Do Oral hygiene and Diet Favor The Development of Non-Carious Cervical Lesions? A Retrospective Study.

Hector deLlanos-Lanchares  
University of Oviedo

Leticia Alvarez-Menendez  
Lucus Augusti University Hospital

Jose Antonio Alvarez-Riesgo  
University of Oviedo

Alicia Celemin-Viñuela  
Complutense University of Madrid

Ildefonso Serrano-Belmonte  
University of Murcia

Angel Alvarez-Arenal  (arenal@uniovi.es)  
University of Oviedo

Research Article

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Abstract

**Purpose:** The aim of this retrospective case-control study was to evaluate the influence of dental brushing factors, diet, the consumption of acidic drinks and Community Periodontal Index of Treatment Needs (CPITN) on the development of non-carious cervical lesions (NCCLs).

**Methods:** The sample consisted of undergraduate dentistry students from different Spanish faculties (age range 18 to 29 years). NCCLs and the CPITN were diagnosed and recorded using a periodontal probe. A questionnaire was used to record different brushing factors, the consumption of extrinsic acids and the presence of intrinsic acids. The data obtained were analysed using unconditional uni- and multivariate logistic regression (significance level p<0.05).

**Results:** Brushing force was a risk factor (OR=1.71). The presence of NCCLs is significantly more frequent in subjects who brush their teeth vigorously. Frequent consumption of salads with vinegar or lemon increases the risk of NCCLs (OR=4.5). As the CPITN score increases, the risk of NCCLs also increases significantly (OR=1.93) for value 1 and OR=6.49 for CPITN of 3. The consumption of extrinsic acids associated with salads seasoned with vinegar or lemon, the brushing force and the CPITN were the risk factors. The model obtained has a 67.14% predictive capacity for NCCLs, a specificity of 76.43%, and a sensitivity of 57.86%.

**Conclusions:** the results of this study show that brushing force, and acidic diet and CPITN significantly increase the risk of NCCLs. Other variables are needed to increase model prediction.

Introduction

A non-carious cervical lesion (NCCL) is defined as a tooth structure loss of non-bacterial etiology in the cervical region at the enamel-cement junction. In dental clinic practice, these lesions are found to occur frequently; however, previous reviews and studies report a wide prevalence range of between 5 and 85% [1–8]. Bias in the selection and sample size and in the lesion diagnosis, age and prolonged exposure to some risk factor may explain this variability.

NCCLs are currently considered a complex process of multifactorial etiology. Several factors with different pathogenic mechanisms can influence the presence and development of these lesions. Wear in the enamel-cement junction related to phenomena of abfraction, dental abrasion or erosion, alone or in combination, are the mechanisms mainly accepted [4, 8–14]. Dental erosion is described as the loss of tooth enamel and dentine as a result of a process of chemical degradation due to the consumption of acidic foods and soft-drinks or due to gastroesophageal reflux or self-induced vomiting. The regurgitated intrinsic acids may dissolve the hydroxyapatite crystals of the enamel because they have a pH well below the average saliva value of pH 6.7. Abrasion is the wear of the dental structure due to a mechanical process related to the brushing of teeth and abfraction is the process of tooth wear due to excessive occlusal force that increases the stress on the amelocementary union causing microfractures of the enamel hydroxyapatite crystals [9].
However, the relationship between the cited factors and the origin and evolution of NCCLs is under discussion and requires further information.

Systematic reviews, different clinical and in vitro studies support the association between occlusal alterations or occlusal stress with the progression of NCCLs [6, 15–18], while other studies reject the influence of traumatic occlusal forces on the pathogenesis and development of NCCLs [4, 8, 19–21]. Likewise, the studies carried out to evaluate the influence of tooth brushing factors (frequency, brushing technique and strength, bristle hardness and use of toothpaste) show contradictory results. NCCLs have been identified in populations that do not brush their teeth [22, 23] and no significant association with oral hygiene factors has been found [24]. In subjects who brush their teeth, several clinical studies have reported a significant relationship of NCCLs with different brushing factors, alone or in combination with other risk factors [1, 3, 5, 8, 20, 25–27], while other studies do not find such a clear relationship [16, 28–30]. A recent meta-analysis even reports that the results that lend weight to the association between tooth brushing and NCCLs are not conclusive [25]. Gastroesophageal reflux or eating disorders [1, 31–33], as well as dietary factors related to the consumption of salads seasoned with vinegar/citrus or cola/citrus flavored soft-drinks that lower pH in the oral cavity below salivary pH, have been implicated in dental wear and in the pathogenesis and progression of NCCLs [1, 2, 26, 33, 34]. Conversely, other clinical studies do not confirm the influence of diet on the progression of NCCLs [16, 30]. On the other hand, irrespective of the effect of a single risk factor in the development of NCCLs, clinical studies have been carried out that have shown different combinations of risk factors as predictors or related to the development of NCCLs [3, 5, 7, 8, 16, 20, 26, 30, 33].

The variability in sample selection, data collection strategy and the heterogeneity of most of these clinical studies with designs of low level of scientific evidence have produced results that have not been able to conclusively relate cause (risk factors) and effect (NCCLs). Therefore, the contribution of the considered risk factors to the origin and development of NCCLs has not been sufficiently proven for dentists to be able to act to prevent their occurrence. More clinical studies with a better scientific evidence are needed to clarify it. With this aim in mind, a retrospective case-control study was carried out with the following hypothesis: “the consumption of acidic foods and drinks represents a more important risk factor than the brushing of teeth in the development of NCCLs”. Furthermore, the objective of this study is to achieve the following: 1. Estimate the frequency and odds ratio (OR) of the independent variables under study. 2. Find the best predictive model to diagnose NCCLs.

