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CLINICAL ARTICLE

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A literature review and hypothesis for the etiologies of cervical and root caries

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Abstract

The presence of endogenous acids from bacteria acting on a suitable substrate combined with sources of exogenous biocorrosives such as exogenous acids and proteolytic enzymes in areas of stress concentration are hypothesized to lead to the development and progression of cervical and root caries (RC). Quantifying the effects of each of the mechanisms (stress and biocorrosion) is a daunting task to investigate since so many factors are involved at various times in the etiology of noncarious cervical lesions (NCCLs), cervical caries (CC), and RC. Frictional action of the tongue has a cleansing effect and lingual serous saliva, which has a high flow rate buffering capacity from bicarbonates seem to account for the paucity of lingual NCCLs, cervical, and RC in these areas of teeth. Future studies are indicated to determine the effects of stress and biocorrosion and their factors in the etiology of CC and RC.

Clinical significance

This manuscript presents hypothetical and literary information that the combined effects of stress concentration and biocorrosion contribute to the formation as well as progression of cervical and root caries.

KEYWORDS
biocorrosion, cervical caries, root caries, stress, stress concentration

1 | HISTORICAL DEVELOPMENT OF CARIES FROM BACTERIA

Following the dawn of microbiology pioneered by Louis Pasteur the first extensive research of bacteria affecting teeth was conducted by W. D. Miller, who studied with Robert Koch a preeminent bacteriologist in Berlin. Pasteur had discovered that bacteria can ferment sugars into lactic acid[1] and another Frenchman Emil Magitot, showed that fermentations of sugars could dissolve teeth in the laboratory.[2] Bacteria had been observed inside carious dentin by Underwood and Miles in 1881, and these researchers also proposed that bacterial acids were necessary for removing the mineral of teeth.[3] With this background, Miller then developed his oral microbiological research. While working in Koch's laboratory he began numerous research projects that introduced biological principles into dentistry. In 1890, he postulated a "chemico-parasitic" origin of caries, which has become the foundation of all modern research in the microbiology of dentistry. His theory contended that caries is caused by acids produced by oral bacteria following fermentation of sugars. In 1891, he published his landmark book "*The Microorganisms of the Human Mouth*," which set forth a new theory regarding the cause of dental caries.[4] His principles of the chemo-parasitic theory have been accepted since that time. Miller believed that no single species of bacteria could cause caries.

Though various bacteria are involved in caries, no specific microorganism has been shown by any researcher to be responsible for RC. More recent examination of the microbiology of carious lesions using 16S rRNA and high-throughput DNA sequencing indicates that colonies of diverse organisms may be more important than individual species such as *Streptococcus mutans* as the primary pathogen, which has been stated.[5] Bowden found in 1990[6] that *Streptococcus sobrinus* in addition to *S. mutans* was also a primary pathogen for RC. *S. sobrinus* produces more acid than other species of mutans streptococci.[7] Newbrun reported that organisms involved in RC are different from those in other smooth surface lesions because the initial lesion is in cementum and dentin, not enamel.[8]

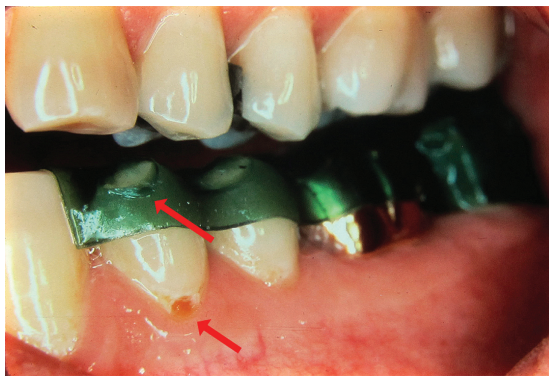

FIGURE 1 An incipient cervical carious lesion at the CEJ on the lower left first bicuspid in a patient who bruxes as is evident by the facets on the upper and lower left cuspids. Stress concentration combined with biocorrosives on #21 at the CEJ can be contributing factors in the genesis of this carious lesion. The eccentric loading areas are depicted by using Kerr's Occlusal Indicator Wax. Kerr Inc. Orange, California

He found from bacteriological investigation that samplings of plaque, now termed biofilm, covering caries of the root surface yielded predominantly *Actinomyces viscosus* and that microbial samplings of softened human dentin from RC also revealed the presence of other species of the genus Actinomyces (viz., *A. naeslundii*, *A. odontolyticus*, *A. eriksonii*) as well as *Rothia dentocariosa*, *Nocardia*, and *S. mutans*.

