Assessment of single risk indicators in relation to caries increment in adolescents

Maarit Raitio, Kaisu Pienihäkkinen and Arje Scheinin
Oulu Municipal Health Center, Dental Health Care, Oulu, and Department of Cariology, Institute of Dentistry, University of Turku, Turku, Finland

Raitio M, Pienihäkkinen K, Scheinin A. Assessment of single risk indicators in relation to caries increment in adolescents. Acta Odontol Scand 1996;54:113–117. Oslo. ISSN 0001–6357.

Base-line data on a series of risk indicators were related to 11-month caries increment in 181 subjects with a mean age of 13 years and 3 months. A caries increment equalling or exceeding one tooth surface was recorded in 21% of the subjects. The risk indicators consisted of past caries experience, white spot lesions, visible plaque and gingivitis, and six salivary tests: secretion rate, buffer effect, sucrase, mutans streptococci, lactobacilli, and *Candida*. Significant associations between caries increment and past caries experience ($p = 0.002$), white spot lesions ($p = 0.01$), lactobacilli ($p = 0.02$), *Candida* ($p = 0.006$), and sucrase ($p = 0.02$) were observed. The ensuing odds ratios were thus recorded: past caries experience, 3.6; white spot lesions, 2.9; salivary sucrase activity, 2.9; lactobacilli, 2.5; and *Candida*, 2.8. □ *Dental caries; risk assessment; saliva; sex-related differences*

Maarit Raitio, Dental Health Care, Oulu Health Center, PL 365, FIN-90101 Oulu, Finland

To reduce the cost of dental health care in children, preventive measures should be targeted to caries risk subjects. Consequently, methods for identifying caries-prone children should be available for practical implementation. Various indicators covering clinical, microbiologic, salivary and socioeconomic factors have thus been analyzed and discussed in relation to caries in children and adolescents (1, 2). So far, previous reports have shown that none of the indicators associated with caries increment might reach the target level of 80% of correct predictions (3–7). Even past caries experience, often shown to be the strongest predictor of future caries (7, 8), has limitations in populations with either very low or very high prevalence of caries (7–11). In contrast, however, multifactorial assessment has proved its value when using caries increment as the dependent variable in longitudinal studies (9–14). The aim of the present study was to analyze the predictive value of a series of caries risk indicators as single factors in relation to 11-month caries increment. The results obtained through multifactorial modeling will be reported separately in a subsequent study.

Materials and methods

Subjects

The initial number of subjects was 197, all attending elementary school in Oulu. After 16 dropped out owing to change of residence, illness or medication, 181 subjects, 101 boys and 80 girls, were observed over a mean 11-month period, in 1989–90. The mean age of the subjects was 13 years and 3 months (range, 11 years and 10 months to 14 years and 11 months). The children were provided with dental health services covering all fields of clinical dentistry at the clinics of the local Municipal Health Center. Fluoride varnish was applied topically once or twice a year in all cases. All the necessary restorative procedures were completed before the commencement of the study. The subjects were asked to refrain from eating and smoking for an hour before the tests and not to brush their teeth in the morning of the examination. Subjects taking drugs that reduce the salivary secretion rate and/or antibiotics within a 2-week period preceding the day of examination were excluded. None of the subjects received topical applications of chlorhexidine before or during the course of the study. The clinical examinations were carried out over a 3-month period in the mornings at the clinic closest to the subjects' schools. The recordings and the tests were conducted by one dentist (M. Raitio) assisted by a chairside assistant.

Caries observations

Coronal caries, including deep enamel and dentinal caries lesions (WHO codes 2–4), was recorded clinically on a tooth surface basis (15); radiographs were not taken owing to existing regulations. Filled surfaces, sealants, and the number of erupted teeth were assessed clinically. Early enamel lesions (WHO code 1) were thus not included in the DFS index. The caries increment (ΔDFS) was calculated as the difference between the figures recorded at the final and the base-line examinations. Any increase in the DFS index was considered a sign of caries activity.

White spot lesions

Incipient lesions, located on buccal and lingual tooth surfaces (WHO code 1), were recorded when the

Table 1. Mean values and standard deviations of age, number of teeth, past caries experience (DFS), decayed surfaces (DS), filled surfaces (FS), and sealants in 11- to 14-year-olds in Oulu, Finland, at the base-line examination in 1989 (n = 181). There were no significant differences between boys and girls (Student's t test)

Variables	Boys		Girls	
	Mean	SD	Mean	SD
Age	13.5	10.8	13.1	9.8
No. of teeth	26.0	3.3	26.6	3.0
DFS	2.8	4.2	2.6	4.1
DS	0.1	0.4	0.3	1.1
FS	2.7	4.0	2.2	3.4
Sealants	5.3	2.7	5.7	2.8

cervical border was ≤1 mm from the gingival margin (15). The presence of white spots was used as a risk indicator.

