

Noncarious cervical lesions among a non-toothbrushing population with Hansen's disease (leprosy): Initial findings

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Objective: The purpose of this preliminary investigation was to examine the presence of noncarious cervical lesions (NCCLs) among a convenience sample of non-toothbrushing subjects with Hansen's disease (leprosy). **Method and Materials:** A cross-sectional sample of 102 non-toothbrushing subjects (20 to 77 years of age) was examined. The clinical parameter of interest for this study was the presence or absence of NCCLs and their probable etiology as it relates to the subjects' diet, occlusion, and use of medication. Subjects were examined clinically and interviewed according to study protocol. **Results:** NCCLs were found in 48 subjects (47% of the studied sample). Widespread consumption of acidic foods and beverages acting as corrodents, signs of parafunction, and use of medication that causes xerostomia were also noted. Thus, all may be contributing factors in the etiology of NCCLs in this population. **Conclusion:** This preliminary report suggests that toothbrush/dentifrice abrasion was not a factor in the etiology of NCCLs in the population studied. The authors intend to expand their study among these non-toothbrushing subjects. (*Quintessence Int* 2006;37:613–619)

Key words: abfraction, corrodent, corrosion, friction, mechanisms, noncarious cervical lesion, schema, stress

Noncarious cervical lesions (NCCLs), which are frequently noted in present-day clinical dentistry, have also been found during anthropologic studies.¹ These lesions appear to be

more common in modern man and exist in a variety of geometric patterns. Their etiology and classification are challenging, for there are often several mechanisms that come into play in their onset, thus making most of these lesions multifactorial in origin and progression. Miller, who conducted an exhaustive investigation of noncarious tooth-surface lesions in 1907, was the first to associate these lesions, which he termed "wastings," with chemical and mechanical factors.^{2–4}

Comprehensive studies demonstrating stress in teeth during loading have been done by German researchers Korber⁵ and Lukas and Spranger,⁶ as well as English researchers Haines et al.⁷ Noteworthy, photoelastic^{8,9} and finite element analysis^{10–13} studies have been done, concluding that stress does occur in teeth under loading forces and is greatest in the cervical region. Subsequently, numerous researchers have supported the multifactorial nature of noncarious tooth-surface lesions.^{14–23}

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Authors	Year	Sample origin/size	Type of lesion	Prevalence
Ten Bruggen Cate ²⁵	1968	Workers/555	Abrasion	32%
Sangnes and Gjermo ²⁶	1976	Clinic/533	Abrasion	45%
Berstrom and Lavstedt ²⁷	1979	General/818	Abrasion	31%
Bergstrom and Eliasson ²⁸	1988	Clinic/250	Abrasion	85%
Hand et al ²⁹	1986	Homes/520	Abrasion	56%
Natusch and Klimm ³⁰	1989	Clinic/300	Abrasion Erosion	13% 4%
Jarvinen et al ³¹	1991	Clinic/206	Erosion	5%
Lussi et al ³²	1991	Clinic/391	Erosion	16%
Faye et al (current data)	2004	Clinic/102	Corrosion*/Abfraction**	47%

* Prior to 1995,¹⁹ the term *erosion* would have been used in place of *corrosion* to designate this mechanism.
 ** These lesions were classified as “combined” since both corrosion and abfraction occurred concurrently.

Grippo and Masi^{16,17} concluded that loading forces creating stress in teeth in the presence of a corrodent causing chemical degradation of the mineralized tooth structure should be considered in the etiology of NCCLs. They proposed the use of the term *stress corrosion*, thus implicating the mechanisms by which these lesions develop. Grippo and Simring suggested that stress combined with biocorrosion, caused by bacterial-producing acidic as well as proteolytic corrodents, may be factors in the etiology of cervical and root caries.¹⁹ Although the term *erosion* has been and still is being used to designate chemical degradation of tooth substance, *corrosion*—which is a more precise term—will be used in this paper as suggested by Grippo and Simring.¹⁹

