Multifactorial modeling for prediction of 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. Multifactorial modeling for prediction of caries increment in adolescents. Acta Odontol Scand 1996;54:118–121. Oslo. ISSN 0001–6357.

The purpose of the study was to develop a multifactorial model for the prediction of 11-month caries increment in adolescents. The mean age of the subjects (n = 181) at the base-line examination was 13 years and 3 months. The risk indicators consisted of past caries experience, white spot lesions, visible plaque, gingivitis, salivary secretion rate, buffer effect, sucrase, mutans streptococci, lactobacilli, and *Candida*. The multifactorial modeling included all the above risk indicators, age, and gender and resulted in different models in boys and girls, indicating the difficulty of caries prediction in adolescents. When boys and girls were combined, the final model included past caries experience, *Candida*, and salivary sucrase. Although the accuracy of the model was slightly below the 80% level recommended for screening purposes, the results provide clinically valuable information. The risk of caries increases with an increasing number of positive tests within the model. □ Candida; *dental caries; gender; risk assessment; saliva; sucrase*

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

Several clinical, microbiologic, and sociodemographic factors have been used separately and in combination to identify children with high caries risk (1–8). Multifactorial modeling has proved its value in longitudinal studies of caries prediction by showing the interrelations and interactions of risk factors. Modeling has usually been based on a dichotomized dependent variable, either as no versus some caries increment (9–11) or with specified cutoff points in populations with high caries incidence (1–3, 7–8, 10, 12). Furthermore, models with continuous or ordinal variables as the dependent variable have been developed for various practical implementations (6, 13). The crude hit rate (accuracy) of the models has rarely reached the 80% level, considered to be the minimum target level for screening purposes. On the other hand, modeling with dichotomized data produces risk-associated estimates (relative risk and odds ratio), which may possess practical value, providing a means of quantitative evaluation of caries risk at the individual level.

The main purpose of the present study was to assess a series of clinical and salivary risk indicators in the prediction of caries increment in adolescents. The description of base-line data and the association of single factors to 11-month caries increment have been reported separately (14). The present report was aimed to analyze the combined effect of the risk indicators and their interaction in relation to caries increment through multifactorial modeling.

Materials and methods

The subjects (101 boys and 80 girls) and methods have previously been reported in detail (14); they are described here insofar as relevant to the present analyses. The mean age of the subjects initially, in 1989, was 13 years and 3 months (from 11 years and 10 months to 14 years and 11 months). The risk indicators included four clinical registrations—that is, past caries experience (DFS), white spot lesions (defined as white, opaque lesions on the buccal or lingual tooth surfaces close to the gingival margin (15)), percentage of teeth with visible plaque, and percentage of teeth with gingivitis—and six salivary determinations—that is, paraffin-stimulated secretion rate (ml/min), buffer effect (Dentobuff®, Orion Diagnostica, Espoo, Finland), sucrase activity (Dextrostix®, Ames Division, Miles, Algol AB, Helsinki, Finland), mutans streptococci (Dentocult®-SM Strip Mutans, Orion Diagnostica), lactobacilli (Dentocult®-LB, Orion Diagnostica), and *Candida*/yeasts (Oricult®-N, Orion Diagnostica). All enamel and dentinal lesions (WHO codes 1–4) and fillings were registered for each tooth surface. Early enamel lesions (WHO code 1) were not included in the DFS index. The caries increment was calculated as the difference between the DFS indices registered at the base-line and the final examinations. A caries increment, considered as a sign of caries activity, was found in 21% of the subjects.

In multifactorial modeling the 11-month caries increment (ΔDFS) was used as the dependent variable. All the above risk factors, together with past caries experience, gender, and age, the latter dichotomized as <13 and ≥ 13 years, were used as independent variables. Logistic regression analysis was used to estimate the difference in the logarithmic risk of caries activity. The models were developed in accordance with Kleinbaum et al. (16), using the BMDPLR procedure in the BMDP statistical software (17). Initially, all univariate factors

Table 1. Regression coefficients, standard errors, odds ratios (OR), and 95% confidence limits for variables related to risk of caries-active status, on the basis of the final model, in 11- to 14-year-old boys and girls and in caries-free subjects in Oulu, Finland

