

Longitudinal validation of a risk calculator for periodontal disease

Roy C. Page¹, John Martin²,
Elizabeth A. Krall^{3,4}, Lloyd Mancil⁵
and Raul Garcia^{3,4}

Page RC, Martin J, Krall EA, Mancil L, Garcia R. Longitudinal validation of a risk calculator for periodontal disease. *J Clin Periodontol* 2003; 30: 819–827. © Blackwell Munksgaard, 2003.

Abstract

Background: Risk assessment and utilization of the results are important components of prevention, diagnosis and treatment of periodontal diseases. Risk assessment is relatively new to dentistry. Currently risk is assessed by subjective evaluation and results vary widely among clinicians. We have developed a computer-based risk assessment tool, the Periodontal Risk Calculator (PRC), for objective, quantitative assessment of risk. The purpose of the study reported here was to evaluate the accuracy and validity of this tool.

Methods: Clinical records and radiographs of 523 subjects enrolled in the VA Dental Longitudinal Study of Oral Health and Disease, covering a period of 15 years, were used. Information from baseline examinations was entered into the risk calculator and a risk score on a scale of 1–5 for periodontal deterioration was calculated for each subject. Actual periodontal status in terms of alveolar bone loss determined using digitized radiographs, and tooth loss determined from the clinical records, was assessed at years 3, 9 and 15. The strength of the association between risk prediction and actual outcome was determined statistically.

Results: The risk scores were strong predictors of future periodontal status measured as worsening severity and extent of alveolar bone loss and tooth loss, especially loss of periodontally affected teeth. Over the entire 15-year period, risk scores consistently ranked groups from least to most bone loss and tooth loss. Risk groups differed greatly from one another. By year 3, the incidence rate of bone loss of group 5 was 3.7-fold greater than for group 2, and by year 15, the loss of periodontally affected teeth was 22.7-fold greater than for group 2 ($p < 0.001$). By year 15, 83.7% of subjects in risk group 5 had lost one or more periodontally affected teeth compared to 20.2% of subjects in group 2.

Conclusions: Risk scores calculated using the PRC and information gathered during a standard periodontal examination predict future periodontal status with a high level of accuracy and validity. Use of the risk assessment tool over time may be expected to result in more uniform and accurate periodontal clinical decision-making, improved oral health, reduction in the need for complex therapy and reduction in health-care cost.

¹Department of Periodontics and the Regional Clinical Dental Research Center, School of Dentistry, University of Washington, Seattle, WA 98195, USA;

²Private practice, 120 South Burrowes St., Suite 102, State College, PA 16801, USA;

³VA Dental Longitudinal Study, Massachusetts Veterans Epidemiology Research and Information Center, VA Boston Healthcare System, Boston, MA, USA;

⁴Department of Health Policy and Health Services Research, Boston University Goldman School of Dental Medicine, Boston, MA 02118, USA; ⁵Department of Dental Public Health Sciences, School of Dentistry, University of Washington, Seattle, WA 98195, USA

Key words: risk factors; periodontal diagnosis; periodontal treatment; quantification of risk

Accepted for publication 26 November 2002

Over the last three decades, research on dental diseases, especially periodontal diseases, has been intensive and our knowledge base and understanding have grown enormously. These studies have demonstrated that the host plays a major role in the pathobiology of periodontitis and that risk varies greatly from one individual to another (Hirschfeld & Wasserman 1978, McFall 1982, Lindhe et al. 1983, 1989, Lang et al. 1986, Jenkins et al. 1988, Beck et al. 1990,

1995, Beck 1998). Several determinants of risk have been identified (Ismail et al. 1990, Beck et al. 1990, Hart & Kornman 1997, Kornman et al. 1997, 1995, Page & Beck 1997, Page et al. 1997, Salvi et al. 1997). Heredity alone appears to account for roughly 50% of the risk for susceptibility to periodontitis (Michalowicz et al. 2000). As a consequence of these findings, management of the major dental diseases is undergoing a transition from the repair

to the medical or wellness model of patient care.