Materials And Methods

SAMPLE. The subjects of the sample were third-, fourth- and fifth-year students of dentistry from 6 randomly selected Spanish university faculties. In Spain, no prevalence data of non-carious cervical lesions are available. For this reason, an odds ratio of 2 and a proportion of cases of 50% were assumed. With these assumptions, a sample size of 274 individuals (137 cases and 137 controls) was obtained without the Yates correction, for a confidence level of 95% and a power (1-β) of 80%. Each School of Dentistry selected an average of 50 subjects (25 cases and 25 controls), the final sample size being 280 subjects (140 cases and 140 controls).
The descriptive data relating to age and sex are compiled in Table 1. The inclusion criteria for the cases were to have at least one NCCL of any shape and with a level of cervical wear corresponding to level 2 or more (defect less than 1 mm deep) of the classification of the Tooth Wear Index [35]. The exclusion criteria were malocclusion treatment, prosthetic restorations and restorations or caries in the cervical region of the teeth. With the same exclusion criteria, controls of similar sex and age were selected. However, no matching strategy was followed in the selection of controls, which may be a bias to transfer the results to the population of different demographics and ages.

In each school of dentistry, the protocol was explained to all participants, who were invited to participate and to sign and informed consent. An initial clinical examination was carried out on students who decided to participate by a single trained dentist (associate professor at each School of Dentistry) in order to select those that met the requirements to be included in the case group and also to select the controls. Once selected, all participants signed an informed consent. Subsequently, both the cases and the controls were asked to answer the questionnaires and a final clinical examination was performed to confirm the NCCLs and to register the variable of interest Community Periodontal Index of Treatment Needs (CPITN), as described below.

Prior to the selection of participants, a scientific committee accepted all the study procedures. This study was also carried out in accordance with the ethical principles of the Declaration of Helsinki of the World Medical Association, revised version, Brazil 2013.

**QUESTIONNAIRES.** Each selected participant was interviewed by a dentist examiner and asked to respond to a structured and questionnaire with no open-ended answers. The questionnaire is based on and is similar to those used in previous epidemiological and clinical studies that evaluate the risk factors of the NCCLs. The questions about NCCL risk factors were read to the subjects and their answers recorded. The questionnaire included questions about oral hygiene habits, eating and drinking habits, and gastroesophageal reflux. Based on the questionnaire, 12 independent variables were collected: age, sex, frequency ($\leq 2$, $>2$ a day) and force of tooth brushing (smooth/medium, hard), toothbrush bristle hardness, method of toothbrushing (mainly vertical/variable, mainly horizontal), consumption of fresh acidic fruits and juices ($\leq 2$, $>2$ a day), drinking soft or carbonated beverages (1, $>1$ a day), eating salads with vinegar or lemon ($\leq 2$, $>2$ a day), and gastroesophageal reflux or frequent vomiting (yes, no). Furthermore, each item included the answer: Doesn't know/No answer (Dk/Na).

**CLINICAL EXAMINATION.** The selected subjects from each School of Dentistry were examined in a dental chair. A case was diagnosed when an NCCL was detected visually or by means of a periodontal probe in the cervical region of any anterior or posterior tooth. The tip of the periodontal probe was placed perpendicularly in the cervical region and was moved gently across the buccal and lingual/palatal tooth surfaces in order to detect the presence or absence of NCCLs. To evaluate the CPITN the probe was carried out with a calibrated periodontal probe, on all 4 faces (mesial, distal, buccal and lingual/palatine) of the teeth 1.6, 1.1, 2.6, 3.6, 3.1 and 4.6. The examiner noted the value of each tooth according to the scale (0 healthy, 1 bleeding on probing, 2 dental calculus, 3 pocket $\leq 5$ mm, 4 pocket $\geq 6$ mm). Only the highest value found in the six teeth probed was recorded in data collection.
The CPITN was evaluated and the definitive case and control selection made in a final clinical examination. The inter-examiner variability for the NCCL or non-NCCL item was evaluated using Cohen's kappa coefficient in 14 subjects of the School of Dentistry of Madrid, with an average result of 0.82 with respect to the gold standard. The intra-observer variability (0.89) was calculated by repeating the same examination on the same subjects 3 weeks later.

STATISTICAL ANALYSIS. The data obtained were analysed descriptively and using unconditional univariate and multivariate logistic regression analysis. The odds ratio (OR) and 95% confidence interval (CI) were calculated. Also evaluated was the predictive capacity of the model and the area under the Receiver Operating Characteristic (ROC) curve. This multivariate analysis shows the effect of each of the independent variables, adjusting the effect of the rest of variables included in the model. In other words, each independent variable is adjusted by the effect of the rest of the independent variables. The data were analysed by means of a Stata v.13 (Stata Corp LLC. College Station, Texas. USA) statistical application package.

Results

A. UNIVARIATE LOGISTIC REGRESSION. Table 1 shows the descriptive data of the cases and controls as well as the OP values and p-values of the CPITN and the frequency and characteristics of tooth brushing variables. Of all the variables related to tooth brushing, only the subjects that have reported vigorous brushing show significantly more NCCLs ($p = 0.035$, 1.7 times more) than those who do not. Likewise, an increase in CPITN significantly increases the risk of NCCLs. Table 2 shows the data relating to extrinsic acid consumption and the presence of intrinsic acids. The consumption of soft drinks as well as the ingestion of acidic or citrus fruits does not reveal a statistical association with the presence or absence of NCCLs. By contrast, the data show a statistically significant association of the NCCLs with frequent consumption of salads with vinegar or lemon and the consumption of extrinsic acids (this last variable includes at least one positive response from subjects to soft drink consumption, consumption of acidic fruits or well-seasoned salads).

