Noncarious cervical lesions – A clinical concept based on the literature review. Part 1: Prevention

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ABSTRACT: Purpose: Due to an increased prevalence of non-carious cervical lesions (NCCL), a clinical strategy for this lesion type should be considered. Previous reviews focused mainly on etiology and prevalence. In Part 1 of this paper, an evidence-based support for a preventive strategy of NCCL was elaborated. **Methods:** Literature over the last 10 years available in the MEDLINE database was reviewed in order to find clinical evidence for a preventive approach to NCCL. Recommendations were based primarily on systematic reviews, clinical evaluations and a monograph. **Results:** The etiology of NCCL is currently considered to be rather multifactorial, as clinical investigations found multiple factors associated with this type of lesions and due to the lack of evidence to support exclusively one or another factor. Based on the hypothesis of multifactorial origin, a preventive protocol has been established. No clinical research exists with respect to the prevention of NCCL and long-term clinical evaluations of the proposed preventive measures are needed. (*Am J Dent* 2011;24:49-56).

CLINICAL SIGNIFICANCE: The slow progression, the high capacity of self-defense by producing sclerotic dentin, and the lack of evidence for tooth weakening in the absence of a restoration are evidence-based findings supporting a "wait and see" philosophy. Restoration could be postponed in the absence of esthetic demands, sensitivity or threat to the integrity of the tooth. Restoration should not always be the first treatment of choice, although there still remains to be established to what extent prevention could replace restoration.

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Introduction

There is an open debate as to which degree tooth wear becomes a pathological condition, and when treatment should be initiated, knowing that tooth substance loss is to some degree a physiological process, being a compensatory mechanism built into our biological system.[1] But, in today's context of increased life expectancy and of an increasing number of people retaining their teeth longer, the consequences of ignoring early signs of such lesions may be severe. Considering that the objective of any preventive or restorative procedure is to maintain tooth function throughout life and to prevent premature destruction, a change of approach concerning noncarious tooth substance loss is needed. Knowing the relatively limited durability of restorative procedures, the approach should not only include a restorative treatment of the already advanced lesion, but also preventive therapy as well. Previous reviews of non-carious cervical lesions (NCCL) focused mainly on restorative options and etiology,[2-12] but no clear guidelines for a preventive approach could be found. Therefore, in Part 1 of this paper an evidence-based support for a preventive strategy of NCCL was searched and a preventive concept was established.

Materials and Methods

The pertinent literature over the last 10 years available in MEDLINE database was reviewed. A search for the following keywords was performed: abfraction, cervical non-carious lesions, cervical erosion, etiology cervical lesions, cervical abrasion, cervical tooth wear and prevalence of non-carious lesions. Recommendations were based primarily on systematic reviews, clinical evaluations and a monograph, in order to detect the prevalence, tooth groups affected, associated risk factors and etiology of NCCL. *In vitro* evaluations of products were considered when clinical trials could not be found.

Answers to the following questions were searched: Are there factors associated with NCCL and could these lesions be prevented by an early detection of these factors? Are there any available preventive measures capable of preventing progression of NCCL? An update on etiology and prevalence of NCCL was also included.

DEFINITION AND CLINICAL APPEARANCE

NCCL are defined as a loss of hard dental tissue close to the cemento-enamel-junction (CEJ) with the distinctive characteristic of hard-mineralized tissue presence, in contrast to caries.[2,3] Commonly, their shape is like a wedge with the apex pointing inwards, but they can appear in various shapes.[3] Historically, NCCL were classified according to their appearance: wedge-shaped, disc-shaped, flattened, irregular, and figured areas. Generally, NCCL vary from shallow grooves to large wedge-shaped defects with sharp line angles.[2] A link between the morphological characteristics of the lesions and the main etiological factor has been suspected.[10,11,13,14,18] Thus, a U-shaped or disk-shaped broad and shallow lesion, with poorly defined margins and adjacent smooth enamel points out an extrinsic erosive cause such as ingestion of acidic foods, beverages, and medication.[11,15] It is important to notice that shallow defects on smooth surfaces coronal to the CEJ are considered to be the best predictive criteria for the diagnosis of erosion, and a pathognomonic sign of erosive tooth wear.[16,17] Lesions caused by abrasive forces, such as improper toothbrushing techniques, generally exhibit sharp defined margins and a hard surface with traces of scratching. Lesions caused by abfraction mechanism due to abnormal occlusal loading would typically be wedge-shaped or saucer-shaped lesions, with sharp internal angles and an apical extent relative to the CEJ.[11] However, the shape cannot be considered predictive for the etiology.[12]

Clinical studies and observations[2,10,28,19] have shown that

Table 1. Etiological mechanisms of NCCL.

Erosive mechanism (Corrosion)	Chemical wear as a result of extrinsic or intrinsic acids or chelators acting on plaque free tooth surfaces. Factors: acidic beverages and foods, acidic medication, gastro esophageal disease with reflux, factors predisposing to gastric reflux (hiatus hernia, sport activities) anorexia, bulimia nervosa, professional exposure to acids (wine tasters).[1,2,10,50,63]
Abrasion mechanism (Exogenous friction)	Physical wear as a result of a mechanical process involving foreign objects. Factors: abrasive toothpaste, improper tooth brushing with a horizontal technique and excessive force, particular dietary habits[1,2,10,23,50]
Abfraction mechanism (Stress)	Physical wear as a result of tensile or shear stress in the cement enamel junction area provoking microfractures in enamel and dentin (fatigue wear). Factors: parafunctions, bruxism, excessive functional load, off axis load.[9,10,31,34,42]
Piezoelectric effect	Acquisition of a surface electrical charge under load causing demineralization.[10,52]
Stress corrosion	Tooth substance loss due to acid in combination with stress. Acid in areas of stress concentration results in either static stress corrosion or cyclic (fatigue) stress corrosion.[8,10,32,52]

NCCL are almost exclusively situated on the facial surfaces of teeth, seldom on lingual surfaces and rarely on proximal surfaces.

