enamel wear by a combination of attrition and erosion is warranted (129, 130).

The potential of CPP–ACP as an anticariogenic agent has been reported both in vitro and in situ (131, 132) with CPP–ACP preventing demineralization and promoting remineralization of subsurface carious lesions in enamel and dentine (133). CPP–ACP maintains saturation levels of calcium and phosphate at the tooth surface and provides a reservoir of neutral ion pairs (CaHPO4 0), which inhibit demineralization and promote the formation of hydroxyapatite crystals inside carious lesions (134). CPP–ACP is also detectable in the plaque matrix and the surface of bacterial cells of subjects 3 h after consuming CPP–ACP-containing mouthrinse or chewing gum (131). The mechanisms by which CPP–ACP reduces erosive tooth wear are unclear. However, the finding that TM increases hardness of enamel eroded by cola drink (135) implies that its erosion inhibiting potential probably involves remineralization action. Unlike the process of remineralization of carious lesions, eroded tooth structure is likely to be repaired by deposition of mineral into the porous zone rather than crystal regrowth (136). This hypothesis is consistent with the observation that superficial granular structures were noted to form on the enamel surface, probably representing remineralized enamel structure, after treatment with a sports drink containing CPP–ACP (137).

Due to the lack of available data, final conclusions about the efficacy of calcium-rich products on dental erosion can not be drawn so far. Further studies must be performed testing the preventive effect of calcium solutions and calcium-rich dentifrices on enamel and dentin erosion (6).

Laser Application

The protective effects of laser application on enamel and dentin demineralization have gained increasing attention in the last years. Several types of lasers, such as ruby, CO_2, Nd:YAG and argon, with different operative modes and energy outputs have been investigated. The laser treatment causes several chemical changes on tooth surface, including the reduction of the carbonate content and the exchange of hydroxyapatite to fluorapatite when applied with fluoride vehicles (138). The melted enamel surface koji može smanjiti interprizmatske prostore te difuziju kiselina tijekom napada kiselina (139). Sve te kemijske i morfološke promjene zubne površine mogu smanjiti osjetljivost na erozivnu demineralizaciju.

Malo je istraživanja u kojima se analizira učinak primjene lasera na prevenciju erozivne demineralizacije - većina su u vezi s karijesom i neerozivnom demineralizacijom. Tsai i suradnici (140) usporedili su djelovanje terapije laserom (pulsni CO_2 i pulsni Nd:YAG - 83.33 J/cm^2) na otpornost humane cakline *in vitro*. Laser Nd:YAG nije mogao povećati otpornost cakline na napad kiselinom (laktatna puferska otopina, pH 4, 5, 24 i 72 sata). Ali, primjena lasera Nd:YAG (0,5, 0,75 i 1 W) u kombinaciji s aplikacijom fluorida (fluoridni gel i premaz) znatno je smanjila erozivno trošenje cakline u petodnevnoj studiji *in vitro* (141). Kad je erozivni napad bio produžen na 10 dana, kombinirana primjena lasera Nd: YAG i fluoridnog gela i dalje je uspješno smanjivala caklinsko trošenje, što se može zahvaliti niskim pH-vrijednostima fluoridnog preparata (142).

Autori su istaknuli da je dentin osvijetljen laserom Nd:YAG 0,6 W otporniji na kisele napitke kao što su *cola* i bezalkoholna pića te voćni sokovi. Magalhãesi i suradnici (143) dokazali su, pak, da primjena lasera Nd:YAG (0,5, 0,75 i 1 W) nije smanjila erozivno trošenje dentina.

Zbog malo podataka još se ništa ne može zaključiti o uspješnosti primjene lasera na eroziju zuba, pa su potrebna dodatna istraživanja.

Inhibitori matriks-metaloproteinaza (MMP-a)

Matriks-metaloproteinaze (MMP) odgovorne su za hidrolizu izvanstaničnog matriksa tijekom remodelacije i razgradnje u oralnom okolišu. Organski matriks dentina (kolagen) može se razgraditi MMP-om u dentinu i slini. Ravnoteža između aktiviranoga MMP-a i tkivnih inhibitora metaloproteinaza (TIMP-a) kontrolira količinu ECM-remodeliranja/razgradnje (144). Čini se da je aktivacija MMP-a vrlo važna u progresiji dentinskog karijesa, budući da je nužan u razgradnji kolagena u karijesnoj leziji. Pojedinci s visokom koncentracijom MMP-a u slini osjetljiviji su na zubni karijes (145). Iako je premalo istraživanja o njegovu učinku u eroziji zuba, vjeruje se da se događaju slični procesi kao i kod karijesa (146). Tjäderhan i suradnici (146) ističu da se laten-

can show a crystal growth that can reduce the interprismatic spaces and consequently, the diffusion of acids during an acid challenge (139). All these chemical and morphological changes of the dental surface might lead to a decreased susceptibility to erosive demineralization.

However, there are few studies available testing the effect of the laser application on the prevention of erosive demineralization and most of them are related to carious and not erosive demineralization. Tsai, et al. (140) compared the effectiveness of laser treatment (pulsed CO_2 and pulsed Nd:YAG - 83.33 J/cm^2) on the acid resistance of human enamel in vitro. The Nd:YAG laser was not able to increase the enamel resistance to an acid challenge (lactate buffer solution, pH 4.5, 24 and 72 h). In contrast, the application of Nd:YAG laser (0.5, 0.75 and 1 W) combined or not to fluoride application (fluoride gel and varnish) significantly reduced the enamel erosive wear in a 5-day-in vitro study (141). Additionally, when the erosive challenge was extended to 10 days, the combined application of Nd:YAG laser and fluoridated gel was still effective on the reduction of the enamel wear, which could be attributed to the low pH of the fluoride agent (142).