Palamara et al,²³ in 2001, subsequently conducted a study using a servo-hydraulic testing machine to demonstrate that the cyclic loading of teeth in the presence of an acidic corrodent caused the loss of enamel in the cervical region; thus they were the first to establish the synergistic mechanisms of stress and corrosion working in concert, resulting in *fatigue corrosion*. Staninec et al, in 2005, corroborated these findings by cyclically loading dentin and demonstrating that both mechanical stress and lower pH accelerated material loss of the dentinal surfaces.²⁴ Notwithstanding this evidence, there still remain differing opinions among clinicians as to the understanding of the interacting mechanisms, precise terminology, and classification of—as well as the treatment of—

NCCLs. A review of some of the research prior to 1995¹⁹ concerning the classification and prevalence of NCCLs revealed a wide variation in the data, with prevalence ranging from 4% to 85%^{25–32} (Table 1).

In the past, NCCLs were believed by many clinicians to be caused solely by toothbrush/dentifrice abrasion.^{26–29,33–37} Additional factors, such as the mechanisms of stress and corrosion, are now being considered in the etiology of NCCLs.^{16–22} A recent study by Miller et al demonstrated that clinical signs of excessive toothbrushing were lacking among 61 persons with 309 abfraction lesions, “whereas signs of occlusal disturbance were very consistent with the presence of these stress-induced lesions.”³⁸ These aforementioned authors concluded that “NCCLs co-exist almost systematically with occlusal wear facets” (94.5% of the sample), and that “lack of canine disclusion was also closely associated with the presence of abfractions” (77.2% of the sample). “Conversely, mobility was seldom found (1.9%)” as a factor.³⁸

The diagrammatic schema of pathodynamic mechanisms causing tooth-surface lesions proposed by Grippo et al²² (Fig 1) enables the researcher, teacher, and clinician to readily identify the causative factors involved in the diagnosis of NCCLs, thus permitting a more accurate classification.

The purpose of this article is to report the findings of a clinical survey conducted among a non-toothbrushing population of subjects with leprosy. The intent of the survey was to identify the presence or absence of NCCLs

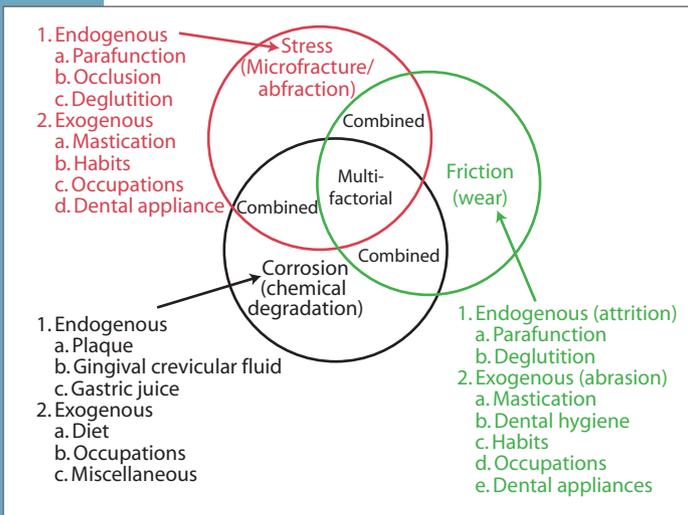


Fig 1 Expanded schema of pathodynamic mechanisms affecting tooth-surface lesions with various etiologic factors. (From Grippo JO, Simring M, Schreiner S. Attrition, abrasion, corrosion, and abfraction revisited: A new perspective on tooth surface lesions. J Am Dent Assoc 2004;135: 1109–1116. Copyright © 2004 American Dental Association. All rights reserved. Reprinted by permission.)

among these subjects and to gather initial demographic and descriptive information to be used for the planning of a clinical trial.