The development of NCCL tends to be a slow process that occurs over an extended period of time, among the consequences being sclerosis and lack of sensitivity.[20] Secondary dentin, occlusion of open dentin tubules, pulpal retreat and other natural tooth protective measures have slowly adapted to the noxious stimuli, thereby minimizing symptoms and maintaining pulpal integrity.[20]

THE PREVALENCE AND AFFECTED TEETH

The prevalence of cervical wear has been reported to vary between 5-85%.[10] Only a few studies described the prevalence of cervical wear alone and as investigation methods and population clusters vary, it is very difficult to compare the results obtained from different authors. Recent studies found a prevalence ranging from 11.4% to 62.2%.[13,19,21-25]

Controversy exists regarding the distribution of NCCL within dentition. A recent study reported mandibular premolars to have the highest odds ratio for developing wedge shaped defects, followed by maxillary premolars. On the other hand, compared to maxillary canines, mandibular canines proved to have a much lower odds ratio of incurring abfractions.[26] Another epidemiological evaluation reinforced these findings, reporting that the most commonly affected teeth were mandibular premolars, having also the highest percentage of high severity lesions, and among them, first premolars were most frequently affected (34.2%), followed by second premolars.[22] Telles *et al*[19] also found a higher prevalence of the lesions among mandibular teeth. Other studies[20,25] however reported maxillary teeth to be more frequently affected.

One more common finding is the fact that prevalence and severity of NCCL appears to increase with age, a hypotheses supported by the majority of studies evaluating a large number of subjects.[19,20,24-29]

ETIOLOGY

A number of theories regarding the etiological mechanism have developed over time. In the 19th century, the etiology of tooth wear in the absence of caries, including NCCL, was unexplainable, and the lesions were not categorized.[11] In 1907, Miller[30] proposed three specific categories of tooth wear – erosion, abrasion, attrition – suggesting also their possible etiology, which represents the current scientific classification for tooth wear. NCCL were first classified according to their supposed origin: erosion or abrasion.

Grippo[31] introduced later the term "abfraction", to refer to the pathological loss of dental hard tissue caused by biomechanical forces. Abfraction represents the mechanical flexure theory, according to which tooth bending phenomena due to parallel or oblique occlusal force components, occurring during parafunctions as well as during normal function, create flexural stress in the cervical area with chipping away of the hard tissues.[3,11,31,32] Tensile stress resulting from oblique occlusal forces was found to be the principal factor responsible for the disruption of the bonds between the hydroxyapatite crystals and the separation of the enamel from the dentin, even if repeated compressive forces acting together with tensile stresses are also considered to cause microfracture, fatigue, flexure, and deformation of the tooth structure.[5,6,9,10,33-35] This hypothesis was supported and reinforced also later by engineering studies.[36-42] Despite the hypothesis of abfraction having a fundamental role in the initiation of the process,[8,10] clinical studies[13,43] suggested that occlusal loading may not always be the primary factor in the formation of noncarious cervical lesions. A review of more recent literature reveals an important number of clinical investigations showing a strong correlation between bruxism, parafunctions and NCCL.[20,23,24,29,44-48] Furthermore, although engineering studies were also questioned regarding their accurate reproduction of tooth environment,[4,49] more recent tests, like dynamic finite element analysis[38,40] also provided evidence in favor of the abfraction theory. In summary, the literature supports a constant implication of occlusal stress, although rather in association with other factors like erosion/abrasion, than alone. Therefore, the etiology of these lesions is still controversial, with older studies pointing out either one or the other mechanism, while more recent studies recognize the multifactorial etiology. Therefore, there is an absence of conclusive evidence to support exclusively one etiology.[2-7,10,12,15] An important finding regarding the etiological mechanism of tooth wear in general is the enhanced effect of causal factors as a consequence of their interaction. Thus, abrasion resulting from toothbrushing or dietary habits is greater if there is a previous and recent exposure of the teeth to acidic challenge such as dietary or gastric acid.[2,32,50,51] The theory of stress corrosion considers also a combined action of occlusal stress and acid environment to be more harmful than either factor acting alone, in the development of cervical tooth loss.[8,9,32]

Controversy exists also regarding the terminology for the mechanisms involved in the etiology of tooth surface loss. While the majority of the literature refers to tooth wear etiology as erosion/abrasion/attrition or abfraction, the correct definition of the physical and chemical processes occurring on tooth surface is a source of confusion. In 2004, Grippo *et al*[32] suggested a modification of terminology by replacing the term "erosion" with "corrosion" and by defining abrasion and attrition as "friction".

Table 2. Risk factors of NCCL.

Age	Prevalence and severity of lesions increases with age.[20,22,24-26,28,29,61] Progression rate of erosion is reported also to be greater in older people.[51,68]
Factors that increase lateral and compressive forces	Wear facets, inlay restorations and occlusal cavities, altered tooth position;[10,13,19,20,26,44,57,60] group function in lateral movements and Class I Angle occlusion[20,44] or increased occlusal contact area;[25] faceting, clicking joints, occlusal splints;[24] parafunctional habits, bruxism.[20,23,24,29,44,45,47,48]
Abrasive factors	Incorrect toothbrushing habits (force and horizontal brushing technique), incorrect hygiene habits (toothbrushing immediately after ingestion of acidic foods/beverages),[15,23,25,60,61] prominent position of the tooth in the arch that leaves it prone to excessive forces from toothbrushing, adjacent teeth with similar lesions.[15]
Erosive factors	Dietary habits like in patients with vegetarian diets and those who reported consuming citrus fruits, soft drinks, alcohol, yogurt and vitamin C drinks[18,23,24,28] (exogen erosion) or acidic medication;[23] erosive tooth substance loss on occlusal or palatal surfaces (smooth silky glazed appearance of the E, grooving on occlusal surfaces) are indicators for existing acidic challenge.[1] Loss of salivary protection caused by work- and sports-related dehydration, drugs and medications and certain medical conditions.[105] Bulimic or alcoholic patients, gastro-esophageal disease (intrinsic erosion).[32,68,105]
Individual variations	Oral and dental anatomy, gingival recession, number of teeth and their mobility,[29] periodontal status or phenotype;[7] saliva properties (amount, flow capacity, buffering capacity),[23,51,81] crevicular fluid.[32]

Nevertheless, recent reviews employ the traditional terms.