The authors suggested that dentin irradiated with 0.6 W Nd:YAG laser presented a higher resistance to acidic beverages such as cola soft drink and passion fruit juice. On the other hand, Magalhães, et al. (143) showed that the application of Nd:YAG laser (0.5, 0.75 and 1 W) was unable to reduce the dentin erosive wear.

Due to the few data available so far, final conclusions about the efficacy of laser application on dental erosion cannot be drawn as yet. Further studies are necessary to clarify this topic.

MMPs (matrix metalloproteinases) inhibitors agents

Matrix metalloproteinases (MMPs) are responsible for hydrolyzing the components of the extracellular matrix (ECM) during the remodeling and degradation processes in the oral environment. Thus, the organic matrix of dentin (collagen) can be degraded by MMPs present in dentin and saliva. The balance between activated MMPs and tissue inhibitors of metalloproteinases (TIMPs) controls the extent of ECM remodeling/degradation (144). The activation of MMPs seems to play a role in dentinal caries progression, since they have a crucial role in the collagen breakdown in caries lesions. Individuals with a high concentration of MMPs in saliva present an increased susceptibility to dental caries (145). Despite tni oblici MMP2 i MMP9 mogu aktivirati u kiselim stanjima, a zatim slijedi neutralizacija kao i u karijesnom procesu kada pH-vrijednost zubnog plaka pada za nekoliko minuta nakon unošenja šećera dok god se ne neutralizira puferom iz sline. Zbog uključenosti MMP-a domaćina u progresiju karijesne lezije na humanim zubima, možda bi bilo zanimljivo pronaći inhibitore MMP-a za pacijente s visokim rizikom od nastanka karijesa, ali i erozije (147).

Za polifenole iz zelenoga čaja, a posebice epigalokatehin galat (EGCG), ustanovljeno je da imaju određen inhibitorni učinak na MMP (148). U nedavnom istraživanju o preventivnom djelovanju zelenoga čaja na trošenje zuba, istaknuto je da ispiranje tim napitkom smanjuje eroziju zuba i abraziju *in situ* (149).

Drugi mogući inhibitori MMP-a su klorheksidini (CHX), antibakterijski preparat za koji je ustanovljeno da inhibira aktivnost MMPa 2, 8 i 9 (154), ali i neutralne kao avokado, zrna soje i oleinska kiselina (150). CHX povoljno djeluje na dentinsku snagu *in vivo* kao MMP-inhibitor (151), kada se primjenjuje između napada kiselina i procedura adhezivnog lijepljenja. Njihov način djelovanja i učinak na eroziju zuba još nije istražen te će se to vjerojatno učiniti u budućim studijama o prevenciji erozije zuba (6).

Restorativna terapija

Ovisno o stupnju trošenja zuba, restorativni postupak može se sastojati od adhezivnog postavljanja kompozitnog ispuna u pojedinim područjima erozije, pa sve do potpune rekonstrukcije u slučajevima jako uništene denticije. Opisi pojedinih tehnika nisu u sklopu našeg zadatka. Bez obzira na vrstu restorativne terapije, prevencija progresije trebala bi biti osnova za liječenje pacijenata s erozijom. To će povećati mogućnosti za uspjeh i osigurati dugotrajnost restorativnog zahvata (6).

Zaključak

U kliničkoj stomatologiji neprestance raste zanimanje za što bolje razumijevanje etiologije i rješavanje problema trošenja zuba (152), budući da sve više starijih pacijenata ima svoje zube dok se na njima ne opaze znakovi velike istrošenosti (2). Sve je više i pacijenata s istrošenim i mliječnim i trajnim

the lack in studies investigating the role of MMPs in dental erosion, processes similar to the caries process can be assumed for erosive lesions (146).

Tjäderhane, et al. (146) found that the latent forms of MMP 2 and MMP 9 can be activated in acidic conditions followed by neutralization, as it occurs during the carious process when the pH in dental plaque drops within minutes after sugar ingestion until neutralized by salivary buffers. Due to the involvement of host MMPs to the progression of dental caries in human teeth, it might be interesting to find MMP inhibitors for patients with high risk for caries but also for erosion (147).

Green tea polyphenols, especially epigallocatechin gallate (EGCG), were found to have distinct inhibitory activities against MMPs (148). A recent study about the preventive effect of green tea on dentin wear has shown that the rinse with green tea reduced dentin erosion and abrasion in situ (149).

Other potential MMP inhibitors are chlorhexidine (CHX), an antibacterial agent, which was found to inhibit the activity of MMPs 2, 8 and 9 (154), as well as natural products such as avocado, soya bean and oleic acid (150). CHX presents beneficial effects on the preservation of dentin bond strength in vivo, as an MMP inhibitor (151), when applied between the acid attack and the bonding procedures. However, the mechanisms of action of these agents and their impact on dental erosion have not been investigated yet. Therefore, this topic might be of interest in further research on the prevention of dental erosion (6).

Restorative Treatment

Depending on the degree of tooth wear, restorative treatment can range from placement of bonded composites in a few isolated areas of erosion, to full mouth reconstruction in the case of the devastated dentition. Description of the specific techniques of restoration is beyond the scope of this article. Regardless of the type of restorative therapy provided, prevention of the progression of erosion should be the basis of management of the patient with erosion. This will increase the likelihood of successful, long-term outcomes of the restorative treatment (6).

Conclusion

There is an increasing awareness in clinical dentistry of the need to better understand the aetiology and management of tooth wear (152) as increasing numbers of elderly patients are retaining their natural teeth to a stage when they present with extensive wear (2). In addition, more younger patients zubima (153, 154). Preporučuje se da se kliničko rješavanje trošenja zuba usredotoči na rano otkrivanje i prevenciju, a ne na razmišljanja o restorativnom pristupu (2).