B. MULTIVARIATE LOGISTIC REGRESSION. Table 3 shows the results of the multivariate logistic regression analysis with variables that are risk factors or have an influence on the onset of NCCLs. A CPITN greater than 1, vigorous brushing of teeth and eating two or more seasoned salads a day are the risk factors of the predictive model of probability for the presence of NCCLs. Figure 1 shows the ROC curve. The area under the curve indicates the predictive power of the model. The 0.725 value of the ROC curve of this study is in an intermediate position between the values 0.5 and 1. A value of 1 corresponds to the model having the greatest discriminative power, 100% specificity and sensibility; a value of 0.5 indicates the lack of predictive power. In addition, the model correctly classifies 67.14% of the cases. Figure 2 shows the behavior of tendencies of sensitivity and specificity for to the chosen cut-off point.

Discussion
This study, which was carried out to evaluate the influence of hygiene, brushing technique and acid consumption factors on the initiation and progression of NCCLs, shows that brushing teeth twice or more times a day does not significantly influence the appearance of lesions of this type. Although brushing the teeth is a basic rule of dental hygiene to prevent tooth decay and periodontal disease, the greater or lesser frequency with which it is performed does not appear to be a preventive factor for NCCLs. Studies that make a reference to this variable show mixed results, with clinical studies and meta-analyses that report a significant association between a frequency of brushing of two or more times a day and NCCLs [7, 25, 36] and others which do not support this relationship [2, 5, 8, 16, 27, 28]. Nevertheless, the frequency of brushing can become an important factor associated with the vertical or horizontal brushing technique [1, 5, 36]. In addition, the parts played by this last factor and the hardness of bristles are also controversial. Similar to the data in this study, previous studies have not reported a significant relationship of these factors in the origin and progression of NCCLs [16, 24, 29, 37]. However, recent cross-sectional studies with and without logistic regression analysis [26–28] and in vitro studies [38] underline the influence of bristle hardness on the onset progression of NCCLs. Also, unlike the findings of this study, the brushing technique, especially horizontal [1, 2, 5, 25, 26] is another factor associated with NCCLs. Irrespective of the frequency, brushing technique and bristle hardness, tooth brushing force is shown in this study as an NCCL risk factor, with NCCLs being 1.7 times more frequent in those subjects who brush their teeth vigorously compared to those who do not. Although with differences in the evaluation of tooth-brushing force, this result concurs with previous studies that have indicated a significant association between brushing force and the presence of NCCLs [1, 3, 8, 20, 27]. However, a 5-year prospective clinical study carried out in 2016 [16] does not find this association, evaluating the brushing force with the aid of videos that record the participants brushing their teeth for a minute. Nor was brushing with excessive force a risk factor in the 185 patients and 5,180 teeth examined in the study by Teixeira et al, 2018 [30].

Although the idea of a model formulated with variables related to tooth brushing (toothbrushing frequency, toothbrushing technique, bristle hardness, duration of tooth-brushing, toothbrushing force and toothpaste) may be attractive for explaining the etiology, progression or aggravation of NCCLs, available scientific evidence does not support this. With differences in the research design, sample selection and size and in the methodology of evaluation and recording of the variables, the results of the various studies for each and every brushing factor differ and are therefore not conclusive. Brushing factors may be necessary but not sufficient for the development of NCCLs. Variables, other than brushing ones, are needed to explain the occurrence of NCCLs in populations that do not brush their teeth or have poor dental hygiene [22–24] or the appearance of NCCLs on the lingual surfaces of the teeth where the brush hardly reaches.

The current study finds that the Community Periodontal Index of Treatment Needs (CPITN) behaves as an important risk factor in the onset of NCCLs, with odds ratio values increasing progressively as the CPITN rises. This data is similar to the results provided by a cross-sectional study that reported a significant association of NCCLs with oral hygiene [30] and also with another study performed with people between 20 and 29 years of age that reported loss of attachment in most teeth with NCCLs. [5]. Likewise, a significant increase in the number of cervical wear lesions in relation to calculus index and the frequency of periodontal pockets has been reported [24], as the simultaneous presence of cervical wear with calculus,
plaque or periodontitis [39]. Notwithstanding, the CPITN data of the present study, considered in isolation, must be viewed with caution. Under normal conditions, considering the result of the inflammatory activity of the periodontal microbiota in the different stages of the CPITN, it is unlikely that a significant change occurs in the pH or acid-base environment in the crevicular fluid at the level of the amelocementary union and that dental wear is initiated. At the same time, it is well known that the gingival sulcus has a slightly alkaline pH that oscillates towards greater alkalinity with an increase in the periodontal conditions [40] or even towards slight acidity in chronic periodontitis [41], though not approaching the value of 5.5, considered to be the critical pH level for enamel demineralization to occur. Therefore, the concurrence of some other factor is required. Therefore, a previous study in older patients (59.3 years average) found no significant association between bacterial plaque accumulation and pocket depth [11]. According to data from the present logistic regression model, CPITN may be considered as a predisposing or additional factor that, together with vigorous brushing and acidic food/beverage consumption, could favor the progression of NCCLs. The action of brushing factors in concert with dental erosion in the development of NCCLs is suggested or reported in different clinical and laboratory studies [1, 3, 11, 14, 30, 42]. Exposure an acidic diet might weaken the enamel/dentin, making it more susceptible to wear through the action of brushing.

Erosive theory without bacterial interaction as a mechanism of dental wear or initiation and progression of NCCLs is highly appealing for clinicians. Thus, both a recent systematic review with meta-analysis [43] and several previous studies support the association with an acidic diet and beverage consumption [1–3, 10, 26, 28, 29], gastroesophageal reflux or eating disorders [1, 30–33], and even with certain professions or work in an acid environment [34, 44]. In contrast, other previous cross-sectional [45, 46] and prospective studies [16] have found no significant relationship between the acidic diet and the progression of NCCLs. This study supports the importance of the consumption of extrinsic acids, and, in particular, the frequent ingestion of salads seasoned as a risk factor for the development of NCCLs. In the univariate logistic regression analysis, NCCLs were significantly more frequent (4.57 times) in those subjects who consumed two or more salads a day (1.75 times in the multivariate analysis) and 2.14 times more frequent with the consumption of extrinsic acids of any nature. These data agree with the main conclusion of a very recent meta-analysis that reports twice the risk of dental erosion (OR = 2.40) in subjects with a vegetarian diet compared to non-vegetarians [43].