As a conclusion, the development of NCCL is supposed to be the consequence of a synergistic action of five etiological mechanisms (Table 1). In addition to the most known three theories, two other original causes have been described but somehow less investigated: the "stress corrosion theory" and the "piezoelectric effect theory".[10,32,52]

THE PREVENTIVE APPROACH

Several anthropologic studies have been undertaken to clarify the physiological extent of tooth wear in general. It is interesting to notice that the anthropologic point of view supports the pathologic nature of NCCL in particular, which have not been observed within ancient populations and therefore should be viewed as "modern-day" pathology.[55] Therefore, no level of the lesion should be considered acceptable and ignored, and preventive or restorative measures should always be implemented,[56] although with different protocols with respect to age, severity of the lesion, risk factors and etiological factors implicated.

The need of changing the approach regarding NCCL is especially important for incipient lesions, because the early detection of NCCL is the best indication for preventive therapy as an alternative to the restorative approach. Three aspects of preventive therapy should be considered:

1. Risk assessment of patients and prevention of development of NCCL lesions by correcting habits and eliminating possible causes;
2. Early detection and management of incipient lesions;
3. Management of patients already presenting advanced NCCL lesions.

The objectives of the preventive treatment are to prevent the progression of incipient lesions or the development of new ones and to assure the longevity of restorations in restored lesions, as early failures of these restorations have often been reported in the literature probably due to the same factors which originally caused the lesions.[3,20,39]

Although there is no consensus regarding the etiology, clinical evaluations of large number of subjects have revealed some factors more frequently associated with the occurrence of NCCL. They could serve for the risk assessment of the patient, by suggesting a higher probability of developing such lesions. Studies have reported the preponderant presence of parafunctional habits and bruxism,[20,24,29,44-47,57] or abrasive and erosive[18,23,58] factors as well as biological individual variations,[7,47,59] which predispose to the development of such lesions (Table 2). It is important to notice that the majority of these studies could not totally exclude one factor or another, pointing into the direction of a multifactorial etiology of NCCL.[15,18,24,45,47,60]

A frequent implication of occlusal factors has been reported,[20,44,57] even though their positive correlation with NCCL or the predictable value of occlusion could not always be established.[15,28,43] A finite element analysis study[39] identified loading direction as a major factor contributing to restoration failure, and showed that oblique-oriented forces induce tensile stresses on the cervical margin, exceeding the strength of the material and the adhesive forces. Several clinical trials found generators for oblique loading such as altered tooth position[26] and group function,[20,24,29,44] as well as parafunctional habits indicated by wear facets[13,19,20,23,24,44,48,60] and bruxism,[19,20,29,45] to be associated with NCCL. Nevertheless, it should be kept in mind that available tests revealed only an association of occlusal loading factors and noncarious cervical lesions, which may not necessarily support a causal relationship. Other types of clinical investigations, such as observational long-term studies, would be necessary to confirm the occlusal etiology theory.

Prevalence, severity and progression rate of NCCL were found to increase with age.[20,22,24-26,28,29] This could be explained by the extended exposure to etiological factors, the increased occurrence of gingival recession and bone loss with more root surface and cementum exposure raising the risk of cervical lesions, the diminished quantity and quality of saliva and the compositional and microstructural changes of enamel and dentin.[20]

THE PREVENTIVE PROTOCOL

Previous research in NCCL focused mainly on etiology and restoration options, with no clear guidelines to a preventive approach. This article proposes a preventive concept based on the hypothesis of a multifactorial etiology of such lesions. Given the confusing multitude of restorative options and the uncertain durability of their results, a global treatment strategy for NCCL is needed, taking into consideration also the etiology and long-term management of such lesions.

The available knowledge on NCCL today allows for the elaboration of a non-restorative, more conservative management, as an alternative to the restorative approach. The slower progression rate in young people,[61] the high capacity of self-

defense by producing sclerotic dentin, and the lack of evidence for tooth weakening in the absence of a restoration[62] could possibly support a "wait and see" philosophy. As suggested for the management of erosion, restoration could be postponed in the absence of esthetic demands, sensitivity or threat to the integrity of the tooth.[63] Restoration should not always be the first treatment choice; although there still remains to be established up to which extent prevention could replace restoration.

Early diagnosis may stop the progression of such lesions, if etiological factors are controlled, a close recall and monitoring is undertaken, and patients comply with the recommendations.

The risk assessment evaluates the risk of a patient to develop NCCL over time and it is a part of the general examination. Practitioners should be aware of the possible causes of such lesions and associated risk factors (Table 2) and search for signs of their presence in every patient to treat. This approach is especially important in young patients presenting premature signs of tooth wear not in accordance with their age and where etiological factors are present which could lead over time to the development of NCCL.

EARLY DETECTION AND TREATMENT OF INCIPIENT NCCL

In some cases where lesions are small and just start to develop, restoration is not the most appropriate strategy, due to the uncertain clinical longevity. In an *in vivo* study,[20] the correlation between age and depth of lesions led to the conclusion that NCCL progression is a slow process. An *in vitro* study[62] concluded that the presence of NCCL on extracted teeth does not negatively affect their fracture resistance, and that restoration does not result in an increase of fracture resistance, despite the belief of strengthening the remaining tooth structure by restoring the defect.[56] Thus, in small lesions, preventive measures together with a causal therapy and a close monitoring of the patient are the strategy of choice. The same is true for lesions which do not cause any esthetic or functional problems, lesions without sensitivity or which do not compromise the integrity of the tooth. As the clinical effectiveness of Class V restorations seems to be controversial, the elimination of the causes, instead, might be more beneficial in the long-term.