CC in the enamel is readily observed as dark brown softened areas coronal to the cemento-enamel junction (CEJ) indicating that caries is occurring (Figures 1–3). This is theoretically due to biofilm retention reacting with fermentable carbohydrates combined with stress concentration from occlusal loading forces in these areas.

RC in contradistinction occurs when the root surface is exposed to the oral environment due to gingival recession[8] (Figure 4). It follows the same pattern as coronal caries with biofilm composed primarily of *S. mutans* followed by *Lactobacillus acidophilus*. However, increased *Actinomyces israeli* salivary counts have been significantly correlated with RC occurrence as the area becomes more acidic. As

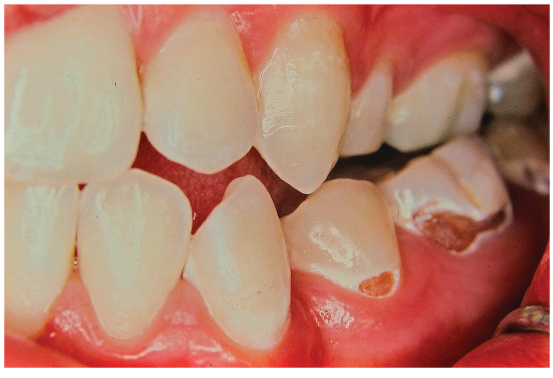

FIGURE 2 CC in #19 and #20 entirely with the coronal of these teeth with areas of demineralization (as white) surrounding the dark brown carious lesions. Caries, which is the biocorrodent, is most advanced in these areas of potential stress concentration

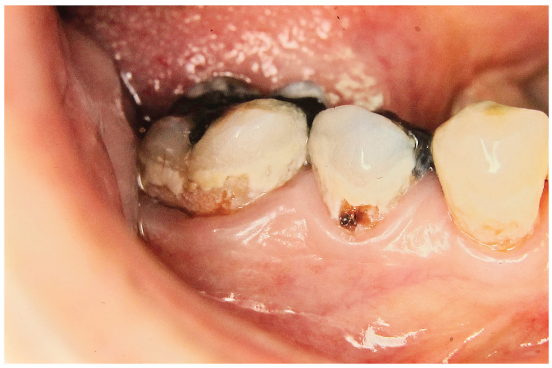

FIGURE 3 CC on #29 and #30 with areas of demineralization more occlusal to the lesions. Tooth # 28 is missing while tooth #27 has a composite restoration which is abfracting from the loading force as noted by the wear facet on the incisal

cited by Shen et al.[9] and Bignozzi et al.,[10] they reported scanning electron microscopy observations revealed various patterns of bacterial coaggregation in RC lesions. Especially in cases of gingival recession, the root surface becomes more exposed, increasing the likelihood of developing RC.[10] Synergistic growth was observed in cocultures of *L. acidophilus* with *S. mutans* and *A. israeli* as well as *L. acidophilus*, producing greater acidogenic and cariogenic effects on the root surface. Although *Candida albicans* has been identified in soft root surface lesions, it is not considered an etiologic factor in the onset of RC.[11,12] Decrease in salivary flow due to various medications which promote xerostomia, cariogenic diet coupled with poor oral hygiene all become risk factors in producing RC. Chronic medical conditions such as Dementia, Alzheimer's, Parkinson's, and radiation treatment to the head and neck also are risk factors in RC.[13] It has been shown that liquid sucrose and glucose frequently ingested will cause the numbers of a cariogenic strains to tremendously multiply.[14] Stephen[15] demonstrated that when sucrose comes into contact with dental biofilm, acid is produced within 20 seconds and may last up to 30 minutes before it is buffered by the saliva.