Nevertheless, the relationship between an acidic diet and NCCLs, without other risk factors being involved, is a matter for debate. In this regard, the data in this study show that aggressiveness in brushing and the CPITN are the risk factors, which, combined with the consumption of salads seasoned with vinegar or lemon, constitute a model that classifies correctly 67.14% of cases, with 57.86% sensitivity, 76.43% specificity and an area under the ROC curve of 0.723. In general, it is not an excessively convincing model, this in turn showing the need to incorporate other risk factors that increase its predictive capacity. It does not agree, therefore, with the results of previous clinical studies based on less scientific evidence (cross-sectional designs) that indicate as predictors of NCCLs only the interaction of frequently eating fresh fruit and power toothbrusing [3] or the consumption of different extrinsic acids in combination with an age of over 35 years old [28] or in combination with bruxism and gastroesophageal reflux [1]. Instead, the data
more closely matches the model of the old Bader’s case-control study [47] that, in addition to diet (fruit juices) and hard brushing as risk factors, includes occlusal alterations and bruxism.

The previous exposure of the amelocementary union to an intrinsic or extrinsic acid environment that demineralizes and weakens the enamel through degradation of the hydroxyapatite favors an increase in tooth wear due to the friction of brushing with and without toothpaste. The lower mineral content, with more voluminous pores, reported in the cervical enamel near the amelocementary junction makes this area more susceptible to demineralization. This favors the formation of steps of different length and depth [48]. This, according to Rees [49], additionally facilitates the entry of erosive agents through the pores, thus weakening the enamel even further. Additionally, proteolytic enzymes (proteases) of the gingival crevicular fluid produced by bacterial plaque microorganisms may also contribute to biocorrosion-induced wear [9]. The present study supports the combination of these risk factors (tooth brushing and acidic diet) in the onset and development of NCCLs, though data analysis of the model indicates that the action of more and different factors may be necessary to increase their predictive capacity. At all events, in accordance with the results of many other previous studies [1, 3, 4, 8–14, 28, 30, 47] this case-control study supports the multifactorial etiology of NCCLs. However, taking into account the obtained results, it seems sensible to remind dentists to recommend to patients the need for control of tooth brushing force and frequent eating of salads (vegetarian diet).

On the other hand, some limitations should be mentioned. In Spain, there are no community studies that report the prevalence of NCCLs to calculate sample size. In agreement with Thompson [50], the size chosen for the assumed OR is a conservative estimate of the sample size to combine clinical and epidemiological usefulness with the cost of the study. The sample comes from a very homogeneous population, dentistry students of the last three academic years. Although the cases and controls are comparable to each other in age and gender, a matching strategy between them was not followed. All this is a limitation for the projection of results to a different population. The average age of the participants can also be a limitation, since the majority of clinical and cross-sectioned studies have reported a significant association of NCCLs with a certain age range and increased percentage of NCCLs as age increases [1–3, 5–8, 10, 13, 30, 37]. To estimate the CPITN value, only six teeth were evaluated from the two dental arches and only the value of the one with the worst periodontal condition was recorded. Therefore, the presence or absence of NCCLs in the examined tooth is not taken into account, neither is the periodontal condition of the teeth with NCCls. At the same time, the OR obtained for the highest CPITN score should be interpreted with caution or not taken into consideration due to the relatively small number of cases and controls but large differences in these numbers. With the exception of the CPITN, the values of the rest of the variables come from the answers of the participating subjects to the questions read to them. These are indirect measures and the data obtained from the questionnaires are sometimes not reliable enough. There is no visual or other verification of the variables related to the brushing factors. Nor are the exact types of beverage included since there are some differences in pH and acidity. Likewise, there is no verification of the value of the salivary or gingival sulcus pH. Nor was the saliva analysed to assess the effect of its composition and of its buffering capacity. A direct association of the consumption of acidic foods/beverages with oral acidic environment is assumed. The morphology of the NCCLs was not taken into consideration; if it had been, it could have pointed
towards an erosive or abrasive etiology, although not in a decisive way [48, 51]. All the aforementioned can be a limitation.

In addition, the heterogeneity, limited comparability and limited quality of most studies make it necessary to improve scientific evidence with high quality studies. These should include more effective and efficient designs that allow progress towards a more reliable knowledge of the risk factors involved in the onset and progression of NCCLs, e.g. longitudinal studies that include the most important variables through multivariate analysis.

Conclusions

On the basis of the results and bearing in mind the aforementioned limitations, the following conclusions may be drawn:

1. The force of tooth brushing (OR = 1.94), the frequent consumption of salads seasoned with vinegar/lemon (OR = 1.75) and the CPITN are the main risk factors. Control of brushing force, decreased intake of acidic foods/beverages, and increased hygiene for a lower CPITN value are factors to consider in preventing and reducing the risk of NCCLs.

2. It is not possible to categorically admit or reject the working hypothesis, since of all the variables relating to brushing and consumption of acidic foods and beverages, only one of each group is a risk factor.

3. The predictive capacity, sensitivity and specificity of the model found are in an intermediate position with respect to the ideal one. Future research should assume the need to identify more variables that may exert an influence to improve the prediction capacity of the model and their influence on the NCCLs.

Declarations

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

Ethics approval and consent to participate

The Review Board of University of Oviedo approved the study data collection protocol. All participants also signed informed consent.

