

Active White Enamel Lesion: a Case-control Study

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Purpose: To assess the risk factors involved for active white enamel lesion in public schoolchildren, aged 7 to 12 years, in Natal, Brazil, using a case-control study.

Materials and Methods: A total of 273 cases of active white enamel lesion and 325 controls (lesion-free) were matched by age, gender and school. The independent variables were: age; gender; visible plaque index (VPI); gingival bleeding index (GBI); decayed, missing and filled surfaces (DMFS); decayed, extracted or filled surfaces (dmfs); DMFS-dmfs; and the number of carious surfaces.

Results: There was no statistical significance for age ($p = 0.57$), gender ($p = 0.428$) and dmfs ($p = 0.06$). Univariate analysis revealed an increased risk of developing active white enamel lesions in individuals with high VPI (OR = 15.5, CI 95% 10.35–23.2), GBI (OR = 2.86, CI 95% 2.05–3.99), DMFS (OR = 18.91, CI 95% 12.51–28.59), DMFS-dmfs (OR 10.22, CI 95% 7.01–14.91) and number of carious surfaces (OR = 5.47, CI 95% 3.85–7.78). Logistic regression analysis identified that GBI (OR = 2.14, CI 95% 1.41–3.25) and DMFS (OR = 17.3, CI 95% 11.39–26.27) were independent risk factors for active white lesions.

Conclusions: The results reaffirm the importance of mechanically controlling biofilm to prevent caries development in its initial stage (white lesion).

Key words: case-control study, dental caries, enamel demineralisation

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Curative and restorative practice has increasingly been replaced by preventive and educational measures, which play a fundamental role in the control and treatment of oral diseases. Despite changes in the population profile, dental caries still affects the Brazilian population, mainly at the lower socio-economic levels.

Cariou lesions in their initial stages, as well as the risk factors that contribute to their development, have assumed particular importance, since they are cur-

rently more prevalent than cavitated lesions (Pereira and Mialhe, 2003). Therefore better understanding of initial caries, represented by white spot lesions, is required. This will enable better intervention by the professional, thus avoiding lesion progression and the use of invasive techniques.

Clinically visible carious lesions are a product of an imbalance between tooth mineral and biofilm fluids over a determinate period of time, resulting in mineral loss. Constant imbalance, due to cariogenic biofilm and the formation of acid by microorganisms, causes opaque and rough-surfaced active white lesions. Increased porosity is clinically visible when the surface is dry because part of the aqueous material of the intercrystalline spaces is replaced by air. The different refractive index results in loss of normal translucency and an opaque surface (Thylstrup, 1998; Kidd and Fejerskov, 2004).

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Oral hygiene has often been assessed as a risk factor for carious lesion development (Ogaard et al, 1994; Mascarenhas, 1998; Bedos and Brodeur, 2000; Harris et al, 2004). In the majority of cases, the measure of oral hygiene is based on the presence of surface biofilm and gingival bleeding indicators. Gingival bleeding demonstrates mature and accumulated biofilm and deserves attention as a caries predictor (Mathiesen et al, 1996; Ekstrand et al, 1998; Ratledge et al, 2001). In addition to oral hygiene, Johnson (2004) emphasises decayed, missing and filled surfaces (DMFS) as an important predictor of caries activity, related to the development of new caries.

Early action against carious lesions in the initial stage requires the identification of individuals or groups considered a priority in oral health programmes. The aim of the present study was to investigate risk factors for active white enamel lesions.

MATERIALS AND METHODS

Participants

The present study was performed in 2005 following a clinical trial (Ferreira et al, 2005) children aged 7 to 12 years of age in Natal, Brazil. The children were selected from 25 schools. Their families had mean monthly incomes of US\$ 290. The project was approved by the Federal University of Rio Grande do Norte (UFRN) Research Ethics Committee and the parents or guardians signed an informed consent form.

Design

The design was a case-control study whose control group participants were matched with the case group by school, sex and age. The case group (with white lesions on the permanent upper incisors) was composed of 273 children and the control (lesion free) of 325.

Data collection

The data were collected by calibrated examiners by means of an intraoral examination of dental caries, oral hygiene and gingival condition. A tactile-visual examination was performed using a mouth mirror and dental probe under natural light in the school environment. Of the children, 5% were reexamined to measure intra-examiner agreement in relation to the diagnosis of white lesion activity, with a Kappa value of 0.62 (Fer-

reira et al, 2005). Oral hygiene was measured using the visible plaque index (VPI) and gingival bleeding index (GBI) (Ainamo and Bay, 1975), while for dental caries DMFS and dmfs (decayed, extracted or filled surfaces) were used for permanent and deciduous teeth respectively (World Health Organization, 1997). The carious component (C) was recorded using C₁ for caries with enamel and dentin cavitation and C₂ for active white lesions (Kingman and Selwitz, 1997). The index of agreement for DMFS was 0.82.

