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INFLUENCE OF ACID DIET AS A BIOCORROSIVE FACTOR IN NON-CARIOUS CERVICAL LESIONS - LITERATURE REVIEW

[1,2]Taylane Berlanga de Araújo Soffener, [2]Stella Nogueira Benedetti, [2]Walquiria Silva Vilela, [2]Juliana dos Reis Dercelli, [2]Vera Lucia Bernardes, [1]Idiberto José Zotarelli Filho and [2]Carolina Almeida Rodrigues

[1]Post Graduate and Continuing Education (Unipos), Street Ipiranga, 3460, São José do Rio Preto SP, Brazil
[2]University Center of the Educational Foundation of Barretos, SP, Brazil- UNIFEB

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ABSTRACT

Introduction: Cervical tooth pathologies, in addition to caries, can be found in the form of carious and non-carious cervical lesions, the occurrence of which generates irreversible loss of dental structure. Non-carious cervical lesions (NCCL) involve the superficial pathological loss of dental tissue by a non-bacterial process different from that of caries. A contributory factor for the appearance of NCCLs that have been widely discussed is the acidic concentration of the diet, which may be justified by the changing eating habits of the general population, resulting in higher levels of acidic foods and beverages.
Objective: to review the literature regarding the onset and progression of non-carious cervical lesions in order to better understand the biocorrosive process of dental structure versus the ingestion of foods and acidic beverages.
Methods: This study consists of a narrative review of the literature where a research was carried out in the databases PUBMed, Scielo and CAPES Periodicals of articles involving subjects related to Non-Carious Cervical Lesions, regarding its definition, etiology and clinical aspects without restriction of year and language.
Conclusion: The increase in the prevalence of NCCL is directly related not only to toothbrushing or abrasion by dentifrice and occlusal trauma, but also to a strong influence of biocorrosive factors such as acidic diet, occupational factors and habits. Therefore, it is of paramount importance to the clinician to distinguish the etiological factors, the characteristics that differentiate them, and their consequences, so that there is a control of the risk factors added to the clarification and awareness of the patient, propitiating to correctly target the appropriate therapy reestablishing function and aesthetics of lasting form.

Corresponding author

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INTRODUCTION

Cervical pathologies of the tooth, in addition to caries, can be found in the form of carious and non-carious cervical lesions, the occurrence of which generates irreversible loss of dental structure. Non-carious cervical lesions (NCCL) involve the superficial pathological loss of dental tissue by a non-bacterial process other than caries (Eccles, 1982; Kliemann, 2002) in the amelo-cement junction region (Mair, 1992).

Frequently, these lesions result in cosmetic problems, dentin hypersensitivity, functional impairment, patient annoyance, need for endodontic treatment and even in dental fractures (Grippo, Chaiyabutir, Kois, 2013). Technological advances in the area of dentistry and the encouragement of public health prevention and awareness policies and methods have enabled the natural dentition to be maintained until advanced ages, exposing natural teeth for a longer time to contributing factors for the onset of NCCL (Marochi and Queiroz, 2001). Thus, it is extremely important to know the etiological agents in order

to contribute to the treatment of lesions already installed and future lesions (Barbosa *et al*., 2009). The clinical aspects of these lesions depend on the type and severity of the etiological factors involved (Bartlette Shah, 2006). The biocorrosion, abrasion and abduction (resulting from flexural stress) are factors involved in the process of removing the thin layer of enamel or the disintegration of the cervical enamel, which may expose dentin or even dental pulp (Lee and Eakle, 1996; Abrahamsen, 2005); Dzakovich, Oslak, 2008). The interaction of these factors is complex, however, it is the justification of some individuals to exhibit more than one type of cervical wear (Nascimento *et al*., 2016). A contributory factor for the emergence of NCCLs that have been widely discussed is the acidic concentration of the diet, which can be justified by the changing eating habits of the general population, resulting in higher levels of foods and beverages with acidic potential (Lussi , Jaeggi, Schaffner, 2002, Mahoney, Kilpatrick, 2003, Bartlett, 2009). In addition, the literature shows that the combined effects of pancreatic trypsin and pepsin on the stomach lead to proteolytic behavior, also interfering with the appearance and progression of these lesions (Schlueter *et al*., 2010). In view of the increased prevalence of NCCL in dental care settings, this study was carried out to understand the etiological mechanism of these lesions, to understand the role of the biocorrosive factor, especially when related to the frequent intake of potentially acidic foods and beverages, on the appearance and progression of these alterations. The objective of this study was to review the literature regarding the onset and progression of non-carious cervical lesions to better understand the biocorrosive process of the dental structure versus the intake of foods and acidic beverages.