The wellness model is new to dentistry and to periodontics, and its application requires an accurate and valid assessment of risk. Most dentists and periodontists are not trained or experienced in risk assessment nor in using interventions aimed at risk reduction in prevention and management of periodontal diseases. Furthermore, tools for quantification of risk have not been

available. Consequently, when performed, risk assessment consists of recognizing that factors enhancing risk are present in a given case, and making subjective judgements as to the magnitude of their role in the disease process. There is evidence that risk assessments based on subjective expert dentist and periodontist opinion vary too greatly to be useful in clinical periodontal decision-making (Persson et al. 2003). We have developed a computer-based tool, the Periodontal Risk Calculator (PRC) (Dental Medicine International Inc., Mount Vernon, WA, USA), that quantifies risk and predicts periodontal deterioration (Page et al. 2002). The PRC is based on mathematically derived algorithms that assign relative weights to the various known risks that enhance susceptibility for periodontitis (Martin & Page, unpublished). It determines the level of risk on a scale of 1–5 and generates suggested treatment options to guide the clinician and patient toward a health-care strategy based on risk reduction. The purpose of the study reported here was to document the extent of agreement between risk scores calculated using the PRC and information gathered during a baseline examination with the actual periodontal status 3, 9 and 15 years later. A portion of the results of this study have been reported previously (Page et al. 2002).

Methods

Study population

The study population consisted of subjects enrolled in the VA Dental Longitudinal Study of Oral Health and Disease, an ongoing closed-panel study begun in 1968.

Comprehensive medical and dental examinations were performed upon enrollment and repeated in cycles of approximately 3 years. Of the 1231 subjects enrolled, 1157 were dentate at baseline. Of these, 523 subjects ranging in age from 25 to 74 years were present at all examinations through cycle 6 (15 years) and had records with complete data. The subjects, who were all men, were not VA patients, but rather men recruited from the Greater Boston area who received their medical and dental care in the private sector. Data from the records and radiographs taken at the baseline examination were entered into the PRC, and risk scores on a scale of 1–5 for periodontal deterioration were calcu-

lated for each subject. Using records and radiographs for years 3, 9 and 15, we determined the actual change in periodontal disease severity and extent measured as change in radiographic alveolar bone height, periodontal probing pocket depth and loss of teeth that were periodontally affected and unaffected at baseline. Additional details about the VA Longitudinal Study of Oral Health and Disease including training and calibration of the clinical examiners have been published (Kapur et al. 1972, Glass et al. 1973, Fleiss & Chilton 1983).

Baseline and subsequent examinations

Medical and dental histories were recorded, and full-mouth radiographs with bitewings were taken at baseline and at each subsequent examination cycle. At each of the examinations during the 15-year study period, each subject was asked to respond yes or no to the question “have you had any gum treatments or gum surgery since your last examination”? These responses were used as a measure of treatment. Clinical examination consisted of charting caries and restorations on all teeth present, and assessment of periodontal status by probing using a hand-held periodontal probe (Feldman et al. 1982). Clinicians measured probing pocket depths at multiple sites around all teeth. A single ordinal score was assigned for each tooth based on the deepest probed site. The ordinal scores were converted to millimeters for use in the risk calculator analysis (for example, the highest ordinal score of 3 was equivalent to a 5 mm or greater probing depth). We read radiographs from the baseline examination to identify the presence of molar furcations, vertical bone lesions, defective restorations and root calculus. Using digitized periapical films (Jeffcoat et al. 1984), we measured the distance from the cemento-enamel junction (CEJ) to the alveolar bone crest at the mesial and distal sites of all teeth present that were measurable. Any tooth having a distance of 2mm or greater from the CEJ to the alveolar crest, or a pocket depth of 5 mm or greater at baseline was designated as periodontally affected. Information obtained from the baseline examination was entered into the PRC, and a risk score on a scale of 1–5 was calculated for each subject. Specific data required by the PRC for the calculation have been reported (Page et al. 2002).