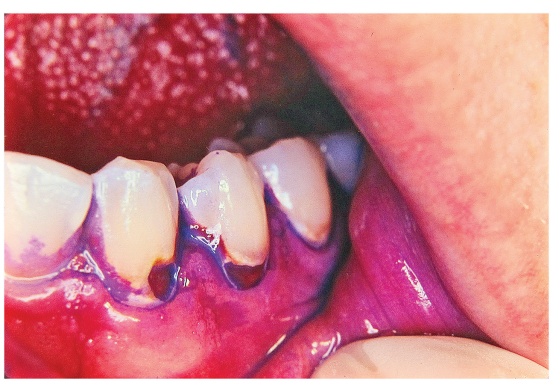

FIGURE 4 Advanced RC as depicted in # 21 and #22 by using disclosing solution to expose the dental plaque. The disclosing solution was Plaque Finder 2-Tone by Pro-Dentec, Batesville, AR

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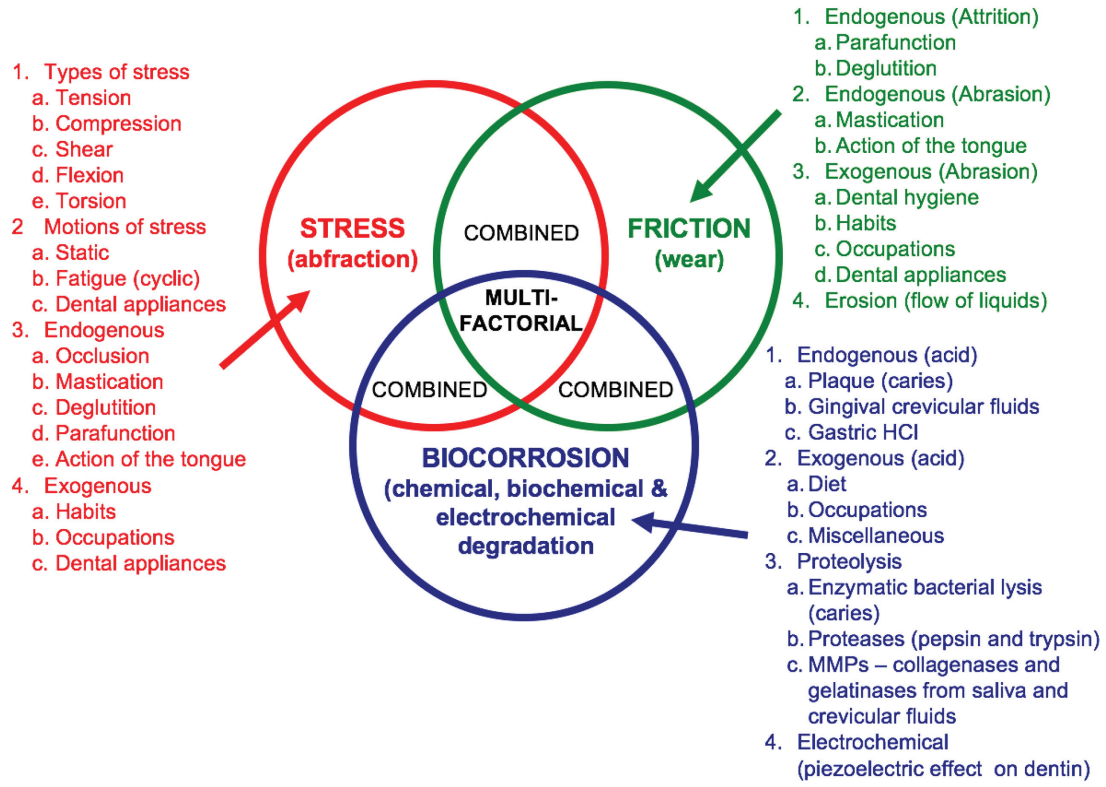

FIGURE 5 Schema of Pathodynamic Mechanisms. The current 2017 amended Schema indicates the combination of Stress and Biocorrosion mechanisms which appear to contribute to cervical and RC. This Schema indicates the initiating and perpetuating etiological factors that could form these lesions. Courtesy of John O. Grippo and Dan S. Oh

2 | MECHANISMS OF STRESS CONCENTRATION AND BIOCORROSIVE EFFECTS TO TEETH, WHICH APPEAR TO CONTRIBUTE TO CERVICAL AND ROOT CARIES (RC)

Over time, teeth experience various stresses in the mouth, namely, tension, compression, shear, flexion, and torsion when combined with bacterial and other biocorrosives form cervical and RC.[16–19] CC and RC appear to have multifactorial etiologic factors related to stress and biocorrosive agents proposed by the recently amended 2017 Venn diagram (Figure 5). A 5-year study by Sawlani et al. investigating noncarious cervical lesions (NCCLs) found that their initiation and progression was strongly related to increased occlusal force, which produces CEJ stress.[20] NCCLs will potentially become CC or RC if bacteria and proteases from the biofilm as well as proteases from saliva in these lesions were not removed. If bone is resistant then caries will occur in the region(s) of greatest stress concentration rather than on all exposed root surfaces.