METHOD AND MATERIALS

Data assembled for this cross-sectional study were obtained from an examination of 102 subjects who had leprosy. Subjects from the rural villages of Peykouc and Mballing, Senegal, Africa, were examined by 4 clinicians from the University of Dakar Dental School at the Applied Leprosy Institute of Dakar (APID). The APID clinic provides health services to persons with leprosy, who are treated by the government social welfare agency. All of the subjects living in these isolated rural areas had contracted the disease at a very young age (between 7 and 10 years). Unfortunately, they did not seek medical care until the condition had progressed enough to cause disfigurement, and many became manually handicapped (Fig 2). This lack of care resulted from their primitive, low educational, and depressed socioeconomic status, such that the toothbrush and dentifrice did not become a part of their culture.



Fig 2 Deformed hands frequently found among patients with leprosy.

These conditions assured the fact that the presence of leprosy invariably indicated a non-toothbrushing subject, thus making it difficult to find a toothbrushing control population subset within the population studied.

When examined, the subjects asserted on their word of honor that they had never used a toothbrush, nor a *miswak*, the latter being a wooden stick used by some Muslims to clean their teeth. A total of 52 male and 50 female subjects, with ages ranging from 20 to 77 years, were examined using a mirror and a no. 6 explorer. The clinical parameter of interest for this preliminary study was the presence or absence of NCCLs and their probable etiology. Designation of the various NCCLs by the 4 dental examiners was based on the mechanisms described in the schema by Grippo et al²² (see Fig 1). The lesions were identified during the examination by determining which of the mechanisms—stress, corrosion, or friction—were involved in their etiology. Since a toothbrush and dentifrice had never been used at any time by these subjects, abrasion was not a factor. Occlusal masticatory, deglutitional, and parafunctional stresses were considered to be significant factors in the etiology of the NCCLs.

An inventory of the subjects' diet was generated to determine if there were corrodents in their foods and beverages that might have affected their teeth. The acidic effect of their foods and beverages that could cause corrosion was determined to be a major factor in the etiology of NCCLs. To strengthen the acidic effect concept, a Piccolo pH meter was used to determine the pH of the various drinks consumed by these subjects. The most frequently consumed beverage (by 31 of the subjects) was Bissap, a drink made from indigenous sorrel plants that has a high content of vitamin C. Their second most preferred beverage (by 10 of the subjects) was Coca-Cola. A notation of the subjects' use of medication was necessary since they are dependent on various antibiotics and anti-inflammatory drugs during the regimen of their treatment.

Data were noted but not tested for statistical significance, as the intent of the examinations was to gather initial demographic and descriptive information to be used for planning a more structured clinical trial.

RESULTS

In the 102 leprosy (non-toothbrushing) subjects who were examined, 48 (47%) had NCCLs, which appeared to be related to resultant incisal and occlusal stress (abfractions), as corresponding signs of parafunction were evident. These 48 patients also frequently drank acidic beverages and ate various acidic fruits; thus corrosion was implicated as a cofactor in their etiology, and the NCCLs were designated as combined²² lesions, that is, corrosion/abfractions (Figs 3 to 6). It was found that the pH values of the beverages commonly used by the leprosy subjects were incredibly low, ranging from 0.9 to 5.4, consequently creating an acidic odontolytic environment (Table 2). All of these pH levels were below the 5.5 critical point at which demineralization can occur, as reported by Stephan,³⁹ Gray,⁴⁰ and Zero.⁴¹ It was noted that the subjects who preferred the beverage Bissap (sorrel) had the highest instances (65%) of NCCLs. Although no statistical data were performed, there seemed to

be a significant correlation between the presence of NCCLs and the consumption of these beverages, which can act as corrodents.

Treatment to arrest leprosy takes 6 months, and there exists a standard multi-drug regimen that can vary during the length of the treatment.⁴² It was noted that the most commonly used medications were the antibiotics rifampicin, dapsone, and clofazamine, as well as the anti-inflammatory diclofenac sodium (Voltaren).⁴² One of the side effects of Voltaren is a reduction of salivary flow, which thus is a factor to consider in the formation of NCCLs in these subjects by reducing the buffering effect of saliva and thereby exacerbating the etiologic mechanism of corrosion.