Determination of changes in periodontal status

Changes in periodontal status over time were determined by comparing the clinical and historic records and radiographs taken at years 3, 9 and 15 with the baseline records and radiographs. Changes in pocket depth over time were assessed as a measure of disease severity. Bone height, defined as the percent distance from the CEJ to the root apex, was measured from digitized films (Jeffcoat et al. 1984). Mean bone loss, a measure of disease severity, was defined as the mean reduction in bone height at all sites that had bone loss greater than the threshold of 2% for all sites that could be compared. Sites manifesting bone height worsening, a measure of the extent of disease within subjects, were defined as the percentage of sites that had a decrease in bone height where each site was required to exceed the bone loss threshold of 2% divided by the total number of sites that could be compared. Incidence rates and relative risk (RR) for mean alveolar bone loss were also calculated. Radiographic data were not available on all sites for all the years.

Teeth extracted over the 15-year period were identified from the clinical records. Tooth loss was defined as the percentage of teeth initially present at baseline that were subsequently extracted. Subjects with tooth loss was defined as the percentage of subjects who had at least one tooth present at baseline that was subsequently extracted. The incidence rates and RR for tooth loss and the annualized rates of loss of periodontally affected and unaffected teeth were calculated.

Development of the PRC

The PRC is a web-based tool that can be accessed through a dental office computer. We developed the PRC using the six design parameters listed below on a desktop computer using Microsoft Excel[®].

1. PRC calculated risk is for future periodontal disease for those patients who do not yet have it and risk for future progression of periodontal disease for those who already have it.
2. A risk factor is defined as a factor that is part of the causal chain of disease, or exposes the patient to the causal chain, which if present directly increases the probability of

disease occurring and if absent reduces the probability.

3. A risk factor must have a scientific basis that is supported by publication in refereed scientific journals.
4. The application of risk assessment information through the development of treatment recommendations to reduce risk must occur.
5. All requisite information must be obtained during a traditional periodontal examination as performed by dentists in the United States; the time required for data collection and input must fit within the usual time these dentists use for diagnosis.
6. A 5-point risk scale is to be used to balance the sensitivity of risk assessment with the time and cost required to obtain the necessary information.

The calculation of risk is a multi-step process involving mathematical algorithms that use nine risk factors, including:

- patient age;
- smoking history;
- diagnosis of diabetes;
- history of periodontal surgery;
- pocket depth;
- furcation involvements;
- restorations or calculus below the gingival margin;
- radiographic bone height;
- vertical bone lesions.

A 3-point scale is used to document pocket depth and radiographic bone height. An algorithm was developed to quantify disease severity from pocket depth and bone height values. The base risk score is calculated using an algorithm that correlates disease severity with age. The risk score is increased if there is a positive history of periodontal surgery and if the patient smokes more than 10 cigarettes per day, or the patient has diabetes that is poorly controlled. The existence of furcation involvements, vertical bone lesions, or subgingival restorations or calculus increase risk when the risk score is otherwise less than 4. Once development of the PRC was completed, we searched for a data set with which to test its accuracy and validity. The VA Dental Longitudinal Study of Oral Health and Disease data set was selected in part because it contained all of the data required by the PRC and the subject population was virtually untreated during the 15-year period covered by the study. Additional details concerning develop-

ment of the PRC will be forthcoming (Martin & Page 2003).

Statistical analysis

Study subjects were grouped based on risk score calculated by the PRC at baseline (year 0). Only two subjects had a risk score of 1, and hence were not included in any of the statistical analyses. Mean bone loss, sites manifesting bone height worsening and mean tooth loss were compared between the four risk groups separately at 3, 9 and 15 years using one-way analysis of variance (ANOVA). The rate of bone loss and incidence of tooth loss were compared between risk groups at 3, 9 and 15 years using linear and Poisson's regression, respectively, by means of generalized estimating equations (Hujuel et al. 1994). The percentage of subjects with tooth loss was compared between risk groups at 3, 9 and 15 years using χ^2 analysis. When risk group differences were present ($p < 0.05$), all pairwise comparisons between risk groups were performed, and a Bonferroni method was used to adjust the significance level for the multiple comparisons ($k = 6$) on each outcome within each year of follow-up. Rates of mean bone loss and tooth loss including loss of teeth periodontally affected and unaffected at baseline were calculated for baseline to year 3, years 3–9 and years 9–15. For the average subject, 23.3 teeth were present at baseline, although the number of teeth present varied greatly among subjects and among

risk groups. For calculation of the number of teeth lost, the data were normalized by assuming that each subject had 23.3 teeth present at baseline.