Statistical analysis

Data for quantitative variables were presented as mean, median, quartiles and standard deviation. The categorisation of continuous variables was based on the median, and the chi-square test was subsequently applied to verify the association between the independent variables and the presence and absence of white lesions. Odds ratio (OR) measured estimated risk. Finally, an analysis of stepwise forward multiple logistic regression was performed to identify the liquid effect of clinical and demographic variables. Those with $p < 0.20$ in the association test were included in the model. Hosmer-Lemeshow's test was used to verify adjustments to the multiple logistic regression model (the larger the p-value, the more adjusted the model).

RESULTS

The characteristics of the sample demonstrate that the schoolchildren exhibited poor oral hygiene, with a VPI around 30% and a high mean of surface caries, as shown in Table 1.

There was no statistical significance for age ($p = 0.566$), gender ($p = 0.458$) and dmfs ($p = 0.07$) (Table 2).

The high biofilm and bleeding indices were considered risk factors for active white enamel lesion development. OR values were 15.5 for VPI and 2.86 for GBI at a 95% confidence level.

DMFS (OR = 18.91, CI 95% 12.51–28.59) and DMFS-dmfs (OR = 10.22, CI 95% 7.01–14.91) indices as well as the number of carious surfaces (OR = 5.47, CI 95% 3.85–7.78) were also greater in the case group, exhibiting a highly significant difference ($p < 0.0001$).

Through the process of stepwise forward modelling using a variety of statistical combinations, the most adjusted model was represented by the independent variables GBI and DMFS, which are considered inde-

Table 1 Mean, median, quartiles and standard deviation of continuous variables related to caries and oral hygiene factors

Variable	Mean	Median	SD	Q 25	Q 75
Age	9.58	9	1.51	8	11
VPI	27.29	21.42	20.50	11.11	40.38
GBI	10.83	8.33	9.87	4.16	15.21
DMFS	5.35	3	7.33	0	8
dmfs	5.67	3	7.24	0	8
DMFS-dmfs	10.42	8	11.34	3	15
No. of carious surfaces	8.15	5	10.83	1	2

Table 2 Distribution of the number and percentage of students with and without white spots, according to independent variables

Variable	Category	White spots				Total		OR	CI	p*
		Present		Absent						
		n	%	n	%	n	%			
Age	7–9 year	141	51.6	160	49.2	301	50.3	1.10	0.79–1.52	0.566
	10–12 years	132	48.4	165	50.8	297	49.7			
Gender	M	118	43.2	151	46.5	269	45.0	0.88	0.63–1.21	0.458
	F	155	56.8	174	53.5	329	55.0			
dmfs	Low	89	43.6	147	52.3	236	48.7	0.70	0.49–1.01	0.07
	High	115	56.4	134	47.7	249	51.3			
PVI	Low	49	17.9	251	77.2	300	50.2	15.5	10.35–23.20	<0.0001
	High	224	82.1	74	22.8	298	49.8			
GBI	Low	102	37.4	205	63.1	307	51.3	2.86	2.05–3.99	<0.0001
	High	171	62.6	120	36.9	291	48.7			
DMFS	Low	50	18.3	263	80.9	313	52.3	18.91	12.51–28.59	<0.0001
	High	223	81.7	62	19.1	285	47.7			
DMFS-dmfs	Low	68	24.9	251	77.2	319	53.3	10.22	7.01–14.91	<0.0001
	High	205	75.1	74	22.8	279	46.7			
No. of carious surfaces	Low	82	30.0	228	70.2	310	51.8	5.47	3.85–7.78	<0.0001
	High	191	70.0	97	29.8	288	48.2			
*Chi-square tests										

*Chi-square tests

Table 3 Result of the final multiple logistic regression model

Variable	OR overall	CI (OR overall)	p	Adjusted OR	CI (adjusted OR)	p*
GBI	2.86	2.05-3.99	<0.0001	2.14	1.41-3.25	<0.001
DMFS	18.91	12.51-28.59	<0.0001	17.30	11.39-26.27	<0.001

*Hosmer and Lemeshow test: p = 0.534

pendent potential risk factors for developing active white enamel lesions (Table 3).