Visible plaque index

The index was calculated as the percentage of teeth with visible plaque buccally and/or lingually.

Gingivitis index

The index was calculated as the percentage of teeth with one or more clinical signs of inflammation—that is, bleeding, swelling, and redness.

Salivary secretion rate

Salivary secretion was prestimulated by chewing paraffin wax for 2 min, followed by inoculation for the Strip Mutans test and further chewing for 5 min. Saliva was collected during the latter phase (ml/min), its buffer effect and sucrase activity were measured, and the rest of the saliva used for the dip-slide tests of lactobacilli and *Candida.*

Buffer effect

The buffer effect was measured by the colorimetric screening method Dentobuff® (Orion Diagnostica, Espoo, Finland), in which the color of the sample was compared against the manufacturer's model chart and interpreted on a scale of nine scores between pH 3.0 and 7 (16).

Sucrase activity

The activity of sucrose-cleaving enzymes was measured in accordance with the method of Hämäläinen et al. (17). The final glucose concentration was measured with Dextrostix® Strips (Ames Division, Miles, Algol AB, Helsinki, Finland), and the results expressed as four scores from 0 to 5.0 mmol/min × g prot × 10^{-3}.

Mutans streptococci

The Dentocult® SM Strip Mutans (Orion Diagnostic) was used to estimate the salivary count of mutans streptococci (18). The results were expressed on a four-step scale ranging from no counts to ≥10^6 colony-forming units per ml (CFU/ml).

Lactobacilli

Dentocult®-LB (Orion Diagnostica) was used for estimation of the salivary lactobacillus count (19). The

Table 2. Range, dimension, selected cutoff point, and proportion of unfavorable (=positive) test values and 11-month caries increment (ΔDFS) in 11- to 14-year-olds in Oulu, Finland, at the base-line examination in 1989 (n = 181). In the marked variables there was a significant difference between boys and girls (chi-square test)

Variable	Range	Dimension	Cutoff point	Proportion of positive tests
DFS	0–24	Frequency	≥1	0.57
White spot lesions	0–14	Frequency	≥1	0.61
Visible plaque, %	0–100	Percentage	≥25	0.31*
Gingivitis, %	0–39.3	Percentage	≥1	0.17
Secretion rate	0.3–3.8	Ml/min	≤0.9	0.18†
Buffer effect	3.5–7	pH	≤4.5	0.23‡
Sucrase	0–5	Mmol/min × g prot 10^{-3}	≥1.5	0.13
Mutans streptococci	0–≥10^6	Cfu/ml	≥10^5	0.35
Lactobacilli	0–≥10^6	Cfu/ml	≥10^{-5}	0.33
Candida	0–≥10^6	Cfu/ml	≥10^3	0.29
ΔDFS	0–15	Frequency	≥1	0.21

* Boys, 0.39; girls, 0.21; p = 0.01.
† Boys, 0.12; girls, 0.26; p = 0.01.
‡ Boys, 0.13; girls, 0.35; p < 0.001.

Table 3. Sensitivity (Sn), specificity (Sp), positive (Pv^+) and negative (Pv^-) predictive values, percentage of correct predictions (A), relative risk (RR), and odds ratio (OR) and its 95% confidence limits of risk variables when related to 11-month caries increment in 11- to 14-year-olds in Oulu, Finland (n = 181)

Variable	Sn, %	Sp, %	Pv^+, %	Pv^-, %	A, %	RR	OR and 95% confid. limits	
DFS	79	49	29	90	55	2.8	3.6	1.5–8.4
White spot lesions	79	43	27	89	51	2.4	2.9	1.2–6.7
Visible plaque, %	37	71	25	81	64	1.3	1.4	0.7–3.0
Gingivitis, %	21	84	26	80	71	1.3	1.4	0.6–3.4
Secretion rate	24	83	27	80	71	1.4	1.5	0.6–3.7
Buffer effect	21	77	20	79	65	0.9	0.9	0.4–2.1
Sucrase	24	90	39	82	76	2.1	2.9	1.1–7.2
Mutans streptococci	42	67	25	81	62	1.4	1.5	0.7–3.1
Lactobacilli	50	71	32	84	67	2.0	2.5	1.2–5.2
Candida	47	76	34	84	70	2.2	2.8	1.3–5.8

results were grouped into five categories between no counts and 10^6 CFU/ml.