Na osnovi podataka iz istraživanja *in vitro* i *in situ*, preventivne mjere za pacijente pogođene erozijom uključuju savjete o prehrani, stimulaciju protoka sline, fluoridaciju, modifikaciju erozivnih napitaka i odgovarajuće mjere oralne higijene (6).

Rano otkrivanje erozije važno je za uspješnu brigu i prevenciju progresije bolesti. Predstavili smo kratak pregled etioloških čimbenika te dali preporuke za procjenu i brigu o pacijentima s erozivnim promjenama. To uključuje cjelovitu medicinsku povijest bolesti kako bi se, uz ostale oblike trošenja zuba, mogli identificirati mogući rizični čimbenici. Važno je i za određivanje etiologije i pomaže usmjeriti terapiju. Bez obzira na to može li se ili ne odrediti etiologija, treba se primijeniti preventivni protokol. Pacijenta se mora redovito nadzirati uz pomoć fotografija ili otisaka te modela zuba, kako bi se odredila prava metoda i postigao uspjeh. Za to je idealan tim primarne stomatološke zaštite, jer može uspješno pružiti tu uslugu pacijentima sa zubnom erozijom i drugim oblicima trošenja zuba (9).

Potrebne su daljne kliničke studije kako bi se potvrdila vrijednost tih mjera. Zbog toga što se erozivno trošenje zuba ne može potpuno prevenirati preporučenim mjerama, potrebno je razviti nove s još većim zaštitnim potencijalom i kliničkom prihvatljivošću.

are presenting with wear of both primary and permanent teeth (153,154). It has been suggested that clinical management of tooth wear should focus on early detection and prevention before a restorative approach is considered (2).

From the available data of in vitro and in situ studies, preventive strategies for patients suffering from erosion include dietary advice, stimulation of salivary flow, optimization of fluoride regimens, modification of erosive beverages and adequate oral hygiene measures (6).

Early recognition of erosion is important to successfully manage and prevent disease progression. A brief review of etiologic factors has been presented and recommendations made for evaluation and management of the patient with erosion. These include a complete problem and medical history aimed at identifying possible risk factors, including those for other forms of tooth wear. This is important to determine the etiology and help direct treatment. Whether or not an etiology can be determined, a prevention protocol for prevention of progression of erosion should be initiated. The patient should be monitored at regular intervals by photographs or impressions of the dentition to determine compliance and success of treatment. The primary dental care team is in the ideal position to provide this care for their patients with dental erosion and other forms of tooth wear (9).

However, clinical trials are required to confirm the relevance of these measures. As erosive tooth wear cannot be prevented totally with the recommended strategies, further research is necessary to develop new measures with higher protective capabilities and good clinical acceptance.

Abstract

Dental erosion is defined as irreversible loss of dental hard tissue by a chemical process (acid acid and/or chelation exposure) that does not involve bacteria. The acidic attack leads to an irreversible loss of dental hard tissue, which is accompanied by a progressive softening of the surface. This softened zone is more susceptible to mechanical forces, such as abrasion , which in turn have little or no effect on sound dental hard tissues. The chemical and mechanical processes can occur individually or together, although the effect of erosion is often dominant. The etiology of erosion is multifactorial and not fully understood. The studies should clearly delineate erosion, attrition, and abrasion with identification of etiologic factors. There are different predisposing factors and aetiologies of the erosive condition. As erosive tooth wear is a multifactorial condition, preventive strategies have to be applied which account for chemical, biological and behavioral factors involved in the etiology and pathogenesis of erosion There is an increasing awareness in clinical dentistry of the need to better understand the aetiology and management of tooth wear as increasing numbers of elderly patients are retaining their natural teeth to a stage when they present with extensive wear. The clinical management of erosion should focus on early detection and prevention before a restorative approach is considered. Early recognition of erosion is important to successfully manage and prevent disease progression.

Received: August 7, 2009
Accepted: August 25, 2009

Address for correspondence
Prof Dr. Betul Kargul
Marmara University,
Dental School
Department of Pediatric Dentistry
Güzelbahçe Büyükçiftlik Sokak
No.6 3436
Istanbul, Turkey
Tel:0212 231 91 20/109
Fax: 0 212 246 52 47
bkargul@marmara.edu.tr