MATERIALS AND METHODS

This study consists of a narrative review of the literature where a research was carried out in the databases PUBMed, Scielo and CAPES Periodicals of articles involving subjects related to Non-Carious Cervical Lesions, regarding its definition, etiology and clinical aspects, without year restriction and language. The keywords used were: Non-Carious Cervical Lesions, Dentinic Hypersensitivity, Abfraction, Abrasion, Erosion, Biocorrosion, Acid Diet, Soft Drinks, Juices, Acidic Drinks, Acidic Foods; using the words, their abbreviations and relating them to each other. The articles selected were systematic reviews of the literature or clinical research involving human subjects. Two authors were responsible for the selection of abstracts and when there was no scientific relevance, the articles were discarded. The articles with relevance selected for study were analyzed in relation to the characteristics of the methodology, results found, way to which the authors discuss the theme and conclusion. Presenting relevance and methodological agreement, these articles were discussed by the same examiners in order to reach a conclusion that would meet the required levels of scientific evidence.

Literature Review

NCCL is a result of wear in the cervical region of the tooth, which is non-bacterial (Eccles, 1982; Kliemann, 2002), and is currently one of the major problems encountered in dental practices due to aesthetic problems, dentin hypersensitivity, functional impairment, sometimes requiring endodontic treatment and even dental fractures due to irreversible loss of tooth structure (Grippo, Chaiyabutir, Kois, 2013). The etiology of non-carious cervical lesions for a long time was attributed only to brushing or abrasion by the dentifrice. Some authors argue that these lesions are caused by an interaction between three main factors: erosion, abfraction and abrasion (Levitch *et al*., 1994). The latest Consensus published by the European Federation of Conservative Dentistry emphasizes that teeth are exposed to a series of physical and chemical insults throughout life, which in various circumstances contributes to the wear and tear of hard tooth tissues. In this Consensus, it refers to the term erosion, which presents the acid as the main cause, resulting in wear on the occlusal surface, which may lead to the disappearance of occlusal morphology, and on smooth surfaces wear is found along the gingival margin and can progress to the concavities (Carvalho *et al*., 2015).

The term erosion for some time is no longer accepted to describe the wear process of the dental structure generated by contact with acid medium, because it comes from the wear generated between the friction and movement of acids against the dental surface. Thus, the term biocorrosion encompasses the wear caused by chemical and biochemical phenomena, whether exogenous or endogenous, by the proteolytic process of some enzymes or even by the direct effect of the piezoelectric effects directly on the organic matrix of the dentin (composed mainly of collagen), representing (Grippo, Chaiyabutr, Kois, 2013), which is the most suitable mechanism for the wear and tear of acidic substances (Grippo, 1991; Dawes, 2003; Clinically the lesions from the biocorrosion are in the form of saucers or "U", shallow, smooth and polished, with defined edges, free of plaque and with little brightness, differing from the abrasion and abfraction lesions that present characteristics since the abrasion is evidenced in the cervical vestibular third of teeth with gingival recession, especially the canines and premolars, with a hard, polished, shallow, regular and V-shaped contour. The abfractions are wedge-shaped, generally deep and with defined margin (Hoeppner, Massarollo, Bremm, 2007). Among the extrinsic causative factors are: diet (fruits, acid drinks), environment (chemical industries, chlorinated pools) and medicines (vitamin C, aspirin, hydrochloric acid). Already, the intrinsic factors are: diseases that provoke regurgitation of the gastric juice or decrease of the salivary flow. Considering that, in relation to the etiology of biocorrosion, the acids have received the greatest attention, and the acids present in the diet are the primary concern (Soares and Grippo, 2017).

Ancient studies have shown that biocorrosion lesions due to fruit and citrus juices are more frequent in the cervical third of the anterior teeth, although there is a possibility of occurring in any region of the dental element. (McIntyre, 1992). However, the intrinsic cause is associated with gastric acids, which are present in the oral cavity after vomiting, regurgitation and gastroesophageal reflux (Dodd *et al*., 1997; Rytomaa *et al*., 1998). After exposure of the root dentin, wear is easily caused by biocorrosion and / or abrasion. (Addy, Shellis, 2006; Lussi *et al*., 2012). Many experts believe that during the last decade there has been a significant increase in the prevalence and severity of tooth decay, the ingestion of soft drinks and other

acidic beverages is an extremely important factor related to dental biocorrosion followed by dentin hypersensitivity. Even when biocorrosive wear occurs in its milder forms, it is disturbing as it can compromise the integrity of a healthy dentition in later life. (Carvalho *et al*., 2006). Frequent consumption of acidic beverages and foods can lead to extrinsic biocorrosive lesions, which have been shown to be increasingly prevalent in children (Johansson *et al*., 2001) and young adults with a high intake of acidic beverages. As the biocorrosive potential of acidic beverages is determined by various chemical factors including pH, buffer capacity, chelation abilities and mineral content, it seems evident that specific modifications of these chemical factors may reduce their biocorrosive potential (Johansson *et al*., 1997).