Cervical and RC have increasingly become an issue for the elder population as there have been countless studies, which support that those in the older age spectrum are much more susceptible to RC lesions.[13,21] RC is a major oral health problem that has been referred to as cemental, cervical, radicular, and senile caries as a soft, progressive lesion affecting cementum and dentin of the root.[22] Advances in dental science and improved systemic health care have increased our lifespan allowing us to retain more of our teeth as we age.[23]

As stated by Banting tissues, namely, enamel, cementum, and dentin: "Under normal circumstances, this loss of calcium (demineralization) is compensated for by uptake of calcium (remineralization) from the tooth's microenvironment."[24] The dynamic process of demineralization and remineralization takes place continually and equally in a favorable oral environment.[25] In an unfavorable environment, the remineralization rate does not sufficiently neutralize the rate of demineralization, thus caries will occur. Wefel et al.[26] stated: "The dental caries process begins with loss of calcium ions from the surface apatite crystals that form the bulk of the calcified dental structures and for RC this process involves the cementum first although in some cases it begins in exposed dentin. There is both loss of mineral and protein degradation (proteolysis) in RC. Remineralization involving dentin has been shown to take place on the remaining mineral content rather than on the protein infrastructure." Banting continues to say that RC, by definition, occurs on the root of the tooth.[24] Some investigators have made a distinction between RC that originates entirely on the root surface and caries that spreads from the coronal region onto the root surface. Primary RC refers to new dental caries occurring in the absence of a restoration. Secondary or "recurrent" RC, refers to caries occurring adjacent to an existing restoration. The authors report that there are conflicting opinions in the literature about RC in the area of the CEJ, as to whether or not to classify caries in this area of the CEJ as RC extending onto the crown, or as coronal caries extending onto the root. It is suggested by the authors to designate these lesions as "cervical caries (CC)." The location of RC has been associated with age and

gingival recession. It occurs predominantly on the proximal (mesial and distal), followed by the facial surfaces.[27,28] Heegaard et al.[29] in a more recent study reported that RC are frequently found on labial surfaces and evenly distributed within the dentition. RC are most commonly located on exposed root surfaces; however, it has been reported that up to 10–20% of these lesions may occur subgingivally.[30]

Kawasaki and Featherstone[31] investigated collagenase activity during tooth root demineralization and remineralization in an "in vitro" demineralization/remineralization pH–cycling model. Human tooth roots were subjected to pH cycling (alternating demineralization and remineralization). The principle was clearly established that partial demineralization of the mineral crystals is first needed, followed by enzymatic breakdown of the collagen even at a neutral pH. Their study has clarified the role of collagenase in experimental RC in the complicated balance between demineralization and remineralization. Loss of mineral by acid dissolution was shown to be necessary prior to proteolytic destruction of collagen, which occurred even in the presence of a mineralizing solution at pH 7.0.

Tjaderhane et al. demonstrated for the first time in 1998 the connection of host Matrix metalloproteinases (MMPs) to the progression of dental caries in human teeth.[32] MMPs are a family of enzymes which in concert, are capable of degrading collagen in dentin. They stated: "The activation of the latent enzymes at pH 4.5 followed by neutralization proves that MMP activity is greatly enhanced in the conditions present in carious lesions." Alternating periods between dentin demineralization in a pH below 5.5 and periods of neutral pH due to salivary buffers provide the sequence in which the collagen fibers of dentin organic matrix are first exposed and then degraded by MMPs.