Results

Some of the characteristics of the study population have been reported (Page et al. 2002). Subjects were well distributed among the age range of 25–74 years. Periodontitis was present in all age groups as demonstrated by the mean bone loss score, which increased from 2.75 (± 0.53) mm for subjects who were 34 years of age or younger to 3.70 (± 1.00) mm for subjects 60–74 years of age (Page et al. 2002). Among all subjects, at baseline there were 101 smokers, nine diabetics and 42 individuals who reported having had some sort of periodontal treatment. Based on the self-report, the proportion of subjects who had only one or no "gum treatments" over the 15-year period of the study for risk groups 2–5 was 98%, 94%, 94% and 80%, respectively. Risk group 1 contained only two subjects and was excluded from all analyses; the numbers of subjects in risk groups 2–5 were well distributed with numbers in each group ranging from 104 to 193 (Table 1).

Changes in cumulative mean bone loss from baseline for each risk group for years 3, 9 and 15 are shown in Table 1. Mean bone loss, a measure of disease severity, increased for all risk groups from years 3 to 15. There was a strong positive association between risk score

Table 1. Increase in disease severity measured as cumulative mean bone loss by years 3, 9 and 15.

Year	Risk score at baseline	N	Mean* bone loss (%)	SE	ANOVA p-value**
3	2	104	0.7 ^A	0.1	<0.00001
	3	193	1.0 ^{A,B}	0.1	
	4	120	1.3 ^B	0.1	
	5	104	2.5 ^C	0.2	
9	2	104	1.8 ^A	0.1	<0.00001
	3	193	2.4 ^A	0.1	
	4	120	3.4 ^B	0.3	
	5	104	5.0 ^C	0.4	
15	2	104	3.3 ^A	0.2	<0.00001
	3	193	4.0 ^{A,B}	0.3	
	4	120	5.1 ^B	0.5	
	5	104	6.9 ^C	0.5	

N is the number of subjects.

*Within each year, mean values with different superscripts (e.g. A, B and C) are significantly different at the 0.05 level (Bonferroni $p < 0.05$).

**Within each year, the (one-way) ANOVA p-value is for any differences between the risk score groups. When group differences were indicated ($p < 0.05$), pairwise comparisons between the groups were performed using a Bonferroni method to adjust the significance level for the multiple comparisons.

at baseline and disease severity measured as mean bone loss at each year. The rank order of change in mean bone loss for year 3 for risk groups 2–5 from most to least was $5 > 4 > 3 > 2$, and this rank order was maintained throughout the 15 years. The mean bone loss at year 3 ranged from 0.7% in risk group 2 to 2.5% in risk group 5. By year 15, the mean bone loss ranged from 3.3% in the risk group 2 to 6.9% in risk group 5. Within each year, mean values for bone loss for risk group 5 differed from risk group 4 and risk group 4 differed from group 3 for year 9 ($p < 0.05$) (Bonferroni), while groups 2 and 3 did not differ significantly from each other in any of the years. As compared to subjects with a risk score of 2, disease severity measured as mean bone loss by years 3, 9 and 15 increased 1.2- to 1.4-fold for group 3, 1.5- to 1.9-fold for group 4, and 2.1- to 3.6-fold for group 5.

Additional calculations were performed to compare the incidence rates of bone loss for the three time intervals. As shown in Table 2, the rates of bone loss among risk groups from baseline to year 3 and between years 3 and 9 were significantly different ($p < 0.0001$), while the rates were similar among groups from years 9 to 15 ($p = 0.85$). For risk groups 3–5, there was a striking decrease in the rates of mean alveolar bone loss for all the three groups from the first time interval to the second and, with one exception, from the second to the third. The most dramatic decrease was for group 5, with a decrease from 0.0083 during the first time interval to 0.0043 for the second and 0.0031 for the third.