Variable	Coefficient	SE	OR and its 95% confidence limits	
Boys; n = 101				
White spots	2.17	1.09	8.8	1.0–76
Candida × white spots	1.31	0.56	3.7	1.2–11
Constant	−3.56	1.01		
Girls; n = 80				
DFS × age	2.13	0.61	8.4	2.5–28
Constant	−2.28	0.47		
Caries-free subjects; n = 78				
Candida × white spots	2.88	0.85	17.8	3.3–96
Constant	−3.06	0.59		

were included; subsequently, all possible two-factor interaction terms were added one by one, and those found significant and 'sensible' were included. Non-significant terms were then excluded from the model. The final model was the most parsimonious and clinically sensible model. As gender had a significant effect on caries increment, the analyses were initially performed for boys and girls separately. The analyses resulted in different models for boys and girls. As this would not be practical, the analysis was carried out also for both sexes combined. Additionally, a separate analysis was carried out in initially caries-free subjects. The risk-odds ratios (OR) and their 95% confidence limits were calculated for the variables of the final model (18). In addition, we calculated the sensitivity, specificity (19), positive and negative predictive values (20), percentage of correct predictions, and relative risk associated with the final model when the best clinically practical criterion was used for screening.

Results

The final model for boys included white spot lesions and *Candida*, with a significant interaction between these variables (Table 1). The estimated odds ratio of being caries-active was 8.8 for those with white spots in comparison with those without. The corresponding additional OR was 3.7 for those with *Candida*-positive tests in comparison with *Candida*-negative subjects, provided that white spots were also present. For those without white spots the presence of *Candida* had no effect. With the best practical cut-off point for screening ('both tests positive'), the overall accuracy of the model with the present material was 78% (Table 4).

The final model for girls included age and past caries experience (DFS), with interaction between the variables (Table 1). In girls ≥13 years old the estimated OR of being caries-active was 8.4 when the subject was DFS-positive in comparison with DFS-negative subjects. In younger girls DFS had no effect on caries activity.

Table 2. Regression coefficients, standard errors, odds ratios (OR), and 95% confidence limits for variables related to risk of caries-active status, on the basis of the final model, in 11- to 14-year-olds (n = 181) in Oulu, Finland

Variable	Coefficient	SE	OR and its 95% confidence limits	
DFS	0.94	0.46	2.6	1.0–6.3
Candida	1.16	0.41	3.2	1.4–7.2
Sucrase × DFS	1.46	0.59	4.3	1.3–14
Constant	−2.54	0.42		

The best practical cut-off point for screening was 'both tests positive', resulting in an accuracy of 76% (Table 4).

When boys and girls were analyzed together, the final model included DFS, *Candida*, and salivary sucrase, with interaction between DFS and sucrase (Table 2). The estimated OR was 2.6 when the subject was DFS-positive; the additional OR for *Candida*-positive subjects was 3.2, and that for sucrase-positive subjects 4.3, provided that also DFS was positive. In DFS-negative subjects sucrase had no effect.

The observed and estimated distributions of the subjects on the basis of positive and negative tests and proportions of subjects with caries activity are shown in Table 3. The best practical criterion—that is, 'presence of past caries and *Candida*- and/or sucrase-positive' resulted in the present subjects in an accuracy of 75% (Table 4). The ability of the model to identify subjects developing caries was 55%, and those not developing caries, 80% (Table 4).

When analyzed separately in caries-free subjects, the final model included white spots and *Candida*, with interaction between the variables (Table 1). In subjects with white spots the estimated OR of being caries-active was 17.8 when the subject was also *Candida*-positive. With the criterion 'both tests positive' the model resulted in an accuracy of 88% (Table 4).

Discussion

The present subjects were teenagers, more than half of them having caries or fillings at the base-line examination. Since approximately one in five developed deep enamel or dentinal caries lesions during the follow-up, the subjects are considered to match Finnish adolescents in general. The relatively low number of subjects limited the analysis to modeling. As the model could not be validated in a separate material, the present accuracy values are probably slightly overestimated. The importance of the results lies in the multifactorial approach to caries prediction and in their clinical implications.