The preventive approach in patients already presenting advanced lesions is more complex. Beside the restorative therapy of the advanced lesions, elimination of causes should always be considered to assure stability and longevity. Local preventive measures like professional application of fluorides and educating the patient is crucial for preventing the development of new lesions.

The strategy in detail is as follows:

1. Elimination of local or general etiological factors.
2. Enhancement of resistance against acid attack.
3. Brushing with desensitizing and fluoride containing dentifrices, daily use of fluoride rinse, fluoride gels, soft toothbrush.
4. Professional application of a fluoride varnish, desensitizer (potassium oxalate, arginine-calcium carbonate or other tubule-occluding agents) or of an adhesive coating.
5. Close monitoring of the patient.
6. Elimination of possible causal factors by providing instructions for correct oral hygiene techniques, dietary counseling, treating of general disorders such as gastric reflux, bulimia, anorexia and correcting parafunctions and occlusal habits. The fabrication of an occlusal guard is a reasonable protective measure in case of high occlusal stress, but there is still a controversy about the need for occlusal equilibration on teeth with NCCL.[12] Occlusal adjustment is suggested by the frequent association of NCCL with functional stresses, especially with oblique forces leading to tensile stress at the cervical region.[10] Nevertheless, very few clinical investigations[64] exist to confirm the positive effect of occlusal therapy, and a recent review[12] reported the ineffectiveness of this measure in prolonging the longevity of cervical restorations. The uncertainty of occlusion as an etiological mechanism of NCCL or a predictability factor[28] constitutes also a contraindication for performing "preventive" invasive occlusal therapy.[43] This is substantiated by study results[45] showing that the presence of occlusal pathology does not always lead to the development of NCCL, even if a positive association may exist. It might be concluded that the presence of NCCL alone should not constitute a recommendation for indiscriminate occlusal adjustment and further clinical investigations are needed to confirm this hypothesis. Dietary counseling should address the uptake frequency of acidic containing foods and beverages, the ingestion habits, as well as the type of foods with a buffering capacity. Thus, the use of a straw for acidic beverages as well as drinking milk or eating a piece of cheese shortly afterwards should be encouraged in young people for whom lifestyle changes would be particularly difficult to achieve.[50]
7. Local chemical preventive measures (educating the patient for correct toothbrushing and regular applications of topical fluorides and professional application of fluoride products and/or adhesive coatings) for enhancing resistance of tooth structures to erosion and abrasion.
8. Regular monitoring of the patient. The recall interval should vary upon age, as the progression rate of erosive/abrasive lesions was found to be higher in older people.[51] It is known that the evolution of NCCL is generally a slow process, but no specific progression rates for NCCL were given in the literature. Therefore, an individual monitoring protocol has to be established, by assessing the severity of the present lesions, the age and the existing etiological and risk factors. For patients particularly exposed to intrinsic or extrinsic acids or presenting a rapid progression, the measurement procedure should be repeated at 6-month intervals, but for most other cases, annually is acceptable.[67] The progression can be assessed clinically by measuring the width and length of the lesion with a graded probe, and also by comparatively examining photographs.

RECOMMENDATIONS FOR PATIENTS PRESENTING NCCL

1. Use a soft toothbrush and low abrasion fluoridated toothpaste (around 1100 pm F) or a calcium containing toothpaste.[50,63,68,69] Avoid toothpaste and mouthrinse with low pH.[50] In case of softened enamel, power or sonic toothbrushes may lead to significantly higher loss of substance.[70]

2. Avoid toothbrushing immediately after an erosive challenge in order to preserve the salivary pellicle.[68,71-73] Brushing is recommended prior to rather than after the erosive challenge.[74-76] Instead, use a fluoride containing mouthrinse or an iron containing mouthrinse[77] after the erosive challenge.[50,68]
3. Gently apply concentrated topical fluoride without disturbing the protective pellicle of the tooth surface.[68]
4. Professional application of fluoride varnishes. Repeated application is necessary, due to temporary protection.[63,78]
5. Adhesive systems may protect dentin from erosion and abrasion for a limited period of time.[63,79,80]
6. Use of sugar-free chewing gum or non-acidic saliva stimulating products. Sugar free chewing gum and even fluoride containing or carbamide containing gum are advised in order to increase salivary flow, knowing that saliva is an important protective factor through the pellicle formation and the buffering capacity.[50,63,68,73,76,81] Stimulating salivary flow has been shown *in vitro* to reduce abrasion/erosion[82] and it might also be a treatment for patients with symptomatic reflux, by helping to reduce postprandial esophageal acid exposure.[68]
7. In case of hypersensitivity, use a toothpaste containing fluoride and desensitizers, and professional application of adhesive coating/desensitizers/fluoride varnish.

LOCAL PREVENTIVE MEASURES

The presumed erosive and abrasive nature of NCCL might support the introduction of local preventive measures. As a consequence of erosive challenges, tooth substance was reported to exhibit a change in microhardness and a higher susceptibility to substance loss by a subsequent abrasive challenge.[59] Therefore, *in vitro* and *in situ* evaluations have measured the effect of different products applied to the altered tooth surfaces. Fluoride products, calcium containing toothpastes, iron containing mouthrinses and adhesive coatings were tested *in vitro* and *in situ* for their protective effect against erosive-abrasive tooth substance loss.

FLUORIDES

The most efficient long-term strategy seems to be a daily repeated application of fluoride products, this being achieved mainly with products used at home.[68,83] Therefore, patient education and compliance becomes a very important part of the preventive strategy. Generally, toothpastes provide fluoride on a regular basis and *in vitro* studies report an anti erosive/ abrasive effectiveness.[74,83,84] Apart from fluoridated toothpaste and mouthrinse,[83] patients with NCCL should regularly apply fluoride gels, as their protective effect seems also to be greater.[85] *In vitro* and *in situ* studies suggest that a combination of different fluoride products used regularly by the patient may significantly reduce erosion.[83] Nevertheless, there are some discrepancies regarding the protective effectiveness against abrasion or attrition, with some studies showing no protection against the abrasive challenge or reporting even an increase of the amount of wear[86,87] probably due to imperfections of laboratory conditions or the highly acidic formula of the employed product.