The change in disease extent, measured as the increase in the percentage of sites with bone loss exceeding the 2% threshold, is illustrated in Figure 1. The risk scores calculated at baseline were very strongly associated with the increase in disease extent over the entire 15-year period. The rank order was group $5 > 4 > 3 > 2$, and it was maintained through year 15. In contrast to disease severity, the slopes of the curves for increasing extent of disease for all of the risk groups except group 2 decreased somewhat from years 9–15 relative to years 3–9. This decrease likely results from extraction of the most severely affected teeth prior to year 9. The within-group values for group 5 differed significantly from group 4 at year 3 ($p < 0.05$), while risk groups 2 and 3 did not differ from one another at any year.

Table 2. Rate of mean alveolar bone loss per year for each time interval

Interval	Risk score at baseline	Rate (mean loss per year)*	SE	Linear regression, p -value**	
Baseline to year 3	2	0.0023 ^A	0.0005	<0.0001	
	3	0.0033 ^{A,B}	0.0005		
	4	0.0043 ^B	0.0005		
	5	0.0083 ^C	0.0012		
Years to year 9	2	0.0018 ^A	0.0006		<0.0001
	3	0.0023 ^A	0.0006		
	4	0.0035 ^B	0.0007		
	5	0.0043 ^B	0.0005		
Year 9 to year 15	2	0.0025	0.0004		
	3	0.0027	0.0004		
	4	0.0028	0.0005		
	5	0.0031	0.0007		

*Within each year, rates with different superscripts (e.g. A, B and C) are significantly different at the 0.05 level (Bonferroni $p < 0.05$).

**Within each time interval, the GEE linear regression is for any differences between the risk score groups. When group differences were indicated ($p < 0.05$), pairwise comparisons between the groups were performed using a Bonferroni method to adjust the significance level for the multiple comparisons.

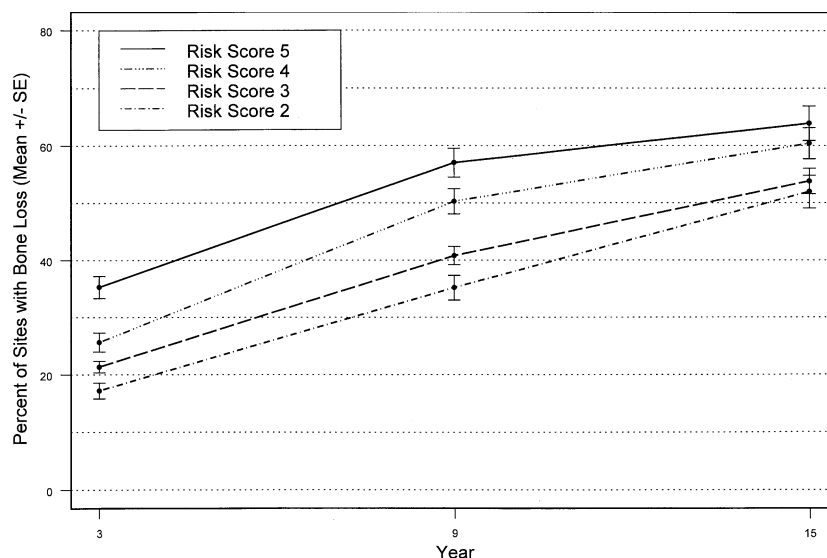


Fig. 1. Mean values with SE for percentage of sites with bone height worsening from baseline for years 3, 9 and 15. Group 2 differed from group 4 and group 4 differed from group 5 at $p < 0.05$, but group 3 did not differ significantly from groups 2 and 4.

The association between risk score at baseline and total tooth loss over the 15-year period was positive and strong (Table 3). The mean percent total tooth loss for group 5 at year 3 (4.9%) was almost 10-fold greater than that for group 2 (0.5%). The mean percent total tooth loss increased over time within each group, especially group 5, and by year 15 ranged from 3.1% in risk group 2 to 23.7% in risk group 5. At all years, the rank order of change in percent tooth loss from most to least was risk group $5 > 4 > 3 > 2$. Within each year, group 5 had more tooth loss than group 4, group 2 had the least tooth loss, and groups 3 and 4 did not differ from each

other at a 0.05 significance level (Bonferroni).