Consent for publication

Not applicable

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.
Competing interests

Not applicable

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Author contributions

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Angel Alvarez-Arenal, Leticia Alvarez-Menendez, Jose Antonio Alvarez-Riesgo, Alicia Celemin-Viñuela, Ildefonso Serrano-Belmonte and Hector deLlanos-Lanchares. The first draft of the manuscript was written by Angel Alvarez-Arenal and Hector deLlanos-Lanchares and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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References

1. Zuza A, Racic M, Ivkovic N, Krunic J, Stojanovic N, Bozovic D, Bankovic-Lazarevic D, Vujaskovic M. Prevalence of non-curious cervical lesions among the general population of the Republic of Srpska, Bosnia and Herzegovina. Int Dent J. 2019; 69:281–8. doi:10.1111/idj.12462.

2. Lai ZY, Zhi QH, Zhou Y, Lin HC. Prevalence of non-curious cervical lesions and associated risk indicators in middle-aged and elderly populations in Southern China. Chin J Dent Res. 2015; 18:41–50. doi:10.3290/j.cjdr.a33966.

3. Yang J, Cai D, Wang F, He D, Ma L, Jin Y, Que K. Non-curious cervical lesions (NCCLs) in a random sampling community population and the association of NCCLs with occlusive wear. J Oral Rehabil. 2016; 43:960–6. doi:10.1111/joor.12445.

4. Senna P, Del Bel Cury A, Rösing C. Non-curious cervical lesions and occlusion: a systematic review of clinical studies. J Oral Rehabil. 2012; 39:450–62. doi:10.1111/j.1365-2842.2012.02290.x.

5. Que K, Guo B, Jia Z, Chen Z, Yang J, Gao P. A cross-sectional study: non-curious cervical lesions, cervical dentine hypersensitivity and related risk factors. J Oral Rehabil. 2013; 40:24–32. doi:10.1111/j.1365-2842.2012.02342.x.

6. Brandini DA, Trevisan CL, Panzarini SR, Pedrini D. Clinical evaluation of the association between noncarious cervical lesions and occlusal forces. J Prosthet Dent. 2012; 08:298–303. doi:10.1016/S0022-3913(12)60180-2.
7. Jiang H, Du MQ, Huang W, Peng B, Bian Z, Tai BJ. The prevalence of and risk factors for non-carious cervical lesions in adults in Hubei Province, China. Community Dent Health. 2011; 28:22–8. doi:10.1922/CDH_2492Tai07.

8. 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; 36:774–9. doi:10.1016/j.jdent.2008.05.009.

9. Grippo JO, Simring M, Coleman TA. Abfraction, abrasion, biocorrosion, and the enigma of noncarious cervical lesions: a 20-year perspective. J Esthet Restor Dent. 2012; 24:10–23. doi:10.1111/j.1708-8240.2011.00487.x.

10. Yoshizaki KT, Francisconi-Dos-Rios LF, Sobral MA, Aranha AC, Mendes FM, Scaramucci T. Clinical features and factors associated with non-carious cervical lesions and dentin hypersensitivity. J Oral Rehabil. 2017; 44:112–8. doi:10.1111/joor.12469.

11. Pikdöken L, Akca E, Gürbüzer B, Aydil B, Taşdelen B. Cervical wear and occlusal wear from a periodontal perspective. J Oral Rehabil. 2011; 38:95–100. doi:10.1111/j.1365-2842.2010.02137.x.

12. Pecie R, Krejci I, Garcia-Godoy F, Bortolotto T. Noncarious cervical lesions - A clinical concept based on the literature review. Part 1: prevention. Am J Dent. 2011; 24:49–56.

13. Wood I, Jawad Z, Paisley C, Brunton P. Non-carious cervical tooth surface loss: a literature review. J Dent. 2008; 36:759–66. doi:10.1016/j.jdent.2008.06.004.

14. 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; 85:306–12. doi:10.1177/154405910608500405.

15. Leal NMS, Silva JL, Benigno MIM, Bemerguy EA, Meira JBC, Ballester RY. How mechanical stresses modulate enamel demineralization in non-carious cervical lesions? J Mech Behav Biomed Mater. 2017; 66:50–7. doi:10.1016/j.jmbbm.2016.11.003.

16. Sawlani K, Lawson NC, Burgess JO, Kinderknecht KE, Givan DA, Ramp L. Factors influencing the progression of noncarious cervical lesions: A 5-year prospective clinical evaluation. J Prosthet Dent. 2016; 115:571–7. doi:10.1016/j.prosdent.2015.10.021.

17. Antonelli JR, Hottel TL, Brandt R, Scarbecz M, Patel T. The role of occlusal loading in the pathogenesis of non-carious cervical lesions. Am J Dent. 2013; 26:86–92.

18. Duangthip D, Man A, Poon PH, Lo ECM, Chu CH. Occlusal stress is involved in the formation of noncarious cervical lesions. A systematic review of abfraction. Am J Dent. 2017; 30:212–20.

19. Silva AG, Martins CC, Zina LG, Moreira AN, Paiva SM, Pordeus IA, Magalhães CS. The association between occlusal factors and noncarious cervical lesions: a systematic review. J Dent. 2013; 41:9–16. doi:10.1016/j.jdent.2012.10.018.

20. Sadaf D, Ahmad Z. Role of brushing and occlusal forces in non-carious cervical lesions (NCCL). Int J Biomed Sci. 2014; 10:265–8.

21. Jepsen S, Caton JG, Albandar JM et al. Periodontal manifestations of systemic diseases and developmental and acquired conditions: Consensus report of workgroup 3 of the 2017 World
Workshop on the Classification of Periodontal and Peri-Implant Diseases and Condition. J Clin Periodontol. 2018; 89 (Suppl 1):S237-S248. doi:10.1002/JPER.17-0733.