Key words
Tooth Erosion; Tooth Attrition; Tooth Abrasion

References

1. Pindborg JJ. Pathology of dental hard tissues. Copenhagen: Munksgaard; 1970. p. 312-21.
2. Lussi A. Erosive tooth wear - a multifactorial condition of growing concern and increasing knowledge. Monogr Oral Sci. 2006;20:1-8.
3. Rios D, Honório HM, Magalhães AC, Buzalaf MA, Palma-Dibb RG, Machado MA, et al. Influence of toothbrushing on enamel softening and abrasive wear of eroded bovine enamel: an in situ study. Braz Oral Res. 2006;20(2):148-54.
4. Addy M, Hunter ML. Can tooth brushing damage your health? Effects on oral and dental tissues. Int Dent J. 2003;53 Suppl 3:177-86.
5 Addy M, Shellis RP. Interaction between attrition, abrasion and erosion in tooth wear. Monogr Oral Sci. 2006;20:17-31.
6. Magalhães AC, Wiegand A, Rios D, Honório HM, Buzalaf MA. Insights into preventive measures for dental erosion. J Appl Oral Sci. 2009;17(2):75-86.
7. Lussi A, Schaffner M, Hotz P, Suter P. Dental erosion in a population of Swiss adults. Community Dent Oral Epidemiol. 1991;19(5):286-90.
8. O'Brien M. Children's dental health in the United Kingdom 1993. Office of Population Censuses and Surveys. London: Her Majesty's Stationery Office; 1994.
9. Gandara BK, Truelove EL. Diagnosis and management of dental erosion. J Contemp Dent Pract. 1999;1(1):16-23.
10. Caglar E, Kargul B, Tanboga I, Lussi A. Dental erosion among children in an Istanbul public school. J Dent Child (Chic). 2005;72(1):5-9.
11. Al-Dlaigan YH, Shaw L, Smith A. Dental erosion in a group of British 14-year-old, school children. Part I: Prevalence and influence of differing socioeconomic backgrounds. Br Dent J. 2001;190(3):145-9.
12. Bardsley PF, Taylor S, Milosevic A. Epidemiological studies of tooth wear and dental erosion in 14-year-old children in North West England. Part 1: The relationship with water fluoridation and social deprivation. Br Dent J. 2004;197(7):413-6.
13. Bartlett DW, Coward PY, Nikkah C, Wilson RF. The prevalence of tooth wear in a cluster sample of adolescent schoolchildren and its relationship with potential explanatory factors. Br Dent J. 1998;184(3):125-9.
14. Dugmore CR, Rock WP. The prevalence of tooth erosion in 12-year-old children. Br Dent J. 2004;196(5):279-82.
15. Bartlett D, Phillips K, Smith B. A difference in perspective--the North American and European interpretations of tooth wear. Int J Prosthodont. 1999;12(5):401-8.
16. Azzopardi A, Bartlett DW, Watson TF, Sherriff M. The surface effects of erosion and abrasion on dentine with and without a protective layer. Br Dent J. 2004;196(6):351-4.
17. Smith BG, Robb ND. The prevalence of toothwear in 1007 dental patients. J Oral Rehabil. 1996;23(4):232-9.
18. Bartlett D, Dugmore C. Pathological or physiological erosion--is there a relationship to age? Clin Oral Investig. 2008;12 Suppl 1:S27-31.
19. Lussi A, Jaeggi T. Erosion--diagnosis and risk factors. Clin Oral Investig. 2008;12 Suppl 1:S5-13.
20. Lussi A, Jaeggi T, Zero D. The role of diet in the aetiology of dental erosion. Caries Res. 2004;38 Suppl 1:34-44.
21. Bartlett D. Intrinsic causes of erosion. Monogr Oral Sci. 2006;20:119-39.
22. Larsen MJ. Chemical events during tooth dissolution. J Dent Res. 1990;69 Spec No:575-80.
23. Featherstone JD, Lussi A. Understanding the chemistry of dental erosion. Monogr Oral Sci. 2006;20:66-76.