DISCUSSION

Few studies have been conducted identifying separately the factors associated with enamel and dentin caries. The majority follow the criteria recommended by the World Health Organization (WHO) and the National Institute of Dental Research (NIDR), which only measure the cavitated lesion, disregarding incipient or non-cavitated lesions. Generally speaking, studies using sectional designs have found that oral hygiene has a strong association with dental caries (Schröder and Granath, 1983; Russel, 1991; Bedos and Brodeur, 2000; Santos and Soviero, 2002).

In children between 6 and 12 years of age, Retnakumari (1999) reported that those with poor oral hygiene had a higher risk of developing caries (OR = 3.59, CI 95% 2.53–5.06). Similar results were found by Kleemola-Kujala and Räsänen (1982) in children aged 5, 9 and 13 years, where the relationship between accumulated biofilm and the presence of caries was also identified. High sugar consumption only represented a significant risk factor when associated with poor oral hygiene.

When assessing enamel and dentin caries separately, Mascarenhas (1998) observed that poor oral hygiene was a common risk factor for both, with a probability of 2.02 (1.46–2.83) for enamel lesions and 3.21 (2.31–4.50) for dentine lesions.

In preschool children, cross-sectional studies also show a relationship between oral hygiene and dental caries (Santos and Soviero, 2002; Jose and King, 2003; Segovia-Villanueva et al, 2005). When active white lesions are included in caries investigations (Schröder and Granath, 1983), the presence of biofilm stands out as the most important factor for developing early childhood caries. This association in preschool children has also been demonstrated by longitudinal studies (Schröder and Granath, 1983; Stecksén-Blicks and Gustafsson, 1986).

In the present study, even though all the school-children displayed poor oral hygiene, the group with active enamel lesions exhibited significantly higher ($p < 0.001$) levels of plaque and gingival bleeding indices. It is important to note that only enamel caries, disregarded by most studies, were assessed. Moreover, the study design was case-control, one of the most effective in assessing risk factors.

Longitudinal studies require a follow-up for a determinate period of time and therefore enable a better ob-

servation of the dynamism between biofilm frequency and caries development, making these studies very important for assessing risk factors. Alaluusua and Malmivirta (1994) found that visible plaque on the vestibular surfaces of maxillary incisors represented the best risk indicator, with a sensitivity of 83% and specificity of 92%, demonstrating that visible plaque on this surface is a risk factor for developing caries.

Similarly, Tucker et al (1976), after a 3-year follow-up, reported a relationship between oral hygiene and toothbrushing frequency and caries incidence in 11-year-old children. The authors concluded that the increase in caries was less in the group with good hygiene that brushed with greater frequency. Toothbrushing frequency, however, has been decreasing as a variable since it does not reflect hygienic conditions, given that it may be ineffective in biofilm removal.

In contrast, Etty et al (1994) concluded that white lesion cavitation did not alter as a result of poor oral hygiene. It should be noted, however, that the five groups investigated were submitted to fluoride intervention, which may have masked the real effect of oral hygiene. Although a large proportion of studies have shown a positive association between poor oral hygiene and caries, some have not reported this relationship, probably owing to unsuitable methodology (Parviainen et al, 1977; Ainamo and Parviainen, 1979; Klock et al, 1989; Etty et al, 1994).

In addition to the methodological differences among studies, another important aspect to be assessed is the data-collection instrument. Questionnaires about toothbrushing frequency (Jose and King, 2003; Segovia-Villanueva et al, 2005) and bleeding during the act of brushing (Tubert-Jeannin et al, 1994) have been used as verification instruments, demonstrating a positive association between dental caries and poor hygiene. Vanobbergen et al (2001), for example, found that brushing less than once a day was a risk factor (OR = 2.43). However, Petry et al (2000) observed in a case-control study that oral hygiene did not influence the presence of caries in adults.

Studies that base themselves on questionnaires to identify oral hygiene levels must be assessed with caution, given the unreliability of verifying the quality of toothbrushing and the presence of biofilm or gingival inflammation without performing a clinical examination. The quality of brushing is more important than the frequency.

A more reliable collection instrument for identifying oral hygiene as a risk factor is the presence of biofilm and bleeding, which were obtained in our study from VPI and GBI. These indices were highly significant in the white lesion group ($p < 0.0001$).

Gingival bleeding is an important predictor of oral hygiene since it is a result of mature biofilm. Investigations have considered the gingival bleeding index as a significant factor for increased caries (Ogaard et al, 1994; Mathiesen et al, 1996). In a longitudinal study with children aged 8 and 13 years in Sweden, Steck-sén-Blicks and Gustafsson (1986) assessed GBI percentage as an objective measure of oral hygiene and found a strong relationship with high caries activity. The authors further affirmed GBI as the most significant variable, allowing differentiation between the group with low caries activity and that with high activity.