22. Faye B, Kane AW, Sarr M, Lo C, Ritter AV, Grippo JO. Noncarious cervical lesions among a non-toothbrushing population with Hansen's disease (leprosy): initial findings. Quintessence Int. 2006; 37:613–9.

23. Ritter AV, Grippo JO, Coleman TA, Morgan ME. Prevalence of carious and non-carious cervical lesions in archaeological populations from North America and Europe. J Esthet Restor Dent. 2009; 21:324–34. doi:10.1111/j.1708-8240.2009.00285.x.

24. Bergström J, Eliasson S. Cervical abrasion in relation to toothbrushing and periodontal health. Scand J Dent Res. 1988; 96:405–11. doi:10.1111/j.1600-0722.1988.tb01575.x.

25. Heasman PA, Holliday R, Bryant A, Preshaw PM. Evidence for the occurrence of gingival recession and non-carious cervical lesions as a consequence of traumatic toothbrushing. J Clin Periodontol. 2015; 42(Suppl 16):S237-55. doi: 10.1111/jcpe.12330.

26. Kumar S, Kumar A, Debnath N, Kumar A, K Badiyani B, Basak D, S A Ali M, B Ismail M. Prevalence and risk factors for non-carious cervical lesions in children attending special needs schools in India. J Oral Sci. 2015; 57:37–43. doi:10.2334/josnusd.57.37.

27. Brandini DA, de Sousa AL, Trevisan CI, Pinelli LA, do Couto Santos SC, Pedrini D, Panzarini SR. Noncarious cervical lesions and their association with toothbrushing practices: in vivo evaluation. Oper Dent. 2011; 36:581-9. doi:10.2341/10-152-S.

28. Kolak V, Pešić D, Melih I, Lalović M, Nikitović A, Jakovljević A. Epidemiological investigation of non-carious cervical lesions and possible etiological factors. J Clin Exp Dent. 2018;10:e648-e656. doi:10.4317/jced.54860.

29. Chan DC, Browning WD, Pohjola R, Hackman S, Myers ML. Predictors of non-carious loss of cervical tooth tissues. Oper Dent. 2006; 31:84–8. doi: 10.2341/04-180.

30. Teixeira DNR, Zeola LF, Machado AC, Gomes RR, Souza PG, Mendes DC, Soares PV. Relationship between noncarious cervical lesions, cervical dentin hypersensitivity, gingival recession, and associated risk factors: A cross-sectional study. J Dent. 2018; 76:93–7. doi:10.1016/j.jdent.2018.06.017.

31. Hermont AP, Oliveira PA, Martins CC, Paiva SM, Pordeus IA, Auad SM. Tooth erosion and eating disorders: a systematic review and meta-analysis. PLoS One. 2014; 9:e111123. doi:10.1371/journal.pone.0111123.

32. Marsicano JA, de Moura-Grec PG, Bonato RC, Sales-Peres Mde C, Sales-Peres A, Sales-Peres SH. Gastroesophageal reflux, dental erosion, and halitosis in epidemiological surveys: a systematic review. Eur J Gastroenterol Hepatol. 2013; 25:135–41. doi:10.1097/MEG.0b013e32835ae8f7.

33. Li W, Liu J, Chen S, Wang Y, Zhang Z. Prevalence of dental erosion among people with gastroesophageal reflux disease in China. J Prosthet Dent. 2017; 117:48–54. doi:10.1016/j.prosdent.2016.04.029.
34. Sirimaharaj V, Brearley Messer L, Morgan MV. Acidic diet and dental erosion among athletes. Aust Dent J. 2002; 47:228–36. doi:10.1111/j.1834-7819.2002.tb00334.x.

35. Smith BG, Knight JK. An index for measuring the wear of teeth. Br Dent J. 1984; 156:435–8. doi:10.1038/sj.bdj.4805394.

36. Özgöz M, Arabaci T, Sumbullu MA, Demir T. Relationship between handedness and toothbrush-related cervical dental abrasion in left- and right-handed individuals. J Dent Sci. 2010; 5:177–82. doi:10.1016/j.jds.2010.11.001.

37. 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; 132:1694–701. doi:10.14219/jada.archive.2001.0122.

38. Turssi CP, Binsaleh F, Lippert F, Bottino MC, Eckert GJ4 Moser EAS, Hara AT. Interplay between toothbrush stiffness and dentifrice abrasivity on the development of non-caries cervical lesions. Clin Oral Investig. 2019; 23:3551–6. doi:10.1007/s00784-018-2776-4.

39. Miller N, Penaud J, Ambrosini P, Bisson-Boutelliez C, Briançon S. Analysis of etiologic factors and periodontal conditions involved with 309 abfractions. J Clin Periodontol. 2003; 30:828–32. doi:10.1034/j.1600-051x.2003.00378.x.

40. Patel RM, Varma S, Suragimath G, Zope S. Estimation and Comparison of Salivary Calcium, Phosphorous, Alkaline Phosphatase and pH Levels in Periodontal Health and Disease: A Cross-sectional Biochemical Study. J Clin Diagn Res. 2016; 10:ZC58-61. doi:10.7860/JCDR/2016/20973.8182.

41. Baliga S, Muglikar S, Kale R. Salivary pH: A diagnostic biomarker. J Indian Soc Periodontol. 2013; 17:461–5. doi:10.4103/0972-124X.118317.

42. Addy M. Tooth brushing, tooth wear and dentine hypersensitivity - are they associated? Int Dent J. 2005; 55(Suppl 1):261–7. doi:10.1111/j.1875-595x.2005.tb00063.x.

43. Smits KPJ, Listl S, Jevdjevic M. Vegetarian diet and its possible influence on dental health: A systematic literature review. Community Dent Oral Epidemiol. 2020; 48:7–13. doi:10.1111/cdoe.12498.