24. Ganss C. How valid are current diagnostic criteria for dental erosion? Clin Oral Investig. 2008;12 Suppl 1:S41-9.
25. Hefferren JJ. Why is there and should there be more attention paid to dental erosion? Compend Contin Educ Dent. 2004;25(9 Suppl 1):4-8.
26. ten Cate JM, Imfeld T. Dental erosion, summary. Eur J Oral Sci. 1996;104(2 (Pt 2)):241-4.
27. Asher C, Read MJ. Early enamel erosion in children associated with the excessive consumption of citric acid. Br Dent J. 1987;162(10):384-7.
28. Lazarchik DA, Frazier KB. Dental erosion and acid reflux disease: an overview. Gen Dent. 2009;57(2):151-6.
29. Knewitz JL, Drisko CL. Anorexia nervosa and bulimia: a review. Compendium. 1988;9(3):244-7.
30. Järvinen VK, Rytömaa II, Heinonen OP. Risk factors in dental erosion. J Dent Res. 1991;70(6):942-7.
31. Cavadini C, Siega-Riz AM, Popkin BM. US adolescent food intake trends from 1965 to 1996. Arch Dis Child. 2000;83(1):18-24.
32. Gleason P, Suitor C. Children's diets in the mid-1990s: dietary intake and its relationship with school meal participation. Alexandria: US Department of agriculture, food and nutrition service, office of analysis, nutrition and evaluation; 2001.
33. Lussi A, Schaffner M. Progression of and risk factors for dental erosion and wedge-shaped defects over a 6-year period. Caries Res. 2000;34(2):182-7.
34. O'Sullivan EA, Curzon ME. A comparison of acidic dietary factors in children with and without dental erosion. ASDC J Dent Child. 2000;67(3):186-92, 160.
35. Bartlett D. The implication of laboratory research on tooth wear and erosion. Oral Dis. 2005;11(1):3-6.
36. Caglar E, Lussi A, Kargul B, Ugur K. Fruit yogurt: any erosive potential regarding teeth? Quintessence Int. 2006;37(8):647-51.
37. Kargul B, Caglar E, Lussi A. Erosive and buffering capacities of yogurt. Quintessence Int. 2007;38(5):381-5.
38. Bowen WH, Lawrence RA. Comparison of the cariogenicity of cola, honey, cow milk, human milk, and sucrose. Pediatrics. 2005;116(4):921-6.
39. Edwards M, Creanor SL, Foye RH, Gilmour WH. Buffering capacities of soft drinks: the potential influence on dental erosion. J Oral Rehabil. 1999;26(12):923-7.
40. Owens BM. The potential effects of pH and buffering capacity on dental erosion. Gen Dent. 2007;55(6):527-31.
41. Rios D, Honório HM, Magalhães AC, Buzalaf MA, Palma-Dibb RG, Machado MA, et al. Influence of toothbrushing on enamel softening and abrasive wear of eroded bovine enamel: an in situ study. Braz Oral Res. 2006;20(2):148-54.
42. Magalhães AC, Rios D, Moino AL, Wiegand A, Attin T, Buzalaf MA. Effect of different concentrations of fluoride in dentifrices on dentin erosion subjected or not to abrasion in situ/ex vivo. Caries Res. 2008;42(2):112-6.
43. Turssi CP, Faraoni JJ, Rodrigues Jr AL, Serra MC. An in situ investigation into the abrasion of eroded dental hard tissues by a whitening dentifrice. Caries Res. 2004;38(5):473-7.
44. Cheng R, Yang H, Shao MY, Hu T, Zhou XD. Dental erosion and severe tooth decay related to soft drinks: a case report and literature review. J Zhejiang Univ Sci B. 2009;10(5):395-9.
45. Zero DT, Lussi A. Etiology of enamel erosion—intrinsic and extrinsic factors. In: Addy M, Embery G, Edgar WM, Orchardson R, editors. Tooth wear and sensitivity. Clinical advances in restorative dentistry. London: Martin Dunitz; 2000. p. 121–39.