When assessing bleeding associated to proximal caries, Ratledge et al (2001) observed a significant relationship: the group with cavitated lesions exhibited a higher gingival bleeding index than the lesion-free group. Proximal caries progression was also investigated in a study by Ekstrand et al (1998), who verified the influence of plaque and bleeding indices as parameters in the development of enamel and dentine caries. The results demonstrated that there was a significant relationship between gingival bleeding and carious surfaces, and highlighted the fact that biofilm occurrence ($p = 0.5$) did not display the same predictive power as bleeding ($p < 0.001$)

Schröder and Granath (1983) used bleeding as a hygiene parameter in preschool children with mean age of 3 years, and concluded that those with no clinical signs of gingival inflammation had a low risk of developing caries. In a longitudinal study, Wendt et al (1994) used visible plaque as hygiene parameter, which was significantly correlated with the development of carious lesions in children from 1 to 3 years of age.

Another factor to consider is the possibility of caries experience influencing the development of new lesions. Poulsen and Holm (1980) and Raadal and Espelid (1992) reported that the presence of caries in deciduous teeth increases the likelihood of caries in permanent teeth. Vanobbergen et al (2001), however, showed in a cross-sectional study that dmfs, with an odds ratio of 1.07, was not an important risk factor for caries in first permanent molars. In the present study, the dmfs of deciduous teeth was not a risk factor for active white lesions.

A 3-year cohort study on permanent teeth found initial DMFT as a risk factor for caries development, represented by increased DMFT after a period of time (Almagro-Nievas et al, 2001). Similarly, Russel (1991) observed that past caries experience (DMFS) and the presence of carious surfaces were important predictive factors for the subsequent increase in carious lesions.

In the present study DMFS was highly significant, with an adjusted OR of 17.3 (11.39–26.27). This demonstrates its importance in identifying groups at risk of developing new carious lesions, despite the fact that DMFS represents accumulated caries experience and not necessarily the current condition of the individual.

CONCLUSIONS

Poor oral hygiene is a risk factor for the presence of active white lesions. Caries experience, measured by DMFS proved to be an important indicator for identifying individuals at risk of developing active white enamel lesions. The results reaffirm the importance of mechanical biofilm control in preventing caries in their initial stage and the need for identifying risk factors in order to provide individuals with improved dental care.

REFERENCES

1. Ainamo J, Bay I. Problems and proposals for recording gingivitis and plaque. *Int Dent J* 1975;25:229-235.
2. Ainamo J, Parviainen K. Occurrence of plaque gingivitis and caries as related to self reported frequency of toothbrushing in fluoride areas in Finland. *Community Dent Oral Epidemiol* 1979;7:142-146.
3. Alaluusua S, Malmivirta R. Early plaque accumulation: a sign for caries risk in young children. *Community Dent Oral Epidemiol* 1994;22:273-276.
4. Almagro-Nievas D, Benítez-Hita JA, García-Aragón MA, López-Lorca MT. Incremento del índice de dientes permanentes cariados perdidos por caries y obturados entre escolares de Loja España. *Salud Publica Mex* 2001;43:192-198.
5. Bedos C, Brodeur JM. Déterminants de la carie dentaire parmi les écoliers haïtiens et implications pour la santé publique. *Sante* 2000;10:161-168.
6. Ekstrand KR, Bruun G, Bruun M. Plaque and gingival status as indicators for caries progression on approximal surfaces. *Caries Res* 1998;32:41-45.
7. Etty EJ, Henneberke M, Gruythuysen RJ, Wöltgens JHM. Influence of oral hygiene on early enamel caries. *Caries Res* 1994;28:132-136.
8. Ferreira MAF, Latorre MRDO, Rodrigues CS, Lima KC. Effect of regular fluoride gel application on incipient carious lesions. *Oral Health Prev Dent* 2005;3:141-149.
9. Harris R, Nicoll AD, Adair PM, Pine CM. Risk factors for dental caries in young children: a systematic review of the literature. *Community Dent Health* 2004;21:71-85.
10. Johnson MF. The role of risk factors in the identification of appropriate subjects for caries clinical trials: design considerations. *J Dent Res* 2004;83:116-118.
11. Jose B, King NM. Early childhood caries lesions in preschool children in Kerala India. *Pediatr Dent* 2003;25:594-600.
12. Kidd EAM, Fejerskov O. What constitutes dental caries? Histopathology of carious enamel and dentin related to the action of cariogenic biofilms. *J Dent Res* 2004;83:35-38.