44. Wiegand A, Attin T. Occupational dental erosion from exposure to acids: a review. Occup Med. 2007; 57:169–76. doi:10.1093/occmed/kql163.

45. Mayhew RB, Jessee SA, Martin RE. Association of occlusal, periodontal, and dietary factors with the presence of non-caries cervical dental lesions. Am J Dent. 1998; 11:29–32.

46. Pegoraro LF, Scolaro JM, Conti PC, Telles D, Pegoraro TA. Noncaries cervical lesions in adults: prevalence and occlusal aspects. J Am Dent Assoc. 2005; 36:1694–700. doi:10.14219/jada.archive.2005.0113.

47. Bader JD, McClure F, Scurria MS, Shugars DA, Heymann HO. Case-control study of non-caries cervical lesions. Community Dent Oral Epidemiol. 1996; 24:286–91. doi:10.1111/j.1600-0528.1996.tb00861.x.

48. Cortellini P, Bissada NF. Mucogingival conditions in the natural dentition: Narrative review, case definitions, and diagnostic considerations. J Periodontol. 2018; 89(Suppl 1):S204-13. doi:10.1002/JPER.16-0671.
49. Rees JS. The biomechanics of abfraction. Proc Inst Mech Eng H. 2006; 220:69–80. doi:10.1243/095441105X69141.

50. Thompson WD. Statistical analysis of case-control studies. Epidemiol Rev. 1994; 16:33–50. doi:10.1093/oxfordjournals.epirev.a036143.

51. Igarashi Y, Yoshida S, Kanazawa E. The prevalence and morphological types of non-carious cervical lesions (NCCL) in a contemporary sample of people. Odontology. 2017; 105:443–52. doi:10.1007/s10266-017-0300-y.

Tables

Table 1. Descriptive data of the sample and of the analysis of the Univariate Logistic Regression related to age, gender, CPITN and brushing factors.

Percentage in brackets. OR (odds ratio) values. Confidence interval (CI) in brackets.

Table 2. Descriptive data of the sample and of the analysis of the Univariate Logistic Regression related to the consumption of extrinsic acids and the presence of intrinsic acids.

Percentage in brackets. OR values (odds ratio). Confidence interval (CI) in brackets.

Table 3. Results of the multivariate logistic regression analysis with Odds Ratio (OR) of the model variables.

Confidence interval (CI).