Tooth loss from baseline was also calculated separately for teeth that were and were not periodontally affected at baseline (Figure 2). Approximately 74% of the teeth extracted over the 15-year period met the criteria for being periodontally affected at baseline. Risk scores were accurate predictors of loss of teeth and teeth periodontally affected but not those periodontally unaffected at baseline. Loss of teeth that were periodontally affected at baseline for all risk groups increased linearly over the 15-year period (Figure 3). Rank order for loss of these teeth at year 3 was risk

Table 3. Percent tooth loss from baseline by years 3, 9 and 15

Year	Risk score at baseline	N	Mean* tooth loss (%)	SE	p-value**	
3	2	104	0.5 ^A	0.1	<0.00001	
	3	193	1.6 ^B	0.4		
	4	120	2.1 ^B	0.5		
	5	104	4.9 ^C	1.0		
9	2	104	1.5 ^A	0.3		
	3	193	4.9 ^B	0.8		
	4	120	6.9 ^B	1.0		
	5	104	14.4 ^C	2.0		
15	2	104	3.1 ^A	0.7		<0.00001
	3	193	8.4 ^B	1.0		
	4	120	11.1 ^B	1.3		
	5	104	23.7 ^C	2.4		

N is the number of subjects.

*Within each year, mean values with different superscripts (e.g. A, B and C) are significantly different at the 0.05 level (Bonferroni $p < 0.05$).

**Within each year, the (one-way) ANOVA p -value is for any differences between the risk score groups. When group differences were indicated ($p < 0.05$), pairwise comparisons between the groups were performed using a Bonferroni method to adjust the significance level for the multiple comparisons.

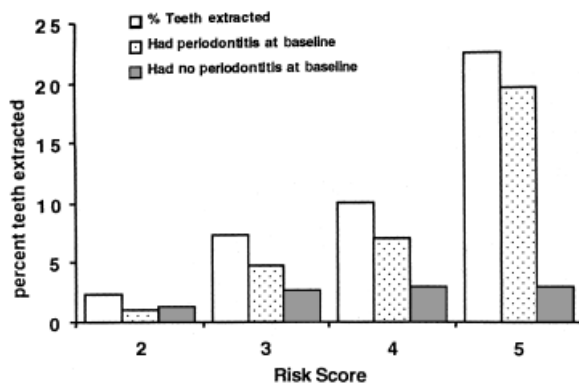


Fig. 2. Percentage of teeth present that were extracted (open bars); percentage of all teeth extracted that had (dotted bars) or did not have (stippled bars) periodontitis at baseline for risk groups 2–5.

group 5 > 4 > 3 > 2, and rank order was maintained at years 9 and 15. There was a strong association between loss of periodontally affected teeth and risk score for all years. Tooth loss for risk group 5 was significantly more than for group 4 for all years ($p < 0.05$), and values for group 3 were significantly greater than for group 2 at years 9 and 15 ($p < 0.05$).

The incidence rates of tooth loss, based on the number of teeth lost and the time at risk for each tooth, were calculated using Poisson's regression as described by Hujoel et al. (1994). Before calculating the common incidence rate over 15 years, the data were tested to determine if the incidence rates differed for the three time intervals (for tooth loss $p = 0.99$, and for loss of teeth periodontally affected or not affected at baseline $p = 0.99$ and 0.57, respec-

tively). The incidence rate of tooth loss appeared to be constant within each risk group over the 15 years. The incidence rate of total tooth loss and loss of periodontally affected and unaffected teeth per tooth year and for 15 tooth years for each risk group are shown in Table 4. The average subject had 23.3 teeth at baseline. Of these, tooth loss over 15 tooth years for group 2 was 0.5 teeth compared to 5.8 teeth for group 5. The incidence rate of total tooth loss and loss of teeth periodontally affected at baseline increased in rank order with increasing risk score. Of the 5.8 teeth lost, 4.9 were periodontally affected at baseline and 0.8 were not. While group 2 had approximately the same annual rate of loss of periodontally affected as unaffected teeth, the annual rate of periodontally affected teeth for group 5 was more than six-fold greater than

for periodontally unaffected teeth (Fig. 2, Table 4). Compared to risk group 2, RRs for tooth loss for groups 3–5 are shown in Table 5. It is notable that RRs for teeth that were periodontally affected and unaffected at baseline were 22.7 and 1.9, respectively.