13. Kingman A, Selwitz RH. Proposed methods for improving the efficiency of the DMFS index in assessing initiation and progression of dental caries. *Community Dent Oral Epidemiol* 1997;25:60-68.
14. Kleemola-Kujala E, Räsänen L. Relationship of oral hygiene and sugar consumption to risk of caries in children. *Community Dent Oral Epidemiol* 1982;10:224-233.
15. Klock B, Emilson CG, Gustavsdotter M, Olhede-Westerlund AM. Prediction of caries activity in children with today's low caries incidence. *Community Dent Oral Epidemiol* 1989;17:285-288.
16. Mascarenhas AK. Oral hygiene as a risk indicator of enamel and dentin caries. *Community Dent Oral Epidemiol* 1998;26:331-339.
17. Mathiesen AT, Ogaard B, Rolla G. Oral hygiene as a variable in dental caries experience in 14-year-olds exposed to fluoride. *Caries Res* 1996;30:29-33.
18. Ogaard B, Seppä L, Rolla G. Relationship between oral hygiene and approximal caries in 15-year-old Norwegians. *Caries Res* 1994;28:297-300.
19. Parviainen K, Nordling H, Ainamo J. Occurrence of dental caries and gingivitis in low, medium and high fluoride areas in Finland. *Community Dent Oral Epidemiol* 1977;5:287-291.
20. Pereira AC, Mialhe FL. Diagnóstico da doença cárie. In: Pereira AC (ed). *Odontologia em saúde coletiva-planejando ações e promovendo saúde*. São Paulo: Artmed 2003;216-264.
21. Petry P, Victora CG, Santos IS. Adultos livres de cárie: estudo de casos e controles sobre conhecimentos atitudes e práticas preventivas. *Cad Saúde Pública* 2000;16:154-153.
22. Poulsen S, Holm AK. The relation between dental caries in the primary and permanent dentition of the same individual. *J Public Health Dent* 1980;40:17-25.
23. Raadal M, Espelid I. Caries prevalence in primary teeth as a predictor of early fissure caries in permanent first molars. *Community Dent Oral Epidemiol* 1992;20:30-34.
24. Ratledge DK, Kidd EAM, Beighton D. A clinical and microbiological study of approximal carious lesions. Part I: the relationship between cavitation, radiographic lesion depth, the site-specific gingival index and the level of infection on the dentine. *Caries Res* 2001;35:3-7.
25. Retnakumari N. Prevalence of dental caries and risk assessment among primary school children of 6-12 years in the Varkala municipal area of Kerala. *J Indian Soc Pedod Prev Dent* 1999;17:135-142.
26. Russel JI. Prediction of caries increment in Scottish adolescents. *Community Dent Oral Epidemiol* 1991;19:74-77.
27. Santos AP, Soviero VM. Caries prevalence and risk factors among children aged 0 to 36 months. *Pesquisa Odont Bras* 2002;16:203-208.
28. Schröder U, Granath L. Dietary habits and oral hygiene as predictors of caries in 3-year-old children. *Community Dent Oral Epidemiol* 1983;11:308-311.
29. Segovia-Villanueva A, Estrella-Rodríguez R, Medina-Solís CE, Maupomé G. Severidad de caries y factores asociados em preescolares de 3-6 años de edad em Campeche México. *Rev Salud Publica (Bogota)* 2005;7:56-69.
30. Stecksén-Blicks C, Gustafsson L. Impact of oral hygiene and use of fluorides on caries increment in children during one year. *Community Dent Oral Epidemiol* 1986;14:185-189.
31. Thylstrup A. How should we manage initial and secondary caries? *Quintessence Int* 1998;29:594-598.
32. Tubert-Jeannin S, Lardon JP, Pham E, Martin JL. Factors affecting caries experience in French adolescents. *Community Dent Oral Epidemiol* 1994;22:30-35.
33. Tucker GJ, Andlaw RJ, Burchell CK. The relationship between oral hygiene and dental caries incidence in 11-year-old children: a 3-year study. *Br Dent J* 1976;141:75-79.
34. Vanobbergen J, Martens L, Lesaffre E, Bogaerts K, Declerck D. The value of a baseline caries risk assessment model in the primary dentition for the prediction of caries incidence in the permanent dentition. *Caries Res* 2001;35:442-450.
35. Wendt LK, Hallonsten AL, Birkhed D. Oral hygiene in relation to caries development and immigrant status in infants and toddlers. *Scand J Dent Res* 1994;102:269-273.
36. World Health Organization. *Oral Health Surveys: Basic Methods*. Geneva: ORH/EPID 1997.