A preventive approach aiming to reduce the contact of the tooth tissues with the erosive agents may pose some difficulties due to patient compliance. In case of erosion due to intrinsic factors, diseases are often difficult to control. Therefore, the use of professional preventive measures was suggested as part of the preventive strategy.[88,89] Beside the known caries protective properties, fluoride products have been proposed as a protective measure against erosion/abrasion. Fluoride has a dynamic implication in the remineralization-dissolution process around tooth surface, suggesting thereby a possible interference with the erosion mechanism, described as a dissolution and outflow of ions towards tooth surface.[59]

Professional applications are also necessary as high concentrations of fluorides are needed to achieve a good protective effect.[89] Concentrated gels and varnishes are the options. Beside the remineralizing effect of fluoride on the erosion lesions, the varnish is expected to provide mechanical protection of the tooth surface against acid diffusion and reduction of hypersensitivity.[78,87,91,92] As long as they remain attached to the tooth surface, fluoride varnishes may be more effective than solutions and gels in prevention of erosive defects due to their better capability to adhere to the tooth surface and create a calcium fluoride reservoir, although they present the shortcomings of short term effectiveness and therefore the need for repeated applications in multiple layers.[78] However, further clinical investigation is required to understand the role of fluoride in protecting mineralized tissues from such processes.[35,50,93]

ADHESIVE COATINGS

In vitro[79] and later *in vivo*[80] tests found dentin sealing with resin based adhesives to be an effective strategy against erosive/abrasive tooth wear as well. Compared to unsealed surfaces, coated dentin exhibited less substance loss after erosive and abrasive challenge.[94] Resin-based adhesives were shown to be more efficient against further substance loss in comparison to fluoride mouthrinses in an *in vitro* study.[94] Nevertheless, they need frequent reapplication due to their low wear resistance. Some tested products are Seal and Protect[a] [79,80,94] and Optibond Solo.[b,79,80] Seal and Protect, a self-adhesive, light-curing, translucent sealing material is designed to prevent the development and progression of wedge-shaped lesions by producing a hard coat increasing the resistance of cervical areas against abrasive and erosive forces. It is specifically designed to protect exposed dentin, similar to pit and fissure sealants.

OTHER PRODUCTS

Another type of topical preventive measures includes calcium and phosphate based remineralization systems like casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) nanocomplexes. Calcium-containing sodium bicarbonate based toothpastes, chewing gums and mouthrinses were tested and reported to be even more efficient than toothpastes containing only fluoride.[96-98] Most of the investigations found these products able to harden enamel surface by delivering minerals and to reduce surface roughness, recommending these products also for erosive/ abrasive tooth substance loss.[96,99,100-102] A recent review[103] however reported the need for more investigation on their long-term effects. Promising results were obtained rather by combining this remineralizing system with fluoride, as the association with fluoride in the same product was shown to be more effective in remineralizing enamel, than

either product alone.[102] The synergistic effect of CPP-ACP and fluoride may result from the formation of CPP-stabilized amorphous calcium fluoride phosphate resulting in the increased incorporation of fluoride ions into plaque, together with increased concentrations of bioavailable calcium and phosphate ions.[102]

Conclusions

The etiology of NCCL is currently considered to be rather multifactorial, as clinical investigations have found multiple factors associated with this type of lesions and due to the lack of evidence to support exclusively one or another factor. No clinical research exists with respect to the prevention of NCCL and long-term clinical evaluations of the proposed preventive measures are needed. The possible erosive or abrasive nature of the lesion and the difficult-to-control etiology may justify professional applications of topical fluorides (especially fluoride varnishes) or of adhesive systems of general use or specially conceived products (*e.g.* Seal and Protect) and the home use of different fluoride or calcium containing products. Their protective effect against erosion/abrasion could be confirmed in several laboratory and *in situ* studies, but the effectiveness and optimum application frequency still need to be established in clinical trials. Additionally, calcium- and phosphate-based remineralization systems obtained promising results, which however, also need further clinical confirmation.

Progression rate of NCCL, generally slow, represents important information which has to be measured in the future on an individual level, in order to establish an optimized recall interval for patients at risk of developing such lesions.

As restorative options for NCCL are still not satisfying regarding esthetics and longevity, prevention should take the leading role within the management strategy of NCCL. Early detection and management of incipient lesions become of primary importance to avoid premature non-carious destruction of teeth.