Gender-related differences in children and adolescents have been demonstrated previously in oral health and in many variables associated with it. For instance, girls have a higher caries experience (21) and a lower

Table 3. Distribution of subjects on the basis of positive and negative tests within the final model, and observed and estimated proportions of caries increment

DFS	*Candida*	Sucrase	Subjects, *n*	Proportion of caries increment	
				Observed	Estimated
–	–	–	55	0.04	0.07
–	–	+	5	0.20	0.07
+	–	–	54	0.17	0.17
–	+	–	16	0.31	0.20
–	+	+	2	0.00	0.20
+	+	–	33	0.39	0.39
+	–	+	14	0.57	0.47
+	+	+	2	0.00	0.74

salivary secretion rate and buffer effect than boys (22, 23). On the other hand, poor oral hygiene is commoner in boys than in girls (24, 25). The relationship between caries risk indicators and caries increment is not necessarily similar in boys and girls, as shown previously by Honkala et al. (9) and Bader et al. (1), and confirmed in the present material. These results, however, are inconsistent with the findings of large field studies with numerous other explanatory variables, in which no association with gender has been observed (2, 7). Whatever the effects, the behavioral and hormonal differences between boys and girls may be additional confounders in caries prediction in adolescents.

The accuracy of the final models—that is, 78% for boys and 76% for girls—is reasonable, although lower than the 80% target level. The use of these tests for mass screening thus cannot be recommended without reservation. The use of different variables and/or criteria for boys and girls would likewise not be practical in clinical caries risk assessment. Thus it is the analysis carried out in the combined material which provides essential information for clinical use. Furthermore, this analysis resulted in marginally lower accuracy (75%) in screening than the analyses with boys and girls separately. The effect of the multifactorial analysis and the combination of information from several tests is demonstrated by the increase in risk ratio values. The better balance between the proportion of positive cases and those with caries increment is an even more important improvement, especially when accompanied by relatively high accuracy values.

Table 4. Sensitivity (Sn), specificity (Sp), positive (Pv^+) and negative (Pv^-) predictive values, percentage of correct predictions (A), and relative risk (RR) associated with the final models in boys, girls, all subjects, and in initially caries-free subjects, with best practical criterion used for screening

Model and criterion	Sn, %	Sp, %	Pv^+, %	Pv^-, %	A, %	RR
Boys (n = 101): white spots, *Candida*						
Both tests positive	57	84	48	88	78	4.0
Girls (n = 80): DFS, age						
Both tests positive	71	78	46	91	76	5.0
Total (n = 181): DFS, *Candida*, sucrase						
DFS positive, and *Candida* and/or sucrase positive	55	80	43	87	75	3.3
Caries-free subjects (n = 78): white spots, *Candida*						
Both tests positive	63	91	45	96	88	9.7

The present findings indicate the importance of past caries experience in the prediction of caries but also that this variable is not acceptable as a single test at either the group or the individual level. This is in line with earlier observations in children and adolescents (5, 6, 9). The presence of *Candida* along with past caries experience provided additional information for the prediction of caries increment in the present subjects. This accords with an earlier study with elderly subjects (11). It is not suggested that the presence of *Candida* is causally associated with caries, their presence being merely an indication of unfavorable conditions in the oral cavity. The activity of salivary sucrase, as the third risk indicator in the final model, may also be of potential value in caries prediction in adolescents, although it has not been found to be significant in the prediction of root caries in the elderly (11).

Surprisingly, the present model does not include lactobacilli, although these have frequently been found to have the greatest power in caries prediction after past caries experience (6, 12, 13). In fact, the present model with adolescents differs from the final root caries model for the elderly only in the replacement of lactobacilli by sucrase (root DFS, *Candida*, lactobacilli) (11).

The model identified 55% of subjects who developed caries within 11 months. This is acceptable, when combined with 80% ability to identify those not developing caries. The figures are comparable also with those of earlier studies in adolescents (1, 2, 7, 8, 10). More strict criteria for ΔDFS (≥2 or ≥3) produced even higher sensitivity and specificity combinations, but clinical approach and low caries prevalence made it most practical to select the 'no versus some caries' approach for the study.

In a selected group of caries-free subjects a clearly higher accuracy of 88% (63% sensitivity, 91% specificity) was obtained, reaching the recommended 80% target level. With caries-free subjects the presence of incipient caries lesions and salivary *Candida* would inform about increased risk for development of caries. Our clinical approach and easily applicable tests increase the clinical value of the results. Comparable 83% accuracy was observed also in a selected material of caries-free 6-year-olds (26). Although informative and valuable as such, these figures cannot be directly compared with those obtained in unselected groups.