Candida

Oricult®-N (Orion Diagnostica), a method for determining yeasts (20), was used for the salivary *Candida* counts. The colonies were read off by reference to the Dentocult-LB model density chart.

Analysis of data

All the variables were evaluated for dichotomization at several cutoff points, the selection being based on the best practical combination of sensitivity and specificity (21). The variables were further analyzed by means of ratios measuring accuracy (percentage of correct predictions), positive and negative predictive values (22), and relative risk and odds ratio (23). The significance of the observations and the effect of age and sex were tested with the chi-square test or Student's t test, the significance level being $p < 0.05$. The SAS statistical software was used for data management and for the statistical tests (24).

Results

Base-line observations

Mean values and standard deviations for age, number of teeth, past caries experience, and decayed and sealed surfaces at the base-line examination were calculated (Table 1). The proportion of subjects with past caries experience was 57%. Significant differences were found between the boys and girls with regard to visible plaque ($p = 0.01$), salivary secretion rate ($p = 0.01$), and buffer effect ($p < 0.001$) (Table 2).

Follow-up observations

The mean caries increment (ΔDFS) for those who developed new caries—that is 21% of the subjects—was 2.4 surfaces. Only 10% (8/78) of the initially caries-free subjects developed caries. The corresponding proportion in those with past caries experience was 29% (30/103).

The cutoff points when relating the risk indicators to the caries increment were ≥1 for white spot lesions, ≥1 for past caries experience, ≥25% for visible plaque, ≥1% for gingivitis, ≤0.9 ml/min for salivary secretion rate, ≤4.5 pH for salivary buffer effect, ≥1.5 mmol/min × g prot 10^{-3} for salivary sucrase, ≥10^5 for mutans streptococci, ≥10^5 for lactobacilli and ≥10^3 for *Candida* (Table 2).

When the boys and girls were analyzed together, past caries experience ($p = 0.002$), white spots lesions ($p = 0.01$), lactobacilli ($p = 0.02$), *Candida* ($p = 0.006$), and sucrase ($p = 0.02$) were significantly associated with ΔDFS (Table 3). However, visible plaque, gingivitis, salivary secretion rate, buffer effect, and mutans streptococci had no significant association with ΔDFS (Table 3). On the other hand, when the boys and girls were analyzed separately, white spot lesions ($p = 0.001$) and *Candida* ($p = 0.002$) were found to be significantly associated with ΔDFS solely in the boys, and past caries experience ($p = 0.003$), gingivitis ($p = 0.02$), and lactobacilli ($p = 0.01$), respectively, only in the girls (Table 4).

Risk indicators were also analyzed separately in subjects with and without past caries experience. *Candida* ($p = 0.005$) and white spots ($p = 0.02$) were significantly associated with ΔDFS in the initially caries-free children, and only sucrase ($p = 0.046$) in those with past caries experience (Table 4).

Discussion

Teenagers are commonly considered to represent a particular risk group for caries (25, 26). Accordingly, such age groups were selected for the present study, and the observed proportion of children with past caries experience represented the average level in Finnish adolescents (27).

Table 4. Sensitivity (Sn), specificity (Sp), positive (Pv^+) and negative (Pv^-) predictive values, percentage of correct predictions (A), relative risk (RR), and odds ratio (OR) and its 95% confidence limits of significant risk variables when related to 11-month caries increment in 11- to 14-year-old boys and girls in Oulu, Finland (upper section). Corresponding figures in initially caries-free children and in those with past caries experience (lower section)

Variable	Sn, %	Sp, %	Pv^+, %	Pv^-, %	A, %	RR	OR and 95% confid. limits	
Boys; n = 101								
White spot lesions	95	44	31	97	55	1.1	15.6	2.0–122
Candida	57	78	40	87	73	3.2	4.6	1.7–13
Girls; n = 80								
DFS	88	52	33	94	60	5.8	8.3	1.7–39
Gingivitis, %	29	92	50	83	79	2.9	4.8	1.2–19
Lactobacilli	53	75	36	86	70	2.5	3.3	1.1–10
Caries-free children; n = 78								
White spot lesions	88	56	18	98	59	7.4	8.8	1.0–75
Candida	63	81	28	95	79	5.6	7.3	1.5–35
Children with past caries experience; n = 103								
Sucrase	27	89	50	75	71	2.0	3.0	1.0–8.8

The high values of the secretion rate and buffer effect in the boys are in accordance with earlier findings (28–30), whereas the plaque index results are in accordance with reports that this is usually lower in girls than in boys (31, 32).