47. Hannig C, Hannig M, Attin T. Enzymes in the acquired enamel pellicle. Eur J Oral Sci. 2005;113(1):2-13.
48. Lendenmann U, Grogan J, Oppenheim FG. Saliva and dental pellicle--a review. Adv Dent Res. 2000;14:22-8.
49. O'Sullivan EA, Curzon ME. A new index for measurement of erosion in children. Caries Research. 1996;30(4):274.
50. Amaechi BT, Higham SM. Dental erosion: possible approaches to prevention and control. J Dent. 2005;33(3):243-52.
51. Barbour ME, Rees JS. The laboratory assessment of enamel erosion: a review. J Dent. 2004;32(8):591-602.
52. Eisenburger M, Hughes J, West NX, Jandt KD, Addy M. Ultrasonication as a method to study enamel demineralisation during acid erosion. Caries Res. 2000;34(4):289-94.
53. Arends J, ten Bosch JJ. Demineralization and remineralization evaluation techniques. J Dent Res. 1992;71 Spec No:924-8.
54. Lussi A, Hellwig E. Erosive potential of oral care products. Caries Res. 2001;35 Suppl 1:52-6.
55. Amaechi BT, Higham SM, Edgar WM. Factors influencing the development of dental erosion in vitro: enamel type, temperature and exposure time. J Oral Rehabil. 1999;26(8):624-30.
56. Pretty IA, Edgar WM, Higham SM. The validation of quantitative light-induced fluorescence to quantify acid erosion of human enamel. Arch Oral Biol. 2004;49(4):285-94.
57. Elton V, Cooper L, Higham SM, Pender N. Validation of enamel erosion in vitro. J Dent. 2009;37(5):336-41.
58. Eccles JD. Dental erosion of nonindustrial origin. A clinical survey and classification. J Prosthet Dent. 1979;42(6):649-53.
59. Smith BG, Knight JK. An index for measuring the wear of teeth. Br Dent J. 1984;156(12):435-8.
60. Ganss C, Lussi A. Diagnosis of erosive tooth wear. In: Lussi A, editor. Dental erosion. Basel: Karger; 2006. p. 32–43.
61. Berg-Beckhoff G, Kutschmann M, Bardehle D. Methodological considerations concerning the development of oral dental erosion indexes: literature survey, validity and reliability. Clin Oral Investig. 2008;12 Suppl 1:S51-8.
62. Milosevic A, Bardsley PF, Taylor S. Epidemiological studies of tooth wear and dental erosion in 14-year old children in North West England. Part 2: The association of diet and habits. Br Dent J. 2004;197(8):479-83.
63. Lussi A. Dental erosion clinical diagnosis and case history taking. Eur J Oral Sci. 1996;104(2 (Pt 2)):191-8.
64. Ganss C, Klimek J, Giese K. Dental erosion in children and adolescents--a cross-sectional and longitudinal investigation using study models. Community Dent Oral Epidemiol. 2001;29(4):264-71.
65. Aine L, Baer M, Mäki M. Dental erosions caused by gastroesophageal reflux disease in children. ASDC J Dent Child. 1993;60(3):210-4.
66. Larsen IB, Westergaard J, Stoltze K, Larsen AI, Gyntelberg F, Holmstrup P. A clinical index for evaluating and monitoring dental erosion. Community Dent Oral Epidemiol. 2000;28(3):211-7.
67. O'Sullivan EA. A new index for the measurement of erosion in children. Eur J Paediatr Dent. 2000;1(2):69–74.
68. Bartlett D, Ganss C, Lussi A. Basic Erosive Wear Examination (BEWE): a new scoring system for scientific and clinical needs. Clin Oral Investig. 2008;12 Suppl 1:S65-8.
69. Zero DT, Lussi A. Behavioral factors. Monogr Oral Sci. 2006;20:100-5.
70. 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(3):216-22.
71. Attin T, Siegel S, Buchalla W, Lennon AM, Hannig C, Becker K. Brushing abrasion of softened and remineralised dentin: an in situ study. Caries Res. 2004;38(1):62-6.
72. Hooper S, West NX, Pickles MJ, Joiner A, Newcombe RG, Addy M. Investigation of erosion and abrasion on enamel and dentine: a model in situ using toothpastes of different abrasivity. J Clin Periodontol. 2003;30(9):802-8.
73. Wiegand A, Attin T. Occupational dental erosion from exposure to acids: a review. Occup Med (Lond). 2007;57(3):169-76.
74. Vieira A, Overweg E, Ruben JL, Huysmans MC. Toothbrush abrasion, simulated tongue friction and attrition of eroded bovine enamel in vitro. J Dent. 2006;34(5):336-42.
75. Wiegand A, Egert S, Attin T. Toothbrushing before or after an acidic challenge to minimize tooth wear? An in situ/ex vivo study. Am J Dent. 2008;21(1):13-6.
76. Ganss C, Schlueter N, Friedrich D, Klimek J. Efficacy of waiting periods and topical fluoride treatment on toothbrush abrasion of eroded enamel in situ. Caries Res. 2007;41(2):146-51.
77. Wiegand A, Lemmrich F, Attin T. Influence of rotating-oscillating, sonic and ultrasonic action of power toothbrushes on abrasion of sound and eroded dentine. J Periodontal Res. 2006;41(3):221-7.
78. Wiegand A, Köwing L, Attin T. Impact of brushing force on abrasion of acid-softened and sound enamel. Arch Oral Biol. 2007;52(11):1043-7.
79. Wiegand A, Begic M, Attin T. In vitro evaluation of abrasion of eroded enamel by different manual, power and sonic toothbrushes. Caries Res. 2006;40(1):60-5.
80. Wiegand A, Schwerzmann M, Sener B, Magalhaes AC, Roos M, Ziebolz D, et al. Impact of toothpaste slurry abrasivity and toothbrush filament stiffness on abrasion of eroded enamel - an in vitro study. Acta Odontol Scand. 2008;66(4):231-5.
81. Hooper S, West NX, Pickles MJ, Joiner A, Newcombe RG, Addy M. Investigation of erosion and abrasion on enamel and dentine: a model in situ using toothpastes of different abrasivity. J Clin Periodontol. 2003;30(9):802-8.
82. Magalhães AC, Rios D, Delbem AC, Buzalaf MA, Machado MA. Influence of fluoride dentifrice on brushing abrasion of eroded human enamel: an in situ/ex vivo study. Caries Res. 2007;41(1):77-9.
83. Hara AT, Lussi A, Zero DT. Biological factors. Monogr Oral Sci. 2006;20:88-99.
84. Hall AF, Buchanan CA, Millett DT, Creanor SL, Strang R, Foye RH. The effect of saliva on enamel and dentine erosion. J Dent. 1999;27(5):333-9.
85. Meurman JH, Frank RM. Scanning electron microscopic study of the effect of salivary pellicle on enamel erosion. Caries Res. 1991;25(1):1-6.
86. Rios D, Honório HM, Magalhães AC, Delbem AC, Machado MA, Silva SM, et al. Effect of salivary stimulation on erosion of human and bovine enamel subjected or not to subsequent abrasion: an in situ/ex vivo study. Caries Res. 2006;40(3):218-23.
87. Gedalia I, Dakuar A, Shapira L, Lewinstein I, Goultschin J, Rahamim E. Enamel softening with Coca-Cola and rehardening with milk or saliva. Am J Dent. 1991;4(3):120-2.
88. Ponduri S, Macdonald E, Addy M. A study in vitro of the combined effects of soft drinks and tooth brushing with fluoride toothpaste on the wear of dentine. Int J Dent Hyg. 2005;3(1):7-12.
89. Hannig M, Balz M. Protective properties of salivary pellicles from two different intraoral sites on enamel erosion. Caries Res. 2001;35(2):142-8.