DISCUSSION

Despite all the research and attention that NCCLs have received during the past 30 years, there still remain those who believe that the toothbrush/dentifrice is the main, or only, cause of NCCL etiology. In this preliminary study, a group of subjects with leprosy who had never brushed nor ever used a dentifrice was examined with attention to the presence or absence of NCCLs. According to Bader et al⁴³ and Levitch et al,⁴⁴ there are substantial differences among clinicians in the recognition of NCCLs; however, the schema of pathodynamic mechanisms was used to designate the lesions in these subjects.²² Since incisal attrition caused by parafunction was present, the mechanism of stress was considered as a factor in the etiology of NCCLs noted in the subjects studied. In addition to parafunction, mastication and deglutition as well as the consumption of mangoes, for which the anterior teeth are used to strip the pulp from the large seed, generate additional stress.

Most of the subjects in this study consumed homemade, highly acidic beverages, predominantly Bissap, which may have been cofactors in the etiology of the NCCLs. The consumption of an acidic diet, of both foods and beverages, would provide degrading corrodents; thus, the mechanism of corrosion would have to be acknowledged as a



Fig 3 A well-defined corrosion/abfraction on the maxillary left central incisor and a beginning lesion on the maxillary left canine of a 68-year-old man with leprosy. The lateral incisor may remain unscathed because of the lesser load placed on this tooth during the dynamics of function and parafunction.



Fig 4 Noncarious cervical lesions on the mandibular right canine and central incisor designated as corrosion/abfractions in a 61-year-old man with leprosy. Signs of parafunction from bruxism can be seen on the anterior teeth.



Fig 5 Generalized facial recession and early cervical corrosion/abfractions in a 50-year-old man with leprosy. The stain present on all of the teeth results from chewing kola seeds. Signs of parafunction may be noted on the central and lateral incisors.



Fig 6 A beginning corrosion/abfraction on the mandibular left canine probably caused by stress from parafunction in the presence of a corrodent, in a non-toothbrushing 51-year-old woman with leprosy. Signs of bruxism may be noted in the maxillary central incisors, left lateral, and canine.

Table 2 Preference of beverages (corrodents) in subjects with leprosy with noncarious cervical lesions (n = 48)

Beverage	pH	Absolute value (no. of subjects)	Relative value (percentage of subjects)
Bissap (sorrel)*	1.9	31	65%
Coca-Cola*	2.5	10	21%
Tamarin*	0.9	1	2%
Ginger*	5.4	1	2%
Citrus fruit**	—	5	10%
Total		48	100%

*The subjects who consumed these preferred beverages frequently drank them daily.

**Five subjects occasionally drank these 4 beverages but also consumed citrus fruits such as lemons and mangoes.

cofactor in the etiology of these lesions. Qualifying or quantifying as to which mechanism dominates—whether it be stress manifested as abfractions, or corrosion manifested as chemical degradation—is of no consequence, since factors coexist and act synergistically in the population studied. Suffice it to say that these lesions would have to be a combination of the dual mechanisms of stress (abfraction) and corrosion (acid degradation), since the mechanism of friction (abrasive wear) would be absent in the case of these non-toothbrushers. Therefore, the presence of NCCLs that existed in this non-toothbrushing group of persons with leprosy appears to be related to stress caused by endogenous (parafunction and deglutition) and exogenous (mastication) factors, and to the high degree of corrosion caused by dietary habits (see Figs 3 to 6). The use of the anti-inflammatory drug Voltaren, which causes a decrease in salivary flow, could have further contributed to the formation of these lesions. The common use of antibiotics to treat leprosy was not considered to be involved in the formation of these lesions.

CONCLUSIONS

1. This preliminary report demonstrated that toothbrush/dentifrice use was not a factor in the etiology of NCCLs, which existed in 48 (47%) of 102 Senegalese subjects with Hansen's disease.
2. In the absence of frictional wear, the following mechanisms should be considered as possible etiologies: (1) Stress caused by mastication, deglutition, and parafunction manifested as abfractions and (2) corrosion manifested by chemical degradation could be cofactors in the etiology of NCCLs in all of the 48 subjects.
3. Using the schema of pathodynamic mechanisms, the lesions found in this study were classified as corrosion/abfractions.
4. The mechanism of friction manifested by wear via abrasion was not a factor since these subjects had never brushed their teeth. Furthermore, frictional wear from foods was considered negligible in the for-

mation of these NCCLs but may have been a small contributing factor.