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References

1. Lussi A. Dental Erosion. *Monogr Oral Sci* 2006;20:9-16.
2. Bartlett DW, Shah P. A critical review of non-carious cervical (wear) lesions and the role of abfraction, erosion, and abrasion. *J Dent Res* 2006;85:306-312.
3. Ceruti P, Menicucci G, Mariani GD, Pittoni D, Gassino G. Non carious cervical lesions. A review. *Minerva Stomatol* 2006;55:43-57.
4. Litonjua LA, Andreana S, Patra AK, Cohen RE. An assessment of stress analyses in the theory of abfraction. *Biomed Mater Eng* 2004;14:311-321.
5. Litonjua LA, Andreana S, Bush PJ, Cohen RE. Tooth wear: Attrition, erosion, and abrasion. *Quintessence Int* 2003;34:435-446.
6. Litonjua LA, Andreana S, Bush PJ, Tobias TS, Cohen RE. Noncarious cervical lesions and abfractions: A re-evaluation. *J Am Dent Assoc* 2003;134:845-850.
7. Litonjua LA, Andreana S, Cohen RE. Toothbrush abrasions and noncarious cervical lesions: Evolving concepts. *Compend Contin Educ Dent* 2005; 26:767-768, 770-774, 776.
8. Lyons K. Aetiology of abfraction lesions. *N Z Dent J* 2001;97:93-98.
9. Rees JS. A review of the biomechanics of abfraction. *Eur J Prosthodont Restor Dent* 2000;8:139-144.
10. Rees JS, Jagger DC. Abfraction lesions: Myth or reality? *J Esthet Restor Dent* 2003;15:263-271.
11. Terry DA, McGuire MK, McLaren E, Fulton R, Swift EJ Jr. Perioesthetic approach to the diagnosis and treatment of carious and noncarious cervical lesions: Part I. *J Esthet Restor Dent* 2003;15:217-232.
12. Wood I, Jawad Z, Paisley C, Brunton P. Non-carious cervical tooth surface loss: A literature review. *J Dent* 2008;36:759-766.
13. Pegoraro LF, Scolaro JM, Conti PC, Telles D, Pegoraro TA. Noncarious cervical lesions in adults: Prevalence and occlusal aspects. *J Am Dent Assoc* 2005;136:1694-700. Erratum in: *J Am Dent Assoc* 2006;137:447.
14. Vandewalle KS, Vigil G. Guidelines for the restoration of Class V lesions. *Gen Dent* 1997;45:254-260.
15. Piotrowski BT, Gillette WB, Hancock EB. Examining the prevalence and characteristics of abfraction like cervical lesions in a population of U.S. veterans. *J Am Dent Assoc* 2001;132:1694-1701.
16. Lussi A: Dental Erosion. *Monogr Oral Sci* 2006;20:32-43.
17. Young A, Amaechi BT, Dugmore C, Holbrook P, Nunn J, Schiffner U, Lussi A, Ganss C. Current erosion indices. Flawed or valid? Summary. *Clin Oral Investig* 2008;12 (Suppl 1):S59-63.
18. Khan F, Young WG, Shahabi S, Daley TJ. Dental cervical lesions associated with occlusal erosion and attrition. *Aust Dent J* 1999;44:176-186.
19. Telles D, Pegoraro LF, Pereira JC. Incidence of noncarious cervical lesions and their relation to the presence of wear facets. *J Esthet Restor Dent* 2006;18:178-183.
20. Aw TC, Lepe X, Johnson GH, Mancl L. Characteristics of noncarious cervical lesions: A clinical investigation. *J Am Dent Assoc* 2002;133:725-733.
21. Lussi AR, Schaffner M, Hotz P, Suter P. Epidemiology and risk factors of wedge-shaped defects in a Swiss population. *Schweiz Monatsschr Zahnmed* 1993;103:276-280. (In German).
22. Borcic J, Anic I, Urek MM, Ferreri S. The prevalence of non-carious cervical lesions in permanent dentition. *J Oral Rehabil* 2004;31:117-123.
23. Faye B, Sarr M, Kane AW, Toure B, Leye F, Gaye F, Dieng MB. Prevalence and etiologic factors of non-carious cervical lesions. A study in a Senegalese population. *Odontostomatol Trop* 2005;28:15-18.
24. Smith WA, Marchan S, Rafeek RN. The prevalence and severity of non-carious cervical lesions in a group of patients attending a university hospital in Trinidad. *J Oral Rehabil* 2008;35:128-134.
25. Takehara J, Takano T, Akhter R, Morita M. Correlations of noncarious cervical lesions and occlusal factors determined by using pressure-detecting sheet. *J Dent* 2008;36:774-779.
26. Bernhardt O, Gesch D, Schwahn C, Mack F, Meyer G, John U, Kocher T. Epidemiological evaluation of the multifactorial aetiology of abfractions. *J Oral Rehabil* 2006;33:17-25.
27. Akgül HM, Akgül N, Karaoglanoglu S, Ozdabak N. A survey of the correspondence between abrasions and tooth brushing habits in Erzurum, Turkey. *Int Dent J* 2003;53:491-495.
28. Chan DC, Browning WD, Pohjola R, Hackman S, Myers ML. Predictors of non-carious loss of cervical tooth tissues. *Oper Dent* 2006;31:84-88.
29. Tomasik M. Analysis of etiological factors involved in noncarious cervical lesions. *Ann Acad Med Stetin* 2006;52:125-136.
30. Miller WD. Experiments and observations the wasting of tooth tissue variously. Designated as erosion, abrasion, chemical abrasion, denudation, etc. *Dent Cosmos* 1907;49:225-247.
31. Grippo JO. Abfractions: A new classification of hard tissue lesions of teeth. *J Esthet Dent* 1991;3:14-19.
32. Grippo JO, Simring M, Schreiner S. Attrition, abrasion, corrosion and abfraction revisited: A new perspective on tooth surface lesions. *J Am Dent Assoc* 2004;135:1109-1118.
33. Lee HE, Lin CL, Wang CH, Cheng CH, Chang CH. Stresses at the cervical lesion of maxillary premolar. A finite element investigation. *J Dent* 2002;30:283-290.
34. Rees JS. The role of cuspal flexure in the development of abfraction lesions: A finite element study. *Eur J Oral Sci* 1998;106:1028-1032.
35. Rees JS. The biomechanics of abfraction. *Proc Inst Mech Eng H* 2006;220:69-80.
36. Borcic J, Anic I, Smojver I, Catic A, Miletic I, Ribaric SP. 3D finite element model and cervical lesion formation in normal occlusion and in malocclusion. *J Oral Rehabil* 2005;32:504-510.
37. Borcić J, Antonić R, Urek MM, Petricević N, Nola-Fuchs P, Catić A, Smojver I. 3-D stress analysis in first maxillary premolar. *Coll Antropol* 2007;31:1025-1029.