Many studies have reported that acids present in foods and soft drinks (fruit juices, sports and energy drinks) represent a major responsible etiological factor for erosive lesions of dental enamel (Benjaukul *et al*., 2011; Gonçalves *et al*., 2012; Lussi *et al*., 2012). In Australia, there was the erosive potential of 15 beverages, including sugar-containing soft drinks, zero sugar soft drinks, diet soft drinks, sports drinks like Gatorade, milk-based drinks and orange juice sold for consumption in schools. Weight loss, surface loss and release of calcium ions from human enamel were evaluated after 30 minutes or 24 hours. All tested beverages, except milk and bottled water, produced significant in vitro tooth erosion (Benjaukul *et al*., 2011). Wongkhantee *et al*. (2006) carried out a study to determine the effect of acidic foods and beverages (cola-type refrigerant, yogurt, orange juice, sports drink, Tom-yum soup) on surface hardness of various substrates (enamel, dentin, and different restorative materials). The glue-type coolant significantly reduced the surface hardness of enamel, dentin, microparticulate composite and resin-modified glass ionomer ($p < 0.05$). Orange juice and sports drink significantly reduced enamel surface hardness ($p < 0.05$). Yogurt and Tom-yum soup did not reduce the surface hardness of any substrate. The authors were able to conclude that several foods and beverages have erosive potential and the public should be informed about them. A similar study, conducted in Brazil, evaluated the effect of processed foods and beverages on deciduous dental enamel previously eroded with hydrochloric acid (HCl). 102 specimens were submitted to an erosive challenge with HCl and randomly divided into six groups (n = 17): chocolate (Toddynho® - Pepsico), Petit Suisse Yogurt (Danoninho® - Danone); Strawberry Yogurt (Vigor); Apple puree (Nestlé); Fermented Milk (Yakult® - Yakult); and orange juice (del Valle). The 28 day immersion cycles for the test products were performed twice daily and were interspersed with exposure of the test substrate to the artificial saliva. The enamel microhardness evaluation showed a significant effect on the interaction between food and beverages and erosive potential ($p < 0.00001$). The orange juice resulted in greater enamel mineral loss after 28 days and none of the evaluated products could be associated to the recovery of the tooth enamel microhardness (Mesquita-Guimarães, 2015).

Barac *et al*., (2015) evaluated the erosive potential of various beverages by measuring the initial pH and titratable acidity (AT), the roughness of the enamel surface at different exposure times, comparing coke, orange juice, cedevita, guarana and strawberry yogurt. Samples from each group were exposed to 50mL of the beverage for 15, 30 and 60 min, 3 times a day for 10 days. The pH values of the refrigerants ranged from 2.52 (Guarana) to 4.21 (strawberry yogurt). Orange juice had the highest TA, requiring 5.70 ml of NaOH to reach pH 7.0, while Coke required only 1.87 ml. The roughness parameters indicated that Coca-Cola showed the strongest erosion potential during the 15 minutes of exposure, while Coca-Cola and orange juice were similar during 30 and 60 min exposures. There were no significant differences related to all exposure times between Guaraná and Cedevita. Strawberry yogurt did not corrode the surface of the enamel, regardless of the exposure time. Another study, 108 bovine enamel specimens evaluated for the purpose of determining the erosive potential of some soft drinks (coke and sprite) and orange juice. The erosive challenge was accomplished by completely immersing the specimen in the solution for 10 minutes at 20 ° C. Erosion generated on the enamel surface was measured using a profilometer, which showed higher enamel loss for Coca-Cola (5.60 ± 1.04 μm), followed by Sprite (5.49 ± 0.94 μm) and lastly the orange juice (1.35 ± 0.4 μm) (Aykut-Yetkiner *et al*., 2014).