5. The use of medication in reducing salivary flow could possibly have also been a factor in exacerbating these NCCLs. The authors intend to continue gathering additional data and expanding on this investigation.

Further studies with a proper control group and a thorough statistical analysis are needed to identify which of the aforementioned possible etiologies play a significant role in the findings presented in this study.

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REFERENCES

1. McEvoy SA, Mitchell RJ, Powell MO. Wedge-shaped dental lesions in two pre-historic Native American populations. *Am J Phys Anthropol Annual Meeting Issue 1996(suppl)*;162.
2. Miller WD. Experiments and observations on the wasting of tooth tissue variously designated as erosion, abrasion, chemical abrasion, denudation, etc. *Dent Cosmos 1907*;XLIX:1–23.
3. Miller WD. Experiments and observations on the wasting of tooth tissue variously designated as erosion, abrasion, chemical abrasion, denudation, etc. *Dent Cosmos 1907*;XLIX:109–124.
4. Miller WD. Experiments and observations on the wasting of tooth tissue variously designated as erosion, abrasion, chemical abrasion, denudation, etc. *Dent Cosmos 1907*;XLIX:225–247.
5. Korber KH. Die elastische Deformierung menschlicher Zahne. *Dtsch Zahnartzl Z 1962*;17:691–694.

6. Lukas D, Spranger H. Experimentelle Untersuchungen über die Auswirkungen unterschiedlich gemessener Gelenkbahn und Bennetwinkel auf die Horizontalbelastung des Zahnes. *Dtsch Zahnärztl Z* 1973;28:755–758.
7. Haines DJ, Berry DC, Poole DFG. Behavior of tooth enamel under load. *J Dent Res* 1963;42:885–888.
8. Lehman ML, Meyer ML. Relationship of dental caries and stress concentrations in teeth as revealed by photoelastic tests. *J Dent Res* 1966;45:706–714.
9. Thresher RW, Saito GE. The stress analysis of teeth. *J Biomech* 1973;6:443–449.
10. Selna LG, Shillingburg HT, Kerr PA. Finite element analysis of dental structures—Axisymmetric and plane stress idealizations. *J Biomed Mater Res* 1975;9:237–254.
11. Yettram AKM, Wright KWL, Pickard HM. Finite element analysis of the crowns of normal and restored teeth. *J Dent Res* 1976;55:4–11.
12. Goel VK, Khera SC, Ralston JL, Chang KH. Stresses at the dentinoenamel junction of human teeth. A finite element investigation. *J Prosthet Dent* 1991;66:451–459.
13. Geramy A, Sharafoddin F. Abfraction: 3D analysis by means of the finite element method. *Quintessence Int* 2003;34:526–533.
14. Grosskopf G. Untersuchungen zur Entstehung der sogenannten keilförmigen Defekte am oranium dentale. Frankfurt/Main, Germany: Med Diss, 1967.
15. Spranger H, Haim G. Zur Analyse hochfrequenter Schwingungen in der Hartschicht menschlicher Zähne. *Stoma (Heidel)* 1969;22:145.
16. Grippo JO, Masi JV. The role of stress corrosion and piezoelectricity in the formation of root caries. In: Foster KR (ed). *Proceedings of the 13th Annual Northeast Bioengineering Conference, 12–13 Mar 1987, Philadelphia*. Philadelphia: University of Pennsylvania, Philadelphia, 1987:93–95.
17. Grippo JO, Masi JV. The role of bioengineering factors (BEF) in the etiology of root caries. *J Esthet Dent* 1991;3:71–76.
18. Spranger H. Investigation into the genesis of angular lesions at the cervical region of teeth. *Quintessence Int* 1995;26:149–154.
19. Grippo JO, Simring M. Dental erosion revisited. *J Am Dent Assoc* 1995;126:286–290.