The present observations on the lack of association of visible plaque, gingivitis, salivary secretion rate, or buffer effect with caries increment comply with earlier findings (4, 29). Surprisingly, however, mutans streptococci are not significantly associated with ΔDFS, although they frequently have been found to possess predictive value in univariate analyses (4, 5, 8, 10). On the other hand, the present associations between ΔDFS and salivary counts of lactobacilli and *Candida* are in accordance with earlier observations (5, 6, 9, 12, 13).

The sucrase activity test is a rather new method among salivary diagnostic indicators. In contrast to earlier observations in elderly subjects with a mean age 62 years (12, 13), it showed a significant association with ΔDFS (Table 3), indicating validity as a caries activity test in adolescents.

Past caries experience, shown to be the most powerful single indicator of the risk of future caries increment in children (7–11), yields a higher odds ratio than the other indicators in the present material, although its close association with ΔDFS is restricted to the girls. Comparable sex-related differences in the prediction of caries have also been reported previously (9, 33). On the other hand, white spot lesions are closely related to ΔDFS in the boys, possibly because of their high plaque indices (Table 2). According to Neilson & Pitts (35), the incipient smooth-surface lesions provide a valuable caries risk indicator, especially in individuals with poor oral hygiene. The white spot lesions have also been used as a diagnostic and/or predictive variable previously and found to be significantly associated with caries increment (4, 34–36). A positive correlation between white spot lesions and past caries experience is recorded in the present study, and both variables possess predictive power with regard to caries when the data are analyzed as a whole. The results indicate the importance of past caries experience and white spot lesions in caries prediction. On the other hand, the effect of past caries experience on the association of risk indicators with caries increment also reflects the difficulties of caries prediction in populations with different caries prevalences.

Acknowledgements.—Grants, for the first author, from the Finnish Dental Organizations and Emil Aaltonen Foundation, and the support of Oulu Municipal Health Center and Orion Diagnostica, Espoo, Finland, are gratefully acknowledged.

References

1. Demers M, Brodeur J-M, Simard PL, Mouton C, Veilleux G, Fréchette S. Caries predictors suitable for mass-screenings in children: a literature review. Community Dental Health 1990; 7:11–21.
2. Hausen H, Seppä L, Fejerskov O. Can caries be predicted? In: Thylstrup A, Fejerskov O, editors. Textbook of clinical cariology. 2nd ed. Copenhagen: Munksgaard, 1994:393–411.
3. Stamm JW, Disney JA, Graves RC, Bohannan HM, Abernathy JR. The University of North Carolina Caries Risk Assessment Study. I. Rationale and content. J Public Health Dent 1988;48: 225–32.
4. Klock B, Krasse B. A comparison between different methods for prediction of caries activity. Scand J Dent Res 1979;87:129–39.
5. Stecksén-Blicks C. Salivary counts of lactobacilli and *Streptococcus mutans* in caries prediction. Scand J Dent Res 1985;93:204–12.
6. Pienihäkkinen K, Scheinin A, Bánóczy J. Screening of caries in children through salivary lactobacilli and yeasts. Scand J Dent Res 1987;95:397–404.
7. Wilson RF, Ashley FP. Identification of caries risk in school-children: salivary buffering capacity and bacterial counts, sugar intake and caries experience as predictors of 2-year and 3-year caries increment. Br Dent J 1989;167:99–102.
8. Alaluusua S, Kleemola-Kujala E, Evälahti M. Salivary caries-related tests as predictors of future caries increment in teenagers. A three year longitudinal study. Oral Microbiol Immunol 1990;5:77–81.
9. Honkala E, Nyyssönen V, Kolmakow S, Lammi S. Factors