90. Ganss C, Klimek J, Brune V, Schürmann A. Effects of two fluoridation measures on erosion progression in human enamel and dentine in situ. Caries Res. 2004;38(6):561-6.
91. Ganss C, Klimek J, Starck C. Quantitative analysis of the impact of the organic matrix on the fluoride effect on erosion progression in human dentine using longitudinal microradiography. Arch Oral Biol. 2004;49(11):931-5.
92. Saxegaard E, Rölla G. Fluoride acquisition on and in human enamel during topical application in vitro. Scand J Dent Res. 1988;96(6):523-35.
93. Lagerweij MD, Buchalla W, Kohnke S, Becker K, Lennon AM, Attin T. Prevention of erosion and abrasion by a high fluoride concentration gel applied at high frequencies. Caries Res. 2006;40(2):148-53.
94. Mok TB, McIntyre J, Hunt D. Dental erosion: in vitro model of wine assessor's erosion. Aust Dent J. 2001;46(4):263-8.
95. Jones L, Lekkas D, Hunt D, McIntyre J, Rafir W. Studies on dental erosion: An in vivo-in vitro model of endogenous dental erosion--its application to testing protection by fluoride gel application. Aust Dent J. 2002;47(4):304-8.
96. Hove L, Holme B, Øgaard B, Willumsen T, Tveit AB. The protective effect of TiF4, SnF2 and NaF on erosion of enamel by hydrochloric acid in vitro measured by white light interferometry. Caries Res. 2006;40(5):440-3.
97. Willumsen T, Ogaard B, Hansen BF, Rølla G. Effects from pretreatment of stannous fluoride versus sodium fluoride on enamel exposed to 0.1 M or 0.01 M hydrochloric acid. Acta Odontol Scand. 2004;62(5):278-81.
98. Attin T, Zirkel C, Hellwig E. Brushing abrasion of eroded dentin after application of sodium fluoride solutions. Caries Res. 1998;32(5):344-50.
99. Attin T, Siegel S, Buchalla W, Lennon AM, Hannig C, Becker K. Brushing abrasion of softened and remineralised dentin: an in situ study. Caries Res. 2004;38(1):62-6.
100. Li H, Watson TF, Sherriff M, Curtis R, Bartlett DW. The influence of fluoride varnish on the attrition of dentine. Caries Res. 2007;41(3):219-22.
101. Kaidonis JA, Gratiaen J, Bhatia N, Richards LC, Townsend GC. Tooth wear prevention: a quantitative and qualitative in vitro study. Aust Dent J. 2003;48(1):15-9.
102. Hove LH, Young A, Tveit AB. An in vitro study on the effect of TiF(4) treatment against erosion by hydrochloric acid on pellicle-covered enamel. Caries Res. 2007;41(1):80-4.
103. Magalhães AC, Kato MT, Rios D, Wiegand A, Attin T, Buzalaf MA. The effect of an experimental 4% Tif4 varnish compared to NaF varnishes and 4% TiF4 solution on dental erosion in vitro. Caries Res. 2008;42(4):269-74.
104. Hughes JA, Jandt KD, Baker N, Parker D, Newcombe RG, Eisenburger M, et al. Further modification to soft drinks to minimise erosion. A study in situ. Caries Res. 2002;36(1):70-4.
105. Barbour ME, Parker DM, Allen GC, Jandt KD. Enamel dissolution in citric acid as a function of calcium and phosphate concentrations and degree of saturation with respect to hydroxyapatite. Eur J Oral Sci. 2003;111(5):428-33.
106. Barbour ME, Parker DM, Allen GC, Jandt KD. Human enamel erosion in constant composition citric acid solutions as a function of degree of saturation with respect to hydroxyapatite. J Oral Rehabil. 2005;32(1):16-21.
107 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(1):81-7.
108. Larsen MJ, Richards A. Fluoride is unable to reduce dental erosion from soft drinks. Caries Res. 2002;36(1):75-80.
109. Fejerskov O. Changing paradigms in concepts on dental caries: consequences for oral health care. Caries Res. 2004;38(3):182-91.
110. Cooley WE. Reactions of tin (II) and fluoride ions with etched enamel. J Dent Res. 1961;40(6):1199–210.
111. Brambilla E. Fluoride - is it capable of fighting old and new dental diseases? An overview of existing fluoride compounds and their clinical applications. Caries Res. 2001;35 Suppl 1:6-9.
112. Babcock FD, King JC, Jordan TH. The reaction of stannous fluoride and hydroxyapatite. J Dent Res. 1978;57(9-10):933-8.
113. Krutchkoff DJ, Jordan TH, Wei SH, Nordquist WD. Surface characterization of the stannous fluoride-enamel interaction. Arch Oral Biol. 1972;17(6):923-30.
114. Wachtel LW. In vitro comparison of effects of topical stannic fluoride and stannous fluoride solutions on enamel solubility. Arch Oral Biol. 1964;32:439-45.
115. Gray JA. Acid dıssolutıon rate of sound and whıte-spot enamel treated wıth tın(ıı) and fluorıde compounds. J Dent Res. 1965;44:493-501.
116. White DJ. A "return" to stannous fluoride dentifrices. J Clin Dent. 1995;6 Spec No:29-36.
117. Azzopardi A, Bartlett DW, Watson TF, Sherriff M. The surface effects of erosion and abrasion on dentine with and without a protective layer. Br Dent J. 2004;196(6):351-4.
118. Ganss C, Klimek J, Brune V, Schürmann A. Effects of two fluoridation measures on erosion progression in human enamel and dentine in situ. Caries Res. 2004;38(6):561-6.
119. Mäkinen KK, Söderling E. Solubility of calcium salts, enamel, and hydroxyapatite in aqueous solutions of simple carbohydrates. Calcif Tissue Int. 1984;36(1):64-71.
120. Arends J, Christoffersen J, Schuthof J, Smits MT. Influence of xylitol on demineralization of enamel. Caries Res. 1984;18(4):296-301.
121. Buzalaf MA, de Moraes Italiani F, Kato MT, Martinhon CC, Magalhães AC. Effect of iron on inhibition of acid demineralisation of bovine dental enamel in vitro. Arch Oral Biol. 2006;51(10):844-8.
122. Bachra BN, van Harskamp GA. The effect of polyvalent metal ions on the stability of a buffer system for calcification in vitro. Calcif Tissue Res. 1970;4(4):359-65.
123. Kato MT, Sales-Peres SH, Buzalaf MA. Effect of iron on acid demineralisation of bovine enamel blocks by a soft drink. Arch Oral Biol. 2007;52(11):1109-11.
124. Lennon AM, Pfeffer M, Buchalla W, Becker K, Lennon S, Attin T. Effect of a casein/calcium phosphate-containing tooth cream and fluoride on enamel erosion in vitro. Caries Res. 2006;40(2):154-7.
125. Rees J, Loyn T, Chadwick B. Pronamel and tooth mousse: an initial assessment of erosion prevention in vitro. J Dent. 2007;35(4):355-7.
126. Piekarz C, Ranjitkar S, Hunt D, McIntyre J. An in vitro assessment of the role of Tooth Mousse in preventing wine erosion. Aust Dent J. 2008;53(1):22-5.
127. Ramalingam L, Messer LB, Reynolds EC. Adding casein phosphopeptide-amorphous calcium phosphate to sports drinks to eliminate in vitro erosion. Pediatr Dent. 2005;27(1):61-7.
128. Ranjitkar S, Narayana T, Kaidonis JA, Hughes TE, Richards LC, Townsend GC. The effect of casein phosphopeptide-amorphous calcium phosphate on erosive dentine wear. Aust Dent J. 2009;54(2):101-7.