Although soft drinks are made up mainly of water, artificial additives and refined sugar, thus offering quite limited nutritional benefit, they still increase energy. Sports drinks, designed to replenish fluids lost during activity, usually contain water, electrolytes, and sugar. The drinks are basically soft drinks containing vitamins and other chemicals that increase energy for a longer period of time (Lussi *et al*., 2012). In addition, differences in lifestyle, behavior and habits during consumption of acidic foods and beverages are often considered as potential erosion factors. Be it eating, drinking or swallowing, certain habits should be avoided, such as keeping a sour drink in your mouth for a while before swallowing, drinking, or sucking on your teeth. These attitudes increase the contact time of an acidic substance with the teeth and thus, the risk of biocorrosion (Torres *et al*., 2010). According to Jager *et al*. (2012), exposure times between 3 and 30 min result in very different estimates of potential biocorrosives. Today the media values "healthy" and "leaner" lifestyles, involving regular exercise and a balanced diet. However, many sportsmen and athletes usually drink beverages that aid in physical exercise or even during a balanced diet, most often acidic and rich in electrolytes and replacement fluids, increasing the chance of developing NCCL from the bioreactor mechanism. In addition, loss of body fluids and decreased salivary flow during exercise may increase the risk for dentition when ingestion of low pH liquids (Young, 1995; Zero, 1996). Thus, Aa dental biocorrosion in athletes is a growing concern. The frequent consumption of sports drinks and mineral water were implicated in the biocorrosion of teeth. In 1997, the association between biocorrosive wear and sports drink consumption was studied in 25 swimmers and 20 cyclists. The questionnaire identified the type of sports drink consumed and consumption patterns. The pH, acidity and concentrations of calcium, phosphate and fluoride, and sports viscosity were analyzed. Cyclists consumed isotonic drinks more frequently than swimmers ($p < 0.05$) and showed greater wear on the palatine surface of the upper teeth ($p < 0.001$), but there was no association between these factors and biocorrosion, even considering that most sports drinks have critical pH levels of 5.5 for enamel demineralization and

therefore have the potential to be biocorrosive (Milosevic, 1997).

The study of Sirimaharaj, Messer and Morgan (2002) evaluated the relationship between food consumption and acid beverages among groups of athletes through a questionnaire regarding healthy habits, diet and oral health. Agglutination losses were found in 25.4% of the evaluated sample, and the consumption of acidic foods and beverages was frequent for most athletes, but no significant correlation was found between dental erosion and frequency of isotonic intake. Correlation was found only between the frequency of juice intake ($p = 0.05$). De Melo *et al*. (2016) evaluated the impact of cabonated beverages on the potential dissolution of enamel and the influence of saliva as biological protection. The pH, AT, and buffer capacity of several beverages ingested by patients under physical activity were analyzed. Natural and industrialized coconut water served as a control solution for the study. High dissolution capacity of the enamel surface was found for all sports drinks evaluated and exposure to human saliva did not significantly promote the protective effect for acidic beverage attack. Thus, the authors concluded that carbohydrate and electrolyte beverages commonly consumed during resistance training may have a greater ability to dissolve the surface of the enamel, depending on its physicochemical properties associated with pH and titratable acidity. A systematic review of the literature was performed through the selection of cross-sectional and longitudinal studies evaluating tooth erosion and diet, performed in children and adolescents aged 8 to 19 years. In this review, the effect of eating habits on the occurrence of dental erosion was calculated using a fixed and random model (95%) and in total, 13 studies were used, totaling 16,661 children / adolescents evaluated. Data on dietary habits were obtained mainly from short dietary evaluations (69.2%) with food quantity (heavy or estimated) and food frequency questionnaires, used less frequently (15.4%). Meta-analyzes were performed for carbonated drinks or soft drinks, sports drinks, milk-based drinks, yogurt, confectionery and snacks and natural fruit acid drinks. Increased consumption of carbonated beverages, acid / sweet snacks and acid fruit juices increased the odds of tooth erosion, while increased intake of milk and yogurt reduced the chances of erosion.