Saliva contains several MMPs including collagenases and gelatinases originating from either the secretion of the salivary glands or gingival crevicular fluid, MMP-9 being the greater concentration is derived from both sources.[33] As saliva bathes carious lesions, the active form of MMP-9 was systematically detected by zymography, which electrophoretically measures the proteolytic activity in dentin extracted from carious teeth.[34]

The purpose in discussing the mechanisms involved the etiology of cervical and RC is that there appears to be a close relationship of these lesions to NCCLs in that stress concentration and biocorrosion are commonly present. Heretofore, most research has focused on the microbiota, which cause cervical and RC but the role of stress concentration working in concert with bacterial biocorrosion has not been investigated. Unfortunately, neither can "in vivo" nor "in vitro" investigations accurately reflect the numerous variables that occur clinically. Variables include amounts of stress, vectors of force, types/amounts/frequency of dietary biocorrosives, which are consumed, and systemic influences such as gastric reflux conditions.

Studies have been reported on the history, incidence, prevalence, diagnosis, and treatment of RC. Leake[35] in 2001 searched the electronic databases related to his study and produced 807 references at that time. If plaque is present in these areas and a fermentable carbohydrate is present then caries could occur and is accelerated by stress concentration in these areas. As the lesions progress more deeply into the tooth stress concentration becomes greater thus causing an

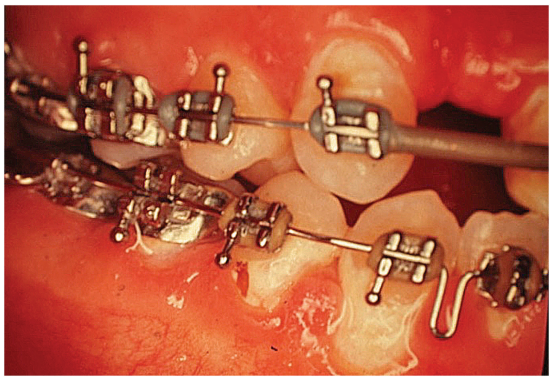

FIGURE 6 Cervical acidic biocorrosion depicted as demineralization on the labial of tooth # 6 and advanced biocorrosion on #27 and #28. Bacterial biocorrosion (CC) is on tooth #28. Both the demineralized areas and carious lesion were accelerated by stress concentration in the cervical region caused by forces from the orthodontic appliances. They were also exacerbated by poor oral hygiene and diet. Courtesy of Robert Angorn, DMD, Melrose, MA

acceleration of caries. The concept of stress concentration was first demonstrated and seldom cited in the literature by Lehman and Meyer since 1966.[36] At that time they stated that: "Problems of initiation and propagation of early caries lesions has been investigated, however their photoelastic tests have shown that biomechanical stresses in teeth are contributory factors. These areas occur at the contact points between adjacent teeth or between natural teeth and parts of artificial appliances like clasps. Spread of caries along the enamelo-dentinal junction has also been studied in relation to the stresses occurring in this region due to occlusal loading or from clasps."[36] Rapid progression of cervical and RC being essentially dentinal caries is undocumented; however, it seems likely that stress concentration would account for their rapid progression. This would occur during the constant recurrence of cyclic loading during deglutition and eating, in addition to parafunction, whenever biofilm is present in these areas and oral hygiene is lacking. However, if good oral hygiene is maintained then these areas become considered as noncarious and become NCCLs over time. A good example of the effects of eccentric loading contributing to cervical and RC is depicted in Figure 1. Advanced RC due to biocorrosion from endogenous acid in biofilm including various proteolytic enzymes in areas of stress concentration is illustrated in Figure 4. A typical response to the effects of stress and various biocorrosives causing both cervical demineralization and CC can be noted in an orthodontic case as shown in Figure 6.

Piezoelectric effects only to the susceptible cementum and dentin can also be a factor in the genesis of cervical and RC.[37]

A most important factor to be considered in the location and etiology of cervical and RC, as well as NCCLs is the modifying effect of the flow rate, buffering capacity, pH, viscosity, and composition of saliva.[38] The role of saliva in caries protection can be summarized under four aspects: (1) diluting and eliminating sugars and other substances; (2) buffer capacity; (3) balancing demineralization/remineralization; (4) antimicrobial action. Llena-Puy[38] stated that there is a correlation

between pH changes in the plaque and sugar clearance from saliva. Furthermore, pH recovery is not the same on all tooth surfaces.