predicting caries risk in children. Scand J Dent Res 1984;92: 134–40.
10. Russell JI, MacFarlane TW, Aitchison TC, Stephen KW, Burchell CK. Prediction of caries increment in Scottish adolescents. Community Dent Oral Epidemiol 1991;19:74–7.
11. Disney JA, Graves RC, Stamm JW, Bohannan HM, Abernathy JR, Zack DD. The University of North Carolina caries risk assessment study: further developments of caries prediction. Community Dent Oral Epidemiol 1992;20:64–75.
12. Scheinin A, Pienihäkkinen K, Tiekso J, Holmberg S. Multifactorial modeling for root caries prediction. Community Dent Oral Epidemiol 1992;21:35–7.
13. Scheinin A, Pienihäkkinen K, Tiekso J, Holmberg S, Fukuda M, Suzuki A. Multifactorial modeling for root caries prediction: 3-year follow-up results. Community Dent Oral Epidemiol 1994; 22:126–9.
14. Beck JD. Identification of risk factors. In: Bader JD, editor. Risk assessment in dentistry. Chapel Hill (NC): University of North Carolina, Dental Ecology 1990:8–13.
15. WHO. A guide to oral health epidemiological investigations. Geneva: World Health Organization, Oral Health Unit, 1979.
16. Frostell G. A colorimetric screening test for evaluation of buffer capacity of saliva. Swed Dent J 1980;4:81–6.
17. Hämäläinen M, Karjalainen S, Söderling E. A simple test for the determination of salivary sucrase activity. Caries Res 1988;22: 174–6.
18. Jensen B, Bratthall D. A new method for the estimation of mutans streptococci in human saliva. J Dent Res 1989;68: 468–71.
19. Larmas M. A new dipslide method for the counting of salivary lactobacilli. Proc Finn Dent Soc 1975;71:31–5.
20. Nickerson WJ. Reduction of inorganic substances by yeasts. I. Extracellular reduction of sulphide by species of candida. J Infect Dis 1953;93:43–56.
21. Thorner RM, Remein QR. Principles and procedures in the evaluation of screening for disease. Washington (DC): US Public Health Service Publication No 846, Monograph No 76, 1961.
22. Vecchio TJ. Predictive value of a single diagnostic test in unselected populations. N Engl J Med 1966;274:1171–3.
23. Fleiss JL. Statistical methods for rates and proportions. 2nd ed. New York: John Wiley & Sons, 1981.
24. SAS Institute Inc. SAS® user's guide: statistics, version 5 edition. Cary (NC): SAS Institute Inc., 1985.
25. Fédération Dentaire Internationale. Principal requirements for controlled clinical trials of caries preventive agents and procedures. FDI Techn Rep 1 1982;32(No 3):292–310.
26. Nordblad A, Larmas M. A three year study of caries prevalence, incidence and risk ages in cohorts of schoolchildren in Espoo, Finland. Proc Finn Dent Soc 1985;81:11–6.
27. Nordblad A, Suominen-Taipale L, Rasilainen J. Suun terveydenhuolto terveyskeskuksissa, 1991. STAKES, 1993, Raportteja 115, ISBN 951-47-7701-8, ISSN 1236-0740. (Oral health care in health centers, 1991. Helsinki: STAKES, Reports 115), 1993.
28. Andersson R, Arvidsson E, Crossner C-G, Holm A-K, Månsson B, Grahnén H. The flow rate, pH and buffer effect of mixed saliva in children. J Int Assoc Dent Child 1974;5:5–12.
29. Pienihäkkinen K, Nemes J, Scheinin A, Bánóczy J. Salivary buffering capacity and its relation to caries increment in children. Proc Finn Dent Soc 1987;83:47–54.
30. Söderling E, Pienihäkkinen K, Alanen M-L, Hietaoja M, Alanen P. Salivary flow rate, buffer capacity, sodium and amylase in adolescents. A longitudinal study. Scand J Dent Res 1993;101: 98–102.
31. Parviainen K, Nordling H, Ainamo J. Occurence of dental caries and gingivitis in low, medium and high fluoride areas in Finland. Community Dent Oral Epidemiol 1977;5:287–91.
33. Kolehmainen L. Effect of systematic oral hygiene instruction on oral health among 13-year-old schoolchildren. Proc Finn Dent Soc 1988;79:211–7.
34. Bader JD, Graves RC, Disney JA, Bohannan HM, Stamm JW, Abernathy JR, et al. Identifying children who will experience high caries increments. Community Dent Oral Epidemiol 1986;14:198–201.
35. Neilson A, Pitts NB. The clinical behaviour of free smooth surface carious lesions monitored over 2 years in a group of Scottish children. Br Dent J 1991;171:313–8.
36. Pienihäkkinen K. Caries prediction through combined use of incipient caries lesions, salivary buffering capacity, lactobacilli and yeasts. Community Dent Oral Epidemiol 1987;15:325–8.
37. Seppä L, Hausen H. Frequency of initial caries lesions as predictor of future caries increment in children. Scand J Dent Res 1988;96:9–13.

Received for publication 31 August 1994
Accepted 15 August 1995