Percentages of subjects who lost one or more teeth that were periodontally affected at baseline, a measure of the distribution of advancing disease among subjects within each risk group, are shown in Fig. 4. The rank order at year 3 was risk group 5 > 4 > 3 > 2, and this rank order was maintained at years 9 and 15. The percentage of subjects losing one or more teeth by year 3 was higher for the risk score group 5 as compared to the other groups, and values for all the four groups differed significantly from one another at both years 9 and 15 ($p < 0.05$). The slopes of curves for risk groups 4 and 5 decreased from years 9 to 15 relative to values from years 3 to 9, possibly because of extraction of the most severely affected teeth prior to year 9. At year 15, the percent of subjects who lost one or more periodontally affected teeth increased linearly from 20.2% for risk group 2 to 83.7% for risk group 5 (Fig. 4). Relative to subjects with a risk score of 2, the loss of one or more periodontally affected teeth for years 3, 9 and 15 was 2.1- to 2.6-fold greater for a risk score of 3, 2.6- to 4.1-fold greater for a risk score of 4, and 4.1- to 6.0-fold greater for a risk score of 5.

The curves for the percentage of individuals who lost one or more teeth regardless of periodontal status of the teeth lost were almost identical to those in Fig. 4, except that the values, especially for group 5, were somewhat higher (data not shown). As compared to subjects with a risk score of 2, the RR of subjects for any tooth loss ranged from 1.6 to 1.7 for a risk score of 3, 2.0 to 2.5 for a risk score of 4, and 2.3 to 3.5 for a risk score of 5.

About 24% of subjects in group 2 compared to approximately 37% in groups 3–5 lost one or more teeth unaffected by periodontal disease at baseline, and percentages were not related to risk score (data not shown). Furthermore, the percentage loss of periodontally unaffected teeth (Fig. 2) and the incidence rate of their loss over the 15-year study period (Table 4) were not predicted by risk score.

Risk scores calculated at baseline were not reliable predictors of increased

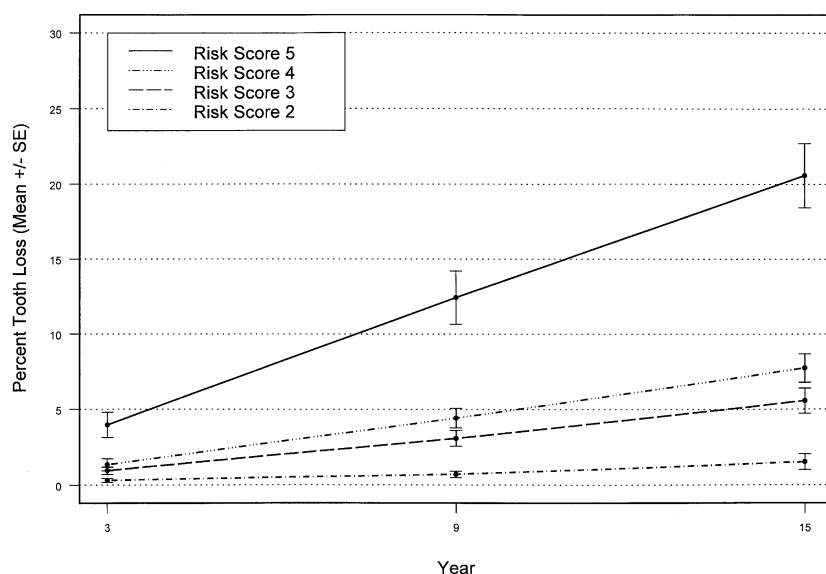


Fig. 3. Percent tooth loss from baseline with SE for teeth that were periodontally affected at baseline for risk groups 2–5 at years 3, 9 and 15. Since 74% of teeth extracted were periodontally affected at baseline and loss of periodontally unaffected teeth was not related to risk score group, curves for percent tooth loss from baseline very closely resembled those in this figure, except that values were somewhat higher (data not shown).