20. Whitehead SA, Lo LY, Watts DC, Wilson NHF. Changes of surface texture of enamel in vivo. *J Oral Rehabil* 1997;24:449–453.
21. Davis MW. Factors associated with cervico-abfraction: Literature review and pilot study, with periodontal and restorative considerations. *J Cosmetic Dent* 2002;17:58–76.
22. Grippo JO, Simring M, Schreiner S. Attrition, abrasion, corrosion and abfraction revisited. *J Am Dent Assoc* 2004;135:1109–1118.
23. Palamara D, Palamara JEA, Tyas MJ, Pintado M, Messer HH. Affects of stress on the acid dissolution of enamel. *Dent Mater* 2001;17:109–115.
24. Staninec M, Nalla RK, Hilton JF, et al. Dentin erosion simulation by cantilever beam fatigue and pH change. *J Dent Res* 2005;84:371–375.
25. ten Bruggen Cate HJ. Dental erosion in industry. *Br J Ind Med* 1968;25:249–266.
26. Sangnes G, Gjermo P. Prevalence of oral soft tissue and hard tissue lesions related to mechanical tooth cleansing procedures. *Community Dent Oral Epidemiol* 1976;4:77–83.
27. Bergstrom G, Lavstedt S. An epidemiologic approach to toothbrushing and dental abrasion. *Community Dent Oral Epidemiol* 1979;7:57–64.
28. Bergstrom G, Eliasson S. Cervical abrasion in relation to toothbrushing and periodontal health. *Scand J Dent Res* 1988;96:405–411.
29. Hand JS, Hunt RJ, Reinhardt JW. The prevalence and treatment implications of cervical abrasion in the elderly. *Gerodontics* 1986;2:167–170.
30. Natusch J, Klimm W. Chronischer Zahnsubstanzverlust im frühen and mittleren Erwachsenenalter. *Zahn Mund Kieferheilkd* 1989;77:123–127.
31. Jarvinen VK, Rytömaa II, Heinonen OP. Risk factors in dental erosion. *J Dent Res* 1991;70:942–947.
32. Lussi A, Shaffner M, Holz P, et al. Dental erosion in a population of Swiss adults. *Community Dent Oral Epidemiol* 1991;19:286–290.
33. Padbury AD, Ash M. Abrasion caused by three methods of tooth brushing. *J Periodontol* 1974;45:434–438.
34. Radentz WH, Barnes GP, Cutright DE. A survey of factors possibly associated with cervical abrasion of tooth surfaces. *J Periodontol* 1976;47:148–154.
35. Saxton CA, Cowell CR. Clinical investigation of the effects of dentifrices on dentin wear at the cemento-enamel junction. *J Am Dent Assoc* 1981;102:38–43.
36. Klees L. Contribution clinique et histologique à l'étude des érosions des dents humaines. *Bull Groupe Int Recherche Sci Stomatol Odontol* 1980;23:87–111.
37. Dyer D, Addy M, Newcombe RG. Studies of in vitro abrasion by different manual toothbrush heads and a standard toothpaste. *J Clin Periodontol* 2000;27:99–103.
38. Miller N, Penaud J, Ambrosini P, Bisson-Boutelliez C, Briançon S. Analysis of the etiologic factors and periodontal conditions involved with 309 abfractions. *J Clin Periodontol* 2003;30:828–832.
39. Stephan RM. Changes in the hydrogen-ion concentration on tooth surfaces and in carious lesions. *J Am Dent Assoc* 1940;27:718–723.
40. Gray JA. Kinetics of the dissolution of human dental enamel in acid. *J Dent Res* 1962;41:633–645.
41. Zero DT. Cariology. *Dent Clin North Am* 1999;43:655.
42. The WHO website. Available at: www.who.int/lep/.
43. Bader JD, Levitch LC, Shugars DA, et al. Dentists' classification and treatment of cervical lesions. *J Am Dent Assoc* 1993;124:46–54.
44. Levitch LC, Bader DA, Shugars DA, Heymann HO. Non-carious cervical lesions. *J Dent* 1994;22:195–207.

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