The information of the present model (DFS, *Candida*, sucrase) is valuable in the targeting of preventive measures at an individual level and in the motivation of patients. For instance, with subjects with earlier or present caries experience, the use of two tests, one for salivary yeasts and/or *Candida* and the other for salivary

sucrase activity, would provide additive, semiquantitative information of caries risk. The risk of caries increases with an increasing number of positive tests. Other risk indicators analyzed, although significant as single tests, do not provide any additional information for risk assessment. It is necessary, however, to study the validity of the results also in separate materials before making any detailed clinical recommendations.

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

References

1. 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.
2. Abernathy JR, Graves RC, Bohannan HM, Stamm JW, Greenberg BG, Disney JA. Development and application of prediction model for dental caries. Community Dent Oral Epidemiol 1987;15:24–8.
3. Pienihäkkinen K. Caries prediction through combined use of incipient caries lesions, salivary buffering capacity, lactobacilli and yeasts in Finland. Community Dent Oral Epidemiol 1987; 15:325–8.
4. Stecksén-Blicks C. Lactobacilli and *Streptococcus mutans* in saliva, diet and caries increment in 8- and 13-year-old children. Scand J Dent Res 1987;95:18–26.
5. Wilson RF, Ashley FP. Identification of caries risk in schoolchildren: salivary buffering capacity and bacterial counts, sugar intake and caries experience as predictors of two year and 3-year caries increment. Br Dent J 1989;166:99–102.
6. 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.
7. Disney JA, Graves RC, Stamm JW, Bohannan HM, Abernathy JR, Zack DD. The University of North Carolina Caries risk assessment study: further developments in caries risk prediction. Community Dent Oral Epidemiol 1992;20:64–75.
8. Steiner M, Helfenstein U, Marthaler TM. Dental predictors of high caries increment in children. J Dent Res 1992;71:1926–33.
9. Honkala E, Nyyssönen V, Kolmakow S, Lammi S. Factors predicting caries risk in children. Scand J Dent Res 1984;92: 134–40.
10. Beck JD, Weintraub JA, Disney JA, Graves RC, Stamm JW, Kaste LM, et al. University of North Carolina caries risk assessment study: comparisons of high risk prediction, any risk prediction, and any risk etiologic models. Community Dent Oral Epidemiol 1992;20:313–21.
11. 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.
12. Kingman A, Little W, Gomez I, Heifetz SB, Driscoll WS, Sheats R, et al. Salivary levels of *Streptococcus mutans* and lactobacilli and dental caries experiences in US adolescent population. Community Dent Oral Epidemiol 1988;16:98–103.
13. Heintze S. Screening von Individuen mit hohem Kariesrisiko, Evaluation verschiedener Kariesprädiktoren im multifaktoriellen Modell [thesis] Berlin: Der Freie Universität, 1991.
14. Raitio M, Pienihäkkinen K, Scheinin A. Assessment of single risk indicators in relation to caries increment in adolescents. Acta Odontol Scand 1996;54:113–17.
15. WHO. A guide to oral health epidemiological investigations. Geneva: World Health Organization, Oral Health Unit, 1979.
16. Kleinbaum DG, Kupper LL, Morgenstern H. Epidemiological research: principles and quantitative methods, Belmont (CA): Lifetime Learning Publications, 1982:434–53.
17. Dixon WJ, editor. BMDP statistical software manual: to accompany the 1990 software release. Vol. 2. Berkeley: University of California Press, 1990.
18. Fleiss JL. Statistical methods for rates and proportions. 2nd ed. New York: John Wiley & Sons, 1981.
19. 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 67, 1961.
20. Vecchio TJ. Predictive value of a single diagnostic test in unselected populations. N Engl J Med 1966;274:1171–3.
21. Stamm JW. Is there a need for dental sealants? Epidemiological indications in the 1980s. J Dent Education 1984;48:9–17.
22. Andersson R, Arvidsson E, Crossner C-G, Holm A-K, Mänsson B, Grahnén H. The flow rate, pH and buffer effect of mixed saliva in children. J Int Assoc Dent Child 1974;5:5–12.
23. 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.
24. 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;6:287–91.
25. Kolehmainen L, Heinonen OP, Haapakoski J. Caries prediction and its evaluation in 13- to 15-year-old schoolchildren. Community Dental Health 1985;2:15–21.
26. Leverett DH, Proskin HM, Featherstone JDB, Adair SM, Eisenberg AD, Mundorff-Shrestha SA, et al. Caries risk assessment in a longitudinal discrimination study. J Dent Res 1993;72:538–43.

Received for publication 22 May 1995
Accepted 15 August 1995