38. Ichim I, Li Q, Loughran J, Swain MV, Kieser J. Restoration of non-carious cervical lesions Part I. Modelling of restorative fracture. *Dent Mater* 2007;23:1553-1561.
39. Ichim IP, Schmidlin PR, Li Q, Kieser JA, Swain MV. Restoration of non-carious cervical lesions. Part II. Restorative material selection to minimise fracture. *Dent Mater* 2007;23:1562-1569.
40. Ichim I, Schmidlin PR, Kieser JA, Swain MV. Mechanical evaluation of cervical glass-ionomer restorations: 3D finite element study. *J Dent* 2007;35:28-35.
41. Rees JS, Hammadeh M, Jagger DC. Abfraction lesion formation in maxillary incisors, canines and premolars: A finite element study. *Eur J Oral Sci* 2003;111:149-154.
42. Rees JS, Hammadeh M. Undermining of enamel as a mechanism of abfraction lesion formation: A finite element study. *Eur J Oral Sci* 2004;112:347-352.
43. Estafan A, Furnari PC, Goldstein G, Hittelman EL. *In vivo* correlation of noncarious cervical lesions and occlusal wear. *J Prosthet Dent* 2005;93:221-226.
44. Miller N, Penaud J, Ambrosini P, Bisson-Boutelliez C, Briaçon S. Analysis of etiologic factors and periodontal conditions involved with 309 abfractions. *J Clin Periodontol* 2003;30: 828–832
45. Ommerborn MA, Schneider C, Giraki M, Schafer R, Singh P, Franz M, Raab WH. *In vivo* evaluation of noncarious cervical lesions in sleep bruxism subjects. *J Prosthet Dent* 2007;98:150-158.
46. Pintado MR, Delong R, Ko CC, Sakaguchi RL, Douglas WH. Correlation of noncarious cervical lesion size and occlusal wear in a single adult over a 14-year time span. *J Prosthet Dent* 2000;84:436-443.
47. Tsiggos N, Tortopidis D, Hatzikyriakos A, Menexes G. Association between self-reported bruxism activity and occurrence of dental attrition, abfraction, and occlusal pits on natural teeth. *J Prosthet Dent* 2008; 100:41-46.
48. Yang WL, Lin XF, Zou B, Li XX. Investigation on relationship between wedge-shaped defects and occlusal interference *Hua Xi Kou Qiang Yi Xue Za Zhi* 2007;25:383-385. (In Chinese).
49. De Las Casas EB, Cornacchia TP, Gouvêa PH, Cimini CA Jr. Abfraction and anisotropy. Effects of prism orientation on stress distribution. *Comput Methods Biomech Biomed Engin* 2003;6:65-73.
50. Holbrook WP, Arnadóttir IB, Kay EJ. Prevention. Part 3: Prevention of tooth wear. *Br Dent J* 2003;195:75-81.
51. Lussi A, Jaeggi T. Erosion. Diagnosis and risk factors. Review. *Clin Oral Investig* 2008;12 Suppl 1:S5-13.
52. Grippo JO, Masi JV Role of biodental engineering factors (BEF) in the etiology of root caries. *J Esthet Dent* 1991;3:71-76.
53. Kaidonis JA. Tooth wear: The view of the anthropologist. *Clin Oral Investig* 2008;12 Suppl 1:S21-26.
54. Aubry M, Mafart B, Donat B, Brau JJ. Brief communication: Study of noncarious cervical tooth lesions in samples of prehistoric, historic, and modern populations from the South of France. *Am J Phys Anthropol* 2003;121:10-14.
55. Ritter AV, Grippo JO, Coleman TA, Morgan ME. Prevalence of carious and non-carious cervical lesions in archaeological populations from North America and Europe. *Esthet Restor Dent* 2009;21:324-334.
56. Grippo JO. Noncarious cervical lesions: The decision to ignore or restore. *J Esthet Dent* 1992;4 Suppl:55-64.
57. Mayhew RB, Jessee SA, Martin RE. Association of occlusal, periodontal, and dietary factors with the presence of non-carious cervical dental lesions. *Am J Dent* 1998;11:29-32.
58. Faye B, Kane AW, Sarr M. Noncarious cervical lesions among a non-toothbrushing population with Hansen's disease (leprosy): Initial findings. *Quintessence Int* 2006;37:613-619.
59. Lussi A. Dental erosion. *Monogr Oral Sci* 2006;20:1-8.
60. Oginni AO, Olusile AO, Udoye CI. Non-carious cervical lesions in a Nigerian population: abrasion or abfraction? *Int Dent J* 2003;53:275-279.
61. Lussi A, Schaffner M. Progression of and risk factors for dental erosion and wedge-shaped defects over a 6-year period. *Caries Res* 2000;34:182-187.
62. Osborne-Smith KL, Burke FJ, Farlane TM, Wilson NH. Effect of restored and unrestored non-carious cervical lesions on the fracture resistance of previously restored maxillary premolar teeth. *J Dent* 1998;26:427-433.
63. Milosevic A, O'Sullivan E, Royal College of Surgeons of England. Diagnosis, prevention and management of dental erosion: Summary of an updated national guideline. *Prim Dent Care* 2008;15:11-12.
64. Coleman TA, Grippo JO, Kinderknecht KE. Cervical dentin hypersensitivity. Part III: Resolution following occlusal equilibration. *Quintessence Int* 2003;34:427-434.
65. Wood ID, Kassir AS, Brunton PA. Effect of lateral excursive movements on the progression of abfraction lesions. *Oper Dent* 2009;34:273-279.
66. Michael JA, Townsend GC, Greenwood LF, Kaidonis JA. Abfraction: Separating fact from fiction. *Aust Dent J* 2009;54:2-8.
67. 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-S68.
68. Lussi A. Dental erosion. *Monogr.Oral.Sci* 2006;20:190-199.
69. Lynch RJ, ten Cate JM. The anti-caries efficacy of calcium carbonate-based fluoride toothpastes. *Int Dent J* 2005;55(3 Suppl 1):175-178.
70. Wiegand A, Begic M, Attin T. *In vitro* evaluation of abrasion of eroded enamel by different manual, power and sonic toothbrushes. *Caries Res* 2006;40:60-65.
71. Wiegand A, Meier W, Sutter E, Magalhães AC, Becker K, Roos M, Attin T. Protective effect of different tetrafluorides on erosion of pellicle-free and pellicle-covered enamel and dentine. *Caries Res* 2008;42:247-254.
72. Wiegand A, Bliggenstorfer S, Magalhaes AC, Sener B, Attin T. Impact of the *in situ* formed salivary pellicle on enamel and dentine erosion induced by different acids. *Acta Odontol Scand* 2008;66:225-230.
73. Hove LH, Holme B, Young A, Tveit AB. The erosion-inhibiting effect of TiF^4, SnF^2, and NaF solutions on pellicle-covered enamel *in vitro*. *Acta Odontol Scand* 2007;65:259-264.
74. Lussi A, Megert B, Eggenberger D, Jaeggi T. Impact of different toothpastes on the prevention of erosion. *Caries Res* 2008;42:62-67.
75. Wiegand A, Buchalla W, Attin T. Review on fluoride-releasing restorative materials. Fluoride release and uptake characteristics, antibacterial activity and influence on caries formation. *Dent Mater* 2007;23:343-362.
76. 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;21:13-16.
77. Sales-Peres SH, Pessan JP, Buzalaf MA. Effect of an iron mouthrinse on enamel and dentine erosion subjected or not to abrasion: An *in situ*/*ex vivo* study. *Arch Oral Biol* 2007;52:128-132.
78. Vieira A, Jager DH, Ruben JL, Huysmans MC. Inhibition of erosive wear by fluoride varnish. *Caries Res* 2007;41:61–67.
79. 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;196:351-354.
80. Azzopardi A, Bartlett DW, Watson TF, Sherriff M. The measurement and prevention of erosion and abrasion. *J Dent* 2001;29:395-400.
81. Wetton S, Hughes J, Newcombe RG, Addy M. The effect of saliva derived from different individuals on the erosion of enamel and dentine. A study *in vitro*. *Caries Res* 2007;41:423-426.
82. Rios D, Honório HM, Magalhães AC, Delbem AC, Machado MA, Silva SM, Buzalaf MA. 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;40:218-223.
83. 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;38:561-566.
84. 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;41:146-151.
85. 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;40:148-153.
86. Li H, Watson TF, Sherriff M, Curtis R, Bartlett DW. The influence of fluoride varnish on the attrition of dentine. *Caries Res* 2007;41:219-222.
87. Magalhães AC, Stancari FH, Rios D, Buzalaf MA. Effect of an experimental 4% titanium tetrafluoride varnish on dental erosion by a soft drink. *J Dent* 2007;35:858-861.
88. Imfeld T. Prevention of progression of dental erosion by professional and individual prophylactic measures. *Eur J Oral Sci* 1996;104:215-220.
89. Zero DT, Lussi A. Erosion. Chemical and biological factors of importance to the dental practitioner. *Int Dent J* 2005;55 (4 Suppl 1):285-290.
90. 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;42:269-274.
91. Ritter AV, de L Dias W, Miguez P, Caplan DJ, Swift EJ Jr. Treating cervical dentin hypersensitivity with fluoride varnish: A randomized clinical study. *J Am Dent Assoc* 2006;137:1013-1020.
92. Vieira A, Ruben JL, Huysmans MC. Effect of titanium tetrafluoride, amine fluoride and fluoride varnish on enamel erosion *in vitro*. *Caries Res* 2005;39:371-379.
93. Barbour ME, Rees GD. The role of erosion, abrasion and attrition in tooth wear. *J Clin Dent* 2006;17:88-93.
94. Sundaram G, Wilson R, Watson TF, Bartlett DW. Effect of resin coating on dentine compared to repeated topical applications of fluoride mouthwash after an abrasion and erosion wear regime. *J Dent* 2007;35:814-818.