Thus, evidence indicated that some eating habits (soft drinks, acid / sweet snacks and acid fruit juices) increased the likelihood of erosion, while milk or yogurt produced a protective effect. Methodological issues have partly explained the heterogeneity of the data for some dietary products (Richards, 2016). Many diets are adept at using teas, or even people consume them by habit; however, fruit teas also have an erosive effect on the enamel, but the effects on dentin were unknown until the work of Rees *et al*. ., (2006) where the erosive potential of a variety of fruit teas was evaluated in the laboratory by measuring pH and titratable acidity. The dentin specimens were prepared from extracted teeth and the ability of each tea to remove the smear layer was assessed by measuring the diameter and area of the open tubules and counting the number of tubules seen in one area of the unit using electron microscopy scanning. A 0.2% citric acid solution was used as a positive control. The pH of the fruit teas varied from 2.98 to 3.95 and the acidity ranged from 10.63 to 33.0 ml of 0.1 M NaOH. All the fruit teas tested were able to remove the smear layer. The mean diameter of the tubules ranged from 0.61 to 1.14 μm and the mean area varied from 0.31 to 1.03 μm2. The number of patent tubules per sample ranged from 13 to 121, showing that all the fruit teas tested were highly acidic and capable of removing the smear layer.
The consumption of energy drinks is also worrying, and they are growing among young people and adults. Studies evaluating the influence of energy drinks on the removal of the smear layer and the exposure of dentinal tubules on the root surface through 35 evaluated specimens showed that the use of energetic consumed in the United States as Flying Horse and Bug, even without association of friction , removed the superficial layer of smear layer from the specimens. When associated with friction, the energetics that resulted in erosion were Brun, Flying Horse, Gadiator and Sport Drinks. Thus, the authors concluded that energy drinks may be an important etiological factor for cervical dentin hypersensitivity (Pinto *et al*., 2013). Another concern is about occupational problems, as for example with sommeliers, winemakers and oenophiles. A study conducted in Queensland, Australia made to assess the bio-corrosive potential of wine in professional wine tasters and the effects that frequent exposure to dentition causes to enamel and dentin. There is a hidden occupational hazard that many professional wine tasters face around the world. The acidity of the wine in the permanent dentition can be quite detrimental with an increased risk of biocorrosion of dental hard tissue (Cheung *et al*., 2005). In Australia, one person on average drinks 23.19 liters of wine in a year (The Wine Institute, 2010). Wine is often consumed in Australia as a way of life, being closely associated with both business and leisure. The critical point at which the enamel dissolves is reported to be between 5.0 and 5.7 pH. The acidity of the wine (pH 3.0 to 3.8) indicates that long-term exposure of teeth may result in severe dental bio-corrosion (Mandel, 2005). Lussi and Jaeggi (2006) reported that full-time wine tasters sample on average 20-50 different wines, working almost five days a week. The tasters are very susceptible to the negative effects of wine on oral health (Mandel, 2005). During wine tasting, wine is taken, rotated, or moved around the mouth for about 30-60 seconds, increasing the risk of enamel and dentin biocorrosion (Mok *et al*., 2001). Mandel reported that wine tasters are very susceptible to acid biocorrosion due to the frequency and time the wine is exposed to the enamel surface (Mandel, 2005). The effects on oral health of a typical adult suffering from long-term dental biocorrosion include blemishes, lesions from demineralization by biocorrosion, dentin sensitivity and exposure of dentin or in more severe cases may lead to irreversible pulpitis and necrosis of the pulp (Wiegand, 2007, Mandel 2005, Lussi, 2006, Lussi *et al*., 2006). A trial simulating 10 episodes of wine erosion for 1 minute showed significant effects on erosion depth ($p < 0.001$) and surface roughness ($p < 0.001$), showing that enamel demineralization occurs at the initial stage of contact with wine , showing the need for clarification of the layman and prevention measures for professionals working in the tasting area (Kwek *et al*., 2015).

DISCUSSION

In 1994 Levitch described that the etiology of non-carious cervical lesions was long attributed only to brushing or abrasion by the dentifrice, but together with some authors it

was stated that these lesions are caused by the interaction between three main factors: biocorrosion, abfraction and abrasion. (Levitch *et al*., 1994). As demonstrated in this review and published by the latest consensus by the European Federation of Conservative Dentistry emphasized that in addition to the injuries caused by brushing or abrasion by the toothpaste, teeth are exposed to a series of physical and chemical insults throughout life, in that consensus refers the term biocorrosion tries the acid as the main cause, resulting in the appearance of these lesions (Carvalho *et al*., 2015). Biocorrosion is a significant problem in oral health today, and the literature is full of studies showing several foods and beverages that have increased consumption over the years, leading to increased concern about loss of dental tissue and often followed of dentin hypersensitivity. The increase in consumption of foods / foods with erosive potential may be associated with simple eating habits, lifestyle and occupation of the individual. Currently, with the great influence of healthier lifestyles, concern has been to the new habits with the inclusion of beverages and foods with strong erosive potential in their diets. Many diets are adept at using teas, drinks with natural acidic juices like water with lemon that is ingested with great frequency throughout the day. Thus, evidence indicates that these eating habits such as acid fruit juices increased the likelihood of biocor- sion occurrence (Richards, 2016). According to Lussi *et al*. (2011) acid diet is the most important cause of dental biocorrosion due to the high consumption of individuals on acidic foods and beverages. More and more children have included in their food practical snacks and are well accepted as yogurt, fermented milk, juices and chocolate milk, however the literature shows conflicting results with certain foods, such as yogurt. While some authors claim that yogurt despite its low pH has no erosive effect (Lussi *et al*., 2011; Wongkhantee *et al*., 2006). and the properties that affect tooth enamel demineralization (pH, buffer capacity, fluoride, calcium and phosphorus content) also showed that in addition to yogurt, other fermented beverages that are rich in lactobacilli did not promote erosion but rather a mineral loss of the enamel around 0.15 μm to 0.18 μm after four cycles of demineralization and remineralization. (Lodi *et al*., 2010).