Table 4. Incidence rates of tooth loss from baseline

	Risk score at baseline	Rate of tooth loss per 1 tooth year (95% CI)	Mean number of teeth lost
Total teeth	2	0.0016 ^A (0.0011–0.0021)	0.5
	3	0.0051 ^B (0.0040–0.0064)	1.8
	4	0.0070 ^B (0.0055–0.0088)	2.4
	5	0.017 ^C (0.013–0.021)	5.8
Teeth periodontally affected	2	0.0007 ^A (0.0004–0.0010)	0.2
	3	0.0033 ^B (0.0025–0.0043)	1.2
	4	0.0049 ^B (0.0038–0.0062)	1.7
	5	0.014 ^C (0.011–0.018)	4.9
Teeth periodontally unaffected	2	0.0009 ^A (0.0006–0.0013)	0.3
	3	0.0018 ^B (0.0014–0.0024)	0.6
	4	0.0021 ^B (0.0014–0.0030)	0.7
	5	0.0022 ^B (0.0016–0.0031)	0.8

Tooth loss rates differed significantly between risk groups from baseline to year 15 with $p < 0.0001$ for total teeth and teeth periodontally affected at baseline, and $p = 0.0011$ for teeth periodontally unaffected at baseline. When group differences were indicated, pairwise comparisons between groups were performed using a Bonferroni method to adjust the significance level for multiple comparisons. Tooth loss rates with different superscripts (e.g. A, B and C) were significantly different at the $p < 0.05$ level.

Table 5. Relative risk for tooth loss*

	Risk group		
	3	4	5
Any tooth loss	3.2 (2.2, 4.8)	4.5 (3.0, 3.6)	10.6 (7.2, 15.6)
Loss of affected teeth	5.5 (2.7, 11.0)	8.1 (4.2, 15.7)	22.7 (11.8, 43.7)
Loss of nonaffected teeth	1.5 (0.8, 2.9)	1.3 (0.6, 2.9)	1.9 (0.9, 3.9)

*Reported as relative risk (95% confidence limit).

pocket depth over time in this population except at year 3 (Fig. 5). The rank order of values for the percent of sites

with pocket depth worsening at year 3 was risk group $5 > 4 > 3 > 2$. The differences between the risk groups at year 3

were small, but the association between risk score and change in pocket depth was significant ($p < 0.0002$). Values increased for all risk groups except group 5 from year 3 to years 9 and 15. By year 9, group differences were not statistically significant ($p < 0.36$) because of failure of the mean score for risk group 5 to increase. At year 15, the rank order was reversed relative to year 3 with risk group $5 < 4 < 3 = 2$.

Discussion

The purpose of the present study was to test the accuracy and validity of risk scores calculated using the PRC as predictors of periodontal status relative to actual outcomes 3, 9 and 15 years later. The methods used to determine changes in periodontal status over time were radiographic assessment of alveolar bone status using digitized radiographs, probing pocket depth and tooth loss. The increase in mean radiographic alveolar bone loss and in tooth loss was used as a measure of disease severity, and the percentage of sites manifesting radiographic alveolar bone loss was used as a measure of increasing disease extent. Assessment of changes in alveolar bone status using digitized radiographs has a high level of sensitivity (Jeffcoat et al. 1996), and loss of teeth is a definitive outcome measure of deteriorating oral health. Deterioration in periodontal disease status of the population over time was evaluated as the increase in percentages of individuals losing one or more teeth. This group of measurements is generally accepted as appropriate for assessing periodontal status and changes in status occurring over time.

The PRC separated subjects into risk groups 1–5, each of which contained more than 100 subjects except for risk group 1 which had only two subjects. Whether measured as changes in disease severity (increasing mean alveolar bone loss and tooth loss), changes in disease extent in individuals (increase in the proportion of affected teeth) or in the study population (increase in the percentage of subjects losing one or more teeth), PRC scores distinguished among groups with differing levels of risk. A high degree of accuracy was also demonstrated whether alveolar bone loss or tooth loss was expressed in terms of incidence rates or RR. RR for tooth loss was 10.6-fold greater, and loss of teeth periodontally affected at