Although saliva plays an important role in balancing the pH, it also contains specific buffering components such as phosphates, bicarbonates with proteins emanating from the salivary glands.[39] Phosphate buffer plays an essential role when salivary flow is low. Buffering mechanisms do not act equally on all tooth surfaces. Their effect is less on interproximal surfaces and greater on the free surfaces which are covered by a thin layer of bacteria.[40,41]

Israel Kleinberg, a preeminent emeritus scholar of saliva, (SUNY Stony Brook of NY, personal communication with JOG, 2006) avows that there is five times more saliva on the lingual surfaces than in the vestibule. That observation was first reported by Jenkins, et al.[42] These reputable sources of information support the contention that saliva, particularly lingual serous saliva, which has a high flow rate and buffering capacity from bicarbonates accounts for the paucity of lingual NCCLs, cervical, and RC on these areas of teeth. Furthermore, the frictional action of the tongue also assists in cleansing the lingual surfaces.

3 | SUMMATION

In summation, little absolute direct evidence currently exists to fully explain the interaction of the various co-variables that exist with regards to the formation of root and CC. However, in light of the many co-related studies that appear in the literature regarding the multiple potential contributing factors of stress and biocorrosion for the etiology of NCCLs, it appears that there is a strong likelihood that the "missing link" to the initiation and rapid progression of cervical and RC could well involve these same two mechanisms. Enzymatic breakdown of CC and RC by proteolysis and stress concentration working in concert with the biocorrosive effects of bacterial endogenous as well as exogenous acidic agents suggests that these factors may all contribute to these types of caries. Future research is needed to fully confirm or refute the proposed mechanisms of stress and biocorrosion with their various related factors in the etiology of CC and RC.

DISCLOSURE

The authors do not have any financial interest in the company whose product is mentioned in this article.

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REFERENCES

[1] Pasteur L. The Physiological Theory of Fermentation. University of Adelaide, 1870.

[2] Magitot E. Treatise on Dental Caries. Houghton, MA, USA: Osgood and Company Boston; 1878.

[3] Underwood, Miles. The British Journal of Dental Science. Vol. 43. London: Oxford House. January–December 1905:3.

[4] Miller WD. The human mouth as a focus of infection. *Dent Cosmos*. 1891;33:689–913.

[5] Gross EM, Beall CJ, Kutsch SR, et al. Beyond *Streptococcus Mutans*: dental caries onset linked to multiple species by 16 S.rRNA community analysis. *PLoS One*, 2012;7(10):e47722. https://doi.org/10.137/journal.pone.0047722

[6] Bowden GH. Microbiology of root surface caries in humans. *J Dent Res*. 1990;69:205–210.

[7] De Soet JJ, Toor FA, de Graff J. Acidogenesis by oral streptococci at different pH values. *Caries Res*. 1989;23:14–17.

[8] Newbrun E. Cariology. 3rd ed. Chicago, IL: Quintessence International Publishing Inc.; 1989.

[9] Shen S, Samaranayake LP, Yip HK. In vitro growth, acidogenicity and cariogenicity of predominant human root caries flora. *J Dent*. 2004;32:667–668.

[10] Bignozzi I, Crea A, Capri D, et al. Root caries: a periodontal perspective. *J Periodont Res*. 2014;49:143–163.

[11] Powell LV, Leroux BG, Persson RE, Kiyak HA. Factors associated with caries incidence in an elderly population. *Commun Dent Oral Epidemiol*. 1998;26:170–176.

[12] Fure S. Ten-year cross-sectional and incidence study of coronal and root caries and some related factors in elderly Swedish individuals. *Gerodontology*, 2004;21:130–140.

[13] Bansal V, Sohi RK, Verresha KL, et al. Root caries: a problem of growing age. *J Indian Acad Dent Spec Res*. 2011;2(2):43–45.

[14] Carlsson J, Sundstrom B. Variation in composition of early dental plaque following the ingestion of sucrose and glucose. *Odont Rev*. 1968;12:1297.

[15] Stephen RM. Changes in hydrogen ion concentration on tooth surfaces and in carious lesions. *J Am Dent Assoc*. 1940;27:718.

[16] 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;24:10–13.

[17] Lee WC, Eakle WS. Possible role of tensile stress in the etiology of cervical erosive lesions of teeth. *J Prosthet Dent*. 1984;52(3):374–380.

[18] Braem M, Lambrechts P, Vanherle G. Stress-induced cervical lesions. *J Prosthet Dent*. 1992;67(5):718–722.

[19] Lee HE, Lin CL, Wang CH, et al. Stresses at the cervical lesion of maxillary premolar–a finite element investigation. *J Dent*. 2002;30 (7–8):283–290.