The occupation of the individual also generates concern, as in the case of wine tasters (sommeliers, winemakers and enophiles) also present a high risk of developing NCCL from bio-corrosion as an aggravating factor, as demonstrated by Cheung *et al*. (2005) who reported that the acidity of the wine in the permanent dentition can be quite detrimental with an increased risk of biocorrosion of the dental hard tissue. The acidity of the wine (pH 3.0 to 3.8) indicates that long-term exposure of teeth may result in severe dental bio-corrosion (Mandel, 2005). The beneficial effects of saliva with its buffering capacity that form the enamel protective film is also diminished by the acidity of the wine (Buzalaf *et al*., 2012). However, some studies have shown that wine tasters with high risk of dental biocorrosion did not develop the lesion, the objective was to investigate the apparent individual susceptibility to enamel biocorrosion. The variation in susceptibility to biocorrosion among individuals seems to be influenced both by the sustainability of the enamel and by factors in the oral environment. This could explain the variation in the prevalence and severity of NCCL among patients exposed to acid attacks, suggesting that, for some individuals, only minimal acid exposure may be sufficient to cause damage to teeth, while others may never develop dental biocorrosion despite extensive acid exposure. Still in relation to the occupation of the individual, it is worth remembering professional swimmers who are constantly in contact with the chlorine used for cleaning and maintenance of swimming pools and releases gases with erosive potential, which, like wine, risk is associated with the susceptibility of the individual, duration of the erosive challenge and amount of ingestion / training (Buczkowska-Radlińska, *et al*., 2013).

Conclusion

The increase in the prevalence of NCCL is directly related not only to toothbrushing or abrasion by dentifrice and occlusal trauma, but also to a strong influence of biocorrosive factors such as acidic diet, occupational factors and habits. Therefore, it is of paramount importance to the clinician to distinguish the etiological factors, the characteristics that differentiate them, and their consequences, so that there is a control of the risk factors added to the clarification and awareness of the patient, propitiating to correctly target the appropriate therapy reestablishing function and aesthetics of lasting form.

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REFERENCES

Abrahamsen T.C. 2005. The worn dentition pathognomonic patterns of abrasion and erosion. *Int Dent J.,* 55:268–76.

Addy M.; Shellis R.P. 2006. Interaction between attrition, abrasion and erosion in tooth wear. *Monogr Oral Sci*., 20:17–31.

Aykut-Yetkiner A.; Wiegand A.; Roay V.; Attin R.; Becker K.; Attin T. 2014. In vitro evaluation of the erosive potential of viscosity-modified soft acidic drinks on enamel. *Clin Orl Invest.* 18(3): 769-773.

Barac R.; Gasic J.; Trutic N.; Sunaric S.; Popovic J.; DJEKIC P.; Radenkovic G.; Mitic A. 2015. Erosive effect of diferente sofht drinks on enamel surfasse in vitro: Application of Stylus Profilometry. *Med Princ Pract*. 2015; 24(5):451-457.

Barbosa L.; Prado-Junior R.; Mendes R. 2009. Lesões cervicais não-cariosas: etiologia e opções de tratamento restaurador. Revista Dentística on line., 8(18):5-10.

Bartlett D. 2009. Etiology and prevention of acid erosion. *Compend Contin Educ Dent.,* 30:616–20.

Bartlett D.W.; Shah P. 2006. A critical review of non-carious cervical (wear) lesions and the role of abfraction, erosion and abrasion. *J Dent Res*. 85(4): 306-312.

Benjakul P.; Chuenarrom C. 2011. Association of dental enamel loss with the pH and titratable acidity of beverages. *J Dent Sci.,* 6:129–133.

Buczkowska-Radlińska J.; Łagocka R.; Kaczmarek W.; Górski M.; Nowicka A. 2013. Prevalence of dental erosion in adolescent competitive swimmers exposed to gas-chlorinated swimming pool water. *Clin Oral Investig* 17(2):579-83.

Buzalaf M.A.R.; Hannas A.R.; Kato M.T. Saliva and dental erosion. *J. Appl. Oral Sci.*, 2012; 20(5).

Carvalho J.M.; Maia G.A.; Sousa P.H.M.; Rodrigues S. 2006. Major compounds profiles in energy drinks: caffeine, taurine, guarana, and glucoronolactone. Rev Inst Adolfo Lutz, 65:78–85.

Carvalho T.S.; Colon P.; Ganss C.; Huysans M.C.; Lussi A.; Schlueter N. et al. 2015. Consensus Reporto f the European Federation of Conservative Dentistry – Erosive tooth wear: diagnosis and management. *Clin Oral Investig.* 19(7): 1557-61. doi: 10.1007/s00784-015-1511-7.

Cheung A.Z.; Zid D.; Hunt and Mcintyre J. 2005. "The potential for dental plaque to protect against erosion using an in vivo-in vitro model: a pilot study," *Australian Dental Journal.* 50(4): 228–234.