129. Ranjitkar S, Rodriguez JM, Kaidonis JA, Richards LC, Townsend GC, Bartlett DW. The effect of casein phosphopeptide-amorphous calcium phosphate on erosive enamel and dentine wear by toothbrush abrasion. J Dent. 2009;37(4):250-4.
130. Ranjitkar S, Kaidonis JA, Richards LC, Townsend GC. The effect of CPP-ACP on enamel wear under severe erosive conditions. Arch Oral Biol. 2009;54(6):527-32.
131. Reynolds EC, Cai F, Shen P, Walker GD. Retention in plaque and remineralization of enamel lesions by various forms of calcium in a mouthrinse or sugar-free chewing gum. J Dent Res. 2003;82(3):206-11.
132. Kumar VL, Itthagarun A, King NM. The effect of casein phosphopeptide-amorphous calcium phosphate on remineralization of artificial caries-like lesions: an in vitro study. Aust Dent J. 2008;53(1):34-40.
133. Rahiotis C, Vougiouklakis G. Effect of a CPP-ACP agent on the demineralization and remineralization of dentine in vitro. J Dent. 2007;35(8):695-8.
134. Reynolds EC, Walsh LJ. Additional aids to remineralisation of tooth structure. In: Mount GJ, Hume WR, editors. Preservation and restoration of tooth structure. Queensland: Knowledge Books and Software; 2005. p. 111–8.
135. Tantbirojn D, Huang A, Ericson MD, Poolthong S. Change in surface hardness of enamel by a cola drink and a CPP-ACP paste. J Dent. 2008;36(1):74-9.
136. Eisenburger M, Addy M, Hughes JA, Shellis RP. Effect of time on the remineralisation of enamel by synthetic saliva after citric acid erosion. Caries Res. 2001;35(3):211-5.
137. Reynolds EC. Remineralization of enamel subsurface lesions by casein phosphopeptide-stabilized calcium phosphate solutions. J Dent Res 1997;76(9):1587-95.
138. Nelson DG, Wefel JS, Jongebloed WL, Featherstone JD. Morphology, histology and crystallography of human dental enamel treated with pulsed low-energy infrared laser radiation. Caries Res. 1987;21(5):411-26.
139. Castellan CS, Luiz AC, Bezinelli LM, Lopes RM, Mendes FM, De P Eduardo C, et al. In vitro evaluation of enamel demineralization after Er:YAG and Nd:YAG laser irradiation on primary teeth. Photomed Laser Surg. 2007;25(2):85-90.
140. Tsai CL, Lin YT, Huang ST, Chang HW. In vitro acid resistance of CO2 and Nd-YAG laser-treated human tooth enamel. Caries Res. 2002;36(6):423-9.
141. Naylor F, Aranha AC, Eduardo Cde P, Arana-Chavez VE, Sobral MA. Micromorphological analysis of dentinal structure after irradiation with Nd:YAG laser and immersion in acidic beverages. Photomed Laser Surg. 2006;24(6):745-52.
142. Buzalaf MA, Rios D, Magalhães AC, Machado MA, Silva SM, Lizarelli RF, et al. The effect of Nd:YAG irradiation and fluoride application on enamel resistance to erosion. Caries Res. 2008;42(3):191.
143. Magalhães AC, Rios D, Machado MA, Da Silva SM, Lizarelli Rde F, Bagnato VS, et al. Effect of Nd:YAG irradiation and fluoride application on dentine resistance to erosion in vitro. Photomed Laser Surg. 2008;26(6):559-63.
144. Reynolds JJ, Meikle MC. The functional balance of metalloproteinases and inhibitors in tissue degradation: relevance to oral pathologies. J R Coll Surg Edinb. 1997;42(3):154-60.
145. Chaussain-Miller C, Fioretti F, Goldberg M, Menashi S. The role of matrix metalloproteinases (MMPs) in human caries. J Dent Res. 2006;85(1):22-32.
146. Tjäderhane L, Larjava H, Sorsa T, Uitto VJ, Larmas M, Salo T. The activation and function of host matrix metalloproteinases in dentin matrix breakdown in caries lesions. J Dent Res. 1998;77(8):1622-9.
147. Baker AH, Edwards DR, Murphy G. Metalloproteinase inhibitors: biological actions and therapeutic opportunities.Baker AH, Edwards DR, Murphy G. J Cell Sci. 2002;115(Pt 19):3719-27.
148. Chaussain-Miller C, Fioretti F, Goldberg M, Menashi S. The role of matrix metalloproteinases (MMPs) in human caries. J Dent Res. 2006;85(1):22-32.
149. Kato MT, Magalhães AC, Rios D, Attin T, Buzalaf MA. The protective effect of green tea on dentin erosion and abrasion: an in situ study. Caries Res. 2008;42(3):188.
150. Gendron R, Grenier D, Sorsa T, Mayrand D. Inhibition of the activities of matrix metalloproteinases 2, 8, and 9 by chlorhexidine. Clin Diagn Lab Immunol. 1999;6(3):437-9.
151. Carrilho MR, Geraldeli S, Tay F, de Goes MF, Carvalho RM, Tjäderhane L, et al. In vivo preservation of the hybrid layer by chlorhexidine. J Dent Res. 2007;86(6):529-33.
152. Nunn JH. Prevalence and distribution of tooth wear. In: Addy M, Embery G, Edgar WM, Orchardson R, editors. Tooth wear and sensitivity: clinical advances in restorative dentistry. London: Martin Dunitz Ltd; 2000. p. 93–103.
153. Khan F, Young WG, Daley TJ. Dental erosion and bruxism. A tooth wear analysis from south east Queensland. Aust Dent J. 1998;43(2):117-27.
154. Jaeggi T, Lussi A. Prevalence, incidence and distribution of erosion. Monogr Oral Sci. 2006;20:44-65.
155. Zheng J, Xiao F, Qian LM, Zhou ZR. Erosion behavior of human tooth enamel in citric acid solution. Tribol Int. 2009.