[20] Sawlani K, Lawson NC, Burgess JO, et al. Factors influencing the progression of noncarious cervical lesions: a 5-year prospective clinical evaluation. *J Prosthet Dent*. 2016;115:571–577.

[21] Hazen SP, Chilton NW, Mumma RD. The problem of root caries: review and clinical description. *J Am Dent Assoc*. 1973;86:137–144.

[22] Newman E, Armitage G, Daniels TE, et al. Root Caries. *Calif Dent J*. 1984;12:68–73.

[23] Griffin SO, Griffin PM, Swann JL, Zlobin N. Estimating rates of new root caries in older adults. *J Dent Res*. 2004;83(8):634–638.

[24] Banting DW. The Diagnosis of Root Caries. *J Dent Educ*. 2001;65 (10):991–996.

[25] Koulourides T. Dynamics of biologic mineralization applied to dental caries. In: L. Menker, ed. The biologic basis of dental caries. New York: Harper Rowe Publishers; 1980.

[26] Wefel JS, Clarkson BH, Heilman JR. Natural root caries: a histology and microradiographic evaluation. *J Oral Pathol*. 1985;14(8):615–623.

[27] Shacken MJ, Keljtjens HM, van der Hoeven JS. Effects of fluoride and chlorhexidine on the microflora of dental root surfaces and progression of root- surface caries. *J Dent Res*. 1991;70(2):150–153.

[28] Fure S. Five–year incidence of coronal and root caries in 60-, 70-, and 80- year–old Swedish individuals. *Caries Res.* 1997;31:249–258.

[29] Heegaard KM, Holm-Pedersen P, Bardow A, et al. The Copenhagen Oral Health Senior Cohort: design, population and dental health. *Gerodontology*, 2011;28:165–176.

[30] Stamm JW, Banting DW, Imrey PB. Adult root caries survey of two similar communities with contrasting natural water fluoride levels. *J Am Dent Assoc.* 1990;120:143–149.

[31] Kawasaki K, Featherstone JD. Effects of Collagnease on root demineralization. *J Dent Res.* 1997;76(1):588–595.

[32] Tjaderhane L, Larjava H, Sorsa T, et al. The activation and function of host matrix metalloproteinases in dentin matrix breakdown in caries lesions. *J Dent Res.* 1998;77(8):1622–1629.

[33] Ingman T, Sorsa T, Lindy O, et al. Multiple forms of gelatinases/type 1V collagenases in saliva and gingival crevicular fluid of periodontitis patients. *J Clin Periodontol.* 1994;21:26–31.

[34] Sorsa T, Ding YL, Ingman T, et al. Cellular source, activation and inhibition of dental plaque collagenase. *J Clin Periodontol.* 1995;22:709–717.

[35] Leake JL. Clinical decision making for caries management in root surfaces. *J Dent Educ.* 2001;10:1147–1153.

[36] Lehman ML, Meyer ML. Relationship of dental caries and stress: concentrations in teeth as revealed by photoelastic test. *J Dent Res.* 1966;45:1706–1714.

[37] Marino AA, Gross BD. Piezoelectricity in cementum, dentine and bone. *Arch Biol.* 1989;4(7):7–9.

[38] Llena-Puy C. The rôle of saliva in maintaining oral health and as an aid to diagnosis. *Med Oral Patol Oral Cir Bucal.* 2006;11(5):E449–455. PMID: 16878065

[39] Nauntofte B, Tenevuo JO, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O, Kidd E, eds. Dental caries. The disease and its clinical management. Oxford. Blackwell Munksgard; 2003:7–29.

[40] Axelson P. Internal modifying factors in dental caries. In: Axelson P, ed. Diagnosis and caries risk prediction of dental caries. Vol. 2. Chicago: Quintessence Publishing; 2000:91–150.

[41] Tenovuo J. Salivary parameters of relevance for assessing caries activity in individuals and populations. *Commun Dent Oral Epidemiol.* 1997;25(1):82–6. Review. PMID: 9088696.

[42] Jenkins GN. Salivary effects on plaque pH. In: Kleinberg I, Ellison SA, Mandel ID, eds. Saliva and dental caries. New York, NY: Information Retrieval, Inc.; 1979 (Microbiology Abstracts):307–322.

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