Corrâ F.O.B.; Sampaio J.E.C.; Rossa J.R.C.; Orrico S.R.P. 2004. Influence of natural fruit juices in removing the smear layer from root surfaces: an in vitro study. JCDA: *J Can Dental Assoc* 70:697–702.

Dawes C. 2003. What is the critical pH and why does a tooth dissolve in acid?. *J Can Dent Assoc*., 69:722–724.

DE Melo M.A.; Passos V.F.; Lima J.P.; Santiago S.L.; Rodrigues L.K. 2016. Carbohydrate-electrolyte drinks exhibit risks for human enamel surfasse loss. *Restor Dent Endod.* 41(4):246-254.

Dodd H.W.J.; Gragg P.P.; Darrel R. 1997. The effect of some Mexican citric acid snacks on in vitro tooth enamel erosion. *Pediatr Dent* 19:339-340.

Dzakovich J.J.; Oslak R.R. 2008. In vitro reproduction of noncarious cervical lesions. *J Prosthet Dent.* 100(1):1–10

Eccles, J.D. Tooth. 1982. Surface lossfrom abrasion, attrition and erosion. Dent Update. 9:373-381.

Gonçalves G.K.M. et al. 2012. Erosive potential of different types of grape juices. *Braz Oral Res.,* (São Paulo) 2012; 26(5):457-63.

Gripo J.O.; Chaiyabutir Y.; Kois J.C. 2013. Effects of cyclic Fatigue Stress-Biocorrosion on Noncarious Cervical Lesion. *Journal of Esthetic na Retorative Dentistry.* 25(4):265-272.

Grippo J.O. 1991. Abfractions: a new classification of hard tissue lesions of teeth. *J Esthet Dent.,* 3:14–19.

Grippo J.O.; Simring M.; Coleman T.A. 2012. Abfraction abrasion, biocorrosion, and the enigma of noncarious cervical lesions: a 20-year perspective. *J Esthet Restor Dent*., 24:10–25.

Hoeppner M.G.; Massarollo S.; Bremm L.L. 2007. Considerações clínicas das lesões cervicais não cariosas. UEPG Ci Biol Saúde. 13:81-86.

Jager D.H.; Vieira A.M.; Ruben J.L., et al. 2012. Estimated erosive potential depends on exposure time. *J Dent* 40:1103–1108.

Johansson A.K.; Johansson A.; Birkhed D.; Omar R.; Baghdadi S.; Khan N.; Carlsson G.E. 1997. Dental erosion associated with softdrink consumption in young Saudi men. *Acta Odontol Scand*., 55:390–397.

Johansson A.K.; Sorvari R.; Birkhed D.; Meurman J.H. 2001. Dental erosion in deciduous teeth—an in vivo and in vitro study. *J Dent.,* 29:333–340.

Kliemann, C. 2002. Lesões cervicais não-cariosas por abrasão: escovação traumática. *Jornal Brasileiro de Clínica Odontológica Integrada.* 6(33): 204-209,.

Kwek S.X.; Mian M.; Hall C.; Xie Z.; Young R.; Kaidonis J.; Townsend G.C.; Ranjikar S. 2015. Nanoscratch testinf for the assessment of enamel demineralization under conditions simulating wine erosion. 2015; 60(1):12-17.

Lee W.C.; Eakle W.S. 1996. Stress-induced cervical lesions: Review of advances in the past 10 years. *J Pristhet Dent.* 75:487-494.

Levitch L.C.; Bader J.D.; Shugars D.A.; Heymann H.O. 1994. Non-carious cervical lesions. *J Dent,* 22(4): 195-207.

Lodi C.S.; Sassaki K.T.; Fraiz F.C.; Delbem A.C.; Martinhon C.C. 2010. Evaluation of some properties of fermented milk beverages that affect the demineralization of dental enamel. *Braz Oral Res.* 24(1):95-101.

Lussi A. 2006. "Erosive tooth wear: a multifactorial condition of growing concern and increasing knowledge," *Monographs in Oral Science.* 20:1-8.

Lussi A. and Jaeggi T. 2006. "Occupation and sports," in Dental Erosion: From Diagnosis to Therapy, A. Lussi, Ed., pp. 106–111, Karger, Basel, Switzerland.

Lussi A.; Hellwig E.; Zero D. and Jaeggi T. 2006. "Erosive tooth wear: diagnosis, risk factors and prevention," *American Journal of Dentistry*. 19(6): 319-325.

Lussi A.; Kohler N.; Zero D.; Schaffner M.; Megert B. 2000. A comparison of the erosive potential of different beverages in primary and permanent teeth using an in vitro model. *Eur J Oral Sci.*, 108(2):110-14.

Lussi A.; Marincek M.V.S.; Ganss C.; Hellwig E.; Cheaib Z.; Jaeggi T. 2012. Clinical study monitoring the pH on tooth surfaces in patients with and without erosion. *Caries Res* 46:507–512.