95. Sundaram G, Wilson R, Watson TF, Bartlett D. Clinical measurement of palatal tooth wear following coating by a resin sealing system. *Oper Dent* 2007;32:539-543.
96. Charig A, Winston A, Flickinger M. Enamel mineralization by calcium-containing-bicarbonate toothpastes: Assessment by various techniques. *Compend Contin Educ Dent* 2004;25(9 Suppl 1):14-24.
97. Kardos S, Shi B, Sipos T. The *in vitro* demineralization potential of a sodium fluoride, calcium and phosphate ion-containing dentifrice under various experimental conditions. *J Clin Dent* 1999;10(1 Spec No):22-25.
98. Litkowski LJ, Quinlan KB, Ross DR, Ghassemi A, Winston A, Charig A, Flickinger M, Vorwerk L. Intraoral evaluation of mineralization of cosmetic defects by a toothpaste containing calcium, fluoride, and sodium bicarbonate. *Compend Contin Educ Dent* 2004;25 (9 Suppl 1):25-31.
99. Cai F, Shen P, Morgan MV, Reynolds EC. Remineralization of enamel subsurface lesions *in situ* by sugar-free lozenges containing casein phosphopeptide-amorphous calcium phosphate. *Aust Dent J* 2003;48:240-243.
100. Cross KJ, Huq NL, Reynolds EC.Casein phosphopeptides in oral health--chemistry and clinical applications. *Curr Pharm Des* 2007;13:793-800.
101. Iijima Y, Cai F, Shen P, Walker G, Reynolds C, Reynolds EC. Acid resistance of enamel subsurface lesions remineralized by a sugar-free chewing gum containing casein phosphopeptide-amorphous calcium phosphate. *Caries Res* 2004;38:551-556.
102. 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;82:206-211.
103. Azarpazhooh A, Limeback H. Clinical efficacy of casein derivatives: A systematic review of the literature. *J Am Dent Assoc* 2008;139:915-924.
104. Reynolds EC, Cai F, Cochrane NJ, Shen P, Walker GD, Morgan MV, Reynolds C. Fluoride and casein phosphopeptide-amorphous calcium phosphate. *J Dent Res* 2008;87:344-348.
105. Young WG. The oral medicine of tooth wear. *Aust Dent J* 2001;46:236-250.