Figures
| Variables          | Cases | Controls | OR (95% CI) | p-value |
|-------------------|-------|----------|-------------|---------|
|                   | Males | Females | Total       | Males | Females | Total |         |
| N (%)             | 53 (18.9) | 87 (31.1) | 140 (50.0) | 53 (18.9) | 87 (31.1) | 140 (50.0) | 1 | 0.98 |
| Age groups (years) |       |          |             |        |          |       | 0.43 |
| 18-20             | 7 (13.2) | 16 (18.4) | 23 (16.4) | 11 (20.8) | 9 (10.3) | 20 (14.3) | Reference category |
| 21-23             | 27 (50.9) | 49 (56.3) | 76 (54.3) | 31 (58.5) | 56 (64.4) | 87 (62.1) | 0.8 | 0.42 |
| 24-26             | 9 (17.0) | 16 (18.4) | 25 (17.9) | 8 (15.1) | 19 (21.8) | 27 (19.3) | 0.8 | 0.60 |
| 27-29             | 10 (18.9) | 6 (6.9) | 16 (11.4) | 3 (5.7) | 3 (3.5) | 6 (4.3) | 2.3 | 0.14 |
| Brushing          |       |          |             |        |          |       | 0.48 |
| Once or twice a day | 14 (10.0) | 19 (13.6) | 33 (23.6) | 9 (6.4) | 14 (10.0) | 23 (16.4) | Reference category |
| More than twice daily | 39 (27.7) | 68 (12.7) | 107 (27.6) | 44 (15.1) | 73 (15.1) | 117 (83.6) | 0.7 |
| Brush bristles    |       |          |             |        |          |       | 0.28 |
| Smooth/medium     | 41 (29.3) | 74 (52.9) | 115 (82.1) | 44 (31.4) | 81 (57.9) | 125 (88.6) | Reference category |
| Hard              | 11 (7.9) | 11 (7.9) | 22 (15.7) | 9 (6.4) | 6 (4.3) | 15 (10.4) | 1.6 | 0.19 |
| Doesn't know/No answer | 1 (0.7) | 2 (1.4) | 3 (2.1) | 0 (0.0) | 0 (0.0) | 0 (0.0) | Reference category |
| Brushing method   |       |          |             |        |          |       | 0.71 |
| Vertical/variable | 46 (32.9) | 76 (54.3) | 122 (87.1) | 47 (31.6) | 77 (57.0) | 124 (88.6) | Reference category |
| Horizontal        | 7 (18.0) | 11 (18.0) | 18 (18.0) | 6 (10.0) | 10 (16.0) | 16 (16.0) | 1.1 |
| Brushing force | 0.28 |
|---------------|------|
| No            |      |
| (No)          |      |
| 23            | 50   |
| (16.4)        | (35.7) |
| 73            | 35   |
| (52.1)        | (25.0) |
| 56            | 91   |
| (40.0)        | (65.0) |
| Reference     |      |
| category      |      |
| Yes           |      |
| (Yes)         |      |
| 25            | 34   |
| (17.9)        | (24.3) |
| 59            | 15   |
| (42.1)        | (10.7) |
| 15            | 28   |
| (25.0)        | (20.0) |
| 43            | 1.7  |
| (30.7)        | (1.0-2.8) |
| Doesn't know/No answer | |
| (Doesn't know/No answer) | |
| 5             | 3    |
| (3.6)         | (2.1) |
| 8             | 3    |
| (5.7)         | (2.1) |
| 3             | 3    |
| (2.1)         | (2.1) |
| 6             | 1.66 |
| (4.3)         | (0.6-5.0) |
| CPITN value   | 0.00 |
| 0             |      |
| (Reference category) | |
| 13            | 25   |
| (9.3)         | (17.7) |
| 38            | 28   |
| (27.1)        | (20.0) |
| 49            | 77   |
| (35.0)        | (55.0) |
| 1             |      |
| (1.1-3.4)     |      |
| 12            | 29   |
| (8.6)         | (20.7) |
| 41            | 15   |
| (29.3)        | (10.7) |
| 28            | 43   |
| (20.0)        | (30.7) |
| 1.9           | 0.03 |
| 2             |      |
| (1.6-9.4)     |      |
| 7             | 10   |
| (5.0)         | (7.1) |
| 17            | 6    |
| (12.1)        | (4.3) |
| 3             | 3    |
| (2.1)         | (6.4) |
| 9             | 3.8  |
| (1.6-9.4)     |      |
| 3             |      |
| (2.88-14.47)  |      |
| 17            | 15   |
| (12.14)       | (10.71) |
| 32            | 3    |
| (22.86)       | (2.14) |
| 7             | 10   |
| (5.00)        | (7.15) |
| 6.48          | 0.00 |
| 4             |      |
| (3.05-193.98) |      |
| 4             | 8    |
| (2.86)        | (5.71) |
| 12            | 1    |
| (8.58)        | (0.71) |
| 1             | 0    |
| (0.00)        | (0.71) |
| 1             | 24.31 |
| (3.05-193.98) |      |
| Variables                        | Cases Males | Cases Females | Cases Total | Controls Males | Controls Females | Controls Total | OR (95% CI) | p-value |
|----------------------------------|-------------|---------------|-------------|----------------|------------------|----------------|-------------|---------|
| **Soft drinks**                  |             |               |             |                |                  |                |            | 0.64    |
| consumed daily                   |             |               |             |                |                  |                |            |         |
| Any                              | 25 (17.7)   | 36 (25.771)   | 61 (43.6)   | 22 (15.7)      | 43 (30.7)        | 65 (46.4)      | Reference category |         |
| Once                             | 21 (15.0)   | 34 (24.3)     | 55 (39.3)   | 21 (15.0)      | 34 (24.3)        | 55 (39.3)      | 1.1 (0.6-1.8)   | 0.81    |
| Twice or more                    | 7 (5.0)     | 17 (12.1)     | 24 (17.1)   | 10 (7.1)       | 10 (7.1)         | 20 (14.3)      | 1.3 (0.6-2.5)   | 0.48    |
| **Acidic or citrus fruits**      |             |               |             |                |                  |                | 0.38        |         |
| Consumed daily                   |             |               |             |                |                  |                |            |         |
| Any                              | 26 (18.6)   | 30 (21.4)     | 56 (40.0)   | 21 (15.0)      | 45 (32.1)        | 66 (47.1)      | Reference category |         |
| Once or twice a day              | 23 (16.4)   | 43 (30.7)     | 66 (47.1)   | 29 (20.7)      | 34 (24.3)        | 63 (45.0)      | 1.23 (0.8-2.0) | 0.40    |
| More than twice daily            | 4 (2.9)     | 14 (10.0)     | 18 (12.9)   | 3 (2.1)        | 8 (5.7)          | 11 (7.9)       | 1.9 (0.8-4.4)  | 0.12    |
| **Salads with vinegar or lemon consumed daily** | | | | | | | | 0.02 |
| Any                              | 19 (13.6)   | 35 (25.0)     | 54 (38.6)   | 37 (26.4)      | 37 (26.4)        | 74 (52.9)      | Reference category |         |
| Once or twice a day              | 29 (20.7)   | 47 (33.6)     | 76 (54.3)   | 14 (10.0)      | 49 (35.0)        | 63 (45.0)      | 1.7 (1.0-2.7)  | 0.04    |
| More than twice daily            | 5 (3.6)     | 5 (3.6)       | 10 (7.1)    | 2 (1.4)        | 1 (0.7)          | 3 (2.2)        | 4.6 (1.2-17.4) | 0.03    |
| **Gastro-esophageal reflux or vomiting/day** | | | | | | | | 0.80 |
| No                               | 52 (37.1)   | 80 (57.1)     | 132 (94.3)  | 49 (35.0)      | 82 (98.6)        | 131 (93.6)     | Reference category |         |
| Variables                      | OR  | Standard error | z    | p-value | 95% CI-OR |
|--------------------------------|-----|----------------|------|---------|-----------|
| **CPITN value**                |     |                |      |         |           |
| 0                              |     |                |      |         | Reference category |
| 1                              | 1.6 | 0.6            | 2.0  | 0.04    | 1.0-3.4   |
| 2                              | 4.3 | 2.0            | 3.1  | 0.00    | 1.7-10.7  |
| 3                              | 6.0 | 2.5            | 4.3  | 0.00    | 2.6-13.7  |
| 4                              | 28.6| 30.7           | 3.1  | 0.00    | 3.5-234.2 |
| **Brushing force**             |     |                |      |         |           |
| No                             |     |                |      |         | Reference category |
| Yes                            | 1.9 | 0.5            | 2.4  | 0.02    | 1.1-3.3   |
| Doesn't know/No answer         | 1.7 | 1.0            | 0.9  | 0.35    | 0.5-5.6   |
| **Salads with vinegar or lemon** |     |                |      |         |           |
| Consumed daily                 | 1.8 | 0.3            | 2.1  | 0.03    | 1.0-3.0   |
| **Constant**                   | 0.3 | 0.1            | 4.6  | 0.00    | 0.2-0.5   |
Figure 1

Graphic representation of Receiver Operating Characteristic curve.
Figure 2

Graph showing the sensitivity and specificity percentage for to the chosen cut-off point.