Lussi A.; Schlueter N.; Rakhmatullina E.; Ganss C. 2011. Dental erosion – an overview with emphasis on chemical and histopathological aspects. *Caries Res.,* 45: 2–12.

Lussi, A.; Jaeggi, T.; Schaffner M. 2002. *Diet an dental erosion. Nutrition. 18*(9):780-781.

Mahoney E.; Beattie J.; Swain M.; Kilpatrick N. 2003. Preliminary in vitro assessment of erosive potential using the ultra-microindentation system. *Caries*; 37: 218–224.

Mair L.H. Wear. 1992. In dentistry – current terminology. *J Dent,* 20:140-144.

Mandel L. 2005. "Dental erosion due to wine consumption," *Journal of the American Dental Association*. 136(1):71–75.

Marochi C.; Queiroz V. 2001. Lesões não-cariosas. In: Gomes J, editor. Estética em clínica odontológica. Curitiba. 151-94.

Mcintyre J.M. Erosion. 1992. *Aust Prosthodont J.* 6:17-25

Mesquita-Guimarães K.S.F.; Scatena C.; Borsatto M.C.; Rodrigues-Júnior A.L.; Serra M.C. 2015. Effect of foods and drinks on primary tooth enamel after erosive challenge with hydrochloric acid. *Braz Oral Res* [online]. 29(1):1-7.

Milosevic, A. 1997. Sport drinks hazard to teeth. *Br J Sports Med., 31*:28-30.

Mok T.B.; Mcintyre J. and Hunt D. 2001. "Dental erosion: in vitro model ofwine assessor's erosion," *AustralianDental Journal.*, 46(4): 263–268.

Nascimento M.M.; Dilbone D.A.; Pereira P.N.R.; Duarte W.R.; Geraldeli S.; Delgado A.J. 2016. Abfraction lesions: etiology, diagnosis, and treatment options. *Clinical, Cosmetic and Investigational Dentistry.* 8 doi: 143.107.153.169.

Pinto S.C.; Bandeca M.C.; Silva C.N.; Cavassim R.; Borges A.H.; Sampaio J.E. 2013. Erosive potential of energy drinks on the dentine surfasse. *BMC Res Notes.* 6:67. doi: 10.1186/1756-0500-6-67.

Ress H.S.; Loyn T.; Rowe W.; Kunst Q.; Mcandrew R. 2006. The ability of fruit teas to remove the smear layer: na in vitro study of tubule patency. *J Dent* 34(1):67-76.

Richards D. 2016. Impacto f diet on tooth erosion. Evid Based Dent. 17(2):40. doi: 10.1038/sj.ebd.6401164.

Rytömaa I.; Järvinen V.K.; Kanerva R.; Heinonen O.P. 1998. Bulimia and tooth erosion. *Acta Odontol Scand* 56:36-40.

Schlueter N.; Hardt M.; Klimek J.; Ganss C. 2010. Influence of the digestive enzymes trypsin and pepsin in vitro on the progression of erosion in dentine. *Arch Oral Biol* 55:294–9.

Shellis R.P.; Barbour M.E.; Jones S.B.; Addy M. 2010. Effects of pH and acid concentration on erosive dissolution of enamel, dentine, and compressed hydroxyapatite. *Eur J Oral Sci*., 118:475–482.

Sirimaharaj, V., Messer L.B., Morgan M.V. 2002. Acid diet and dentl erosion among athletes. *Australian Dental Journal.* 47(3):228-236.

Soares, P.V.; Grippo, J.O. 2017. Noncarious Cervical Lesions and Cervical Dentin Hypersensitivity. 1st edition.

Thewine Institute, "Per capita wine consumption by country", 2010, http://www.wineinstitute.org/files/PerCapita WineConsumptionCountries. pdf.

Torres C.P.; Chinelatti M.A.; Gomes-Silva J.M., et al. 2010. Surface and subsurface erosion of primary enamel by acid beverages over time. *Braz Dent J.,* 21:337–345.

Wiegand A. and Attin T. 2007. "Occupational dental erosion from exposure to acids: a review," *Occupational Medicine.* 57(3):169–176.

Wongkhantee S.; Patanapiradej V.; Maneenut C.; Tantbirojn D. Effect of acidic food and drinks on surfasse hardness of enamel, dentine, and tooth-coloured filling materials. *J Dent.*, 34(3): 214-220.

Young W.G. 1995. Diet and nutrition for oral health: advice for patients with tooth wear. Aust Dent Assoc News Bulletin., 224:8-10.

Zero D.T. 1996. Etiology of dental erosion – extrinsic factors. *Eur J Oral Sci.,* 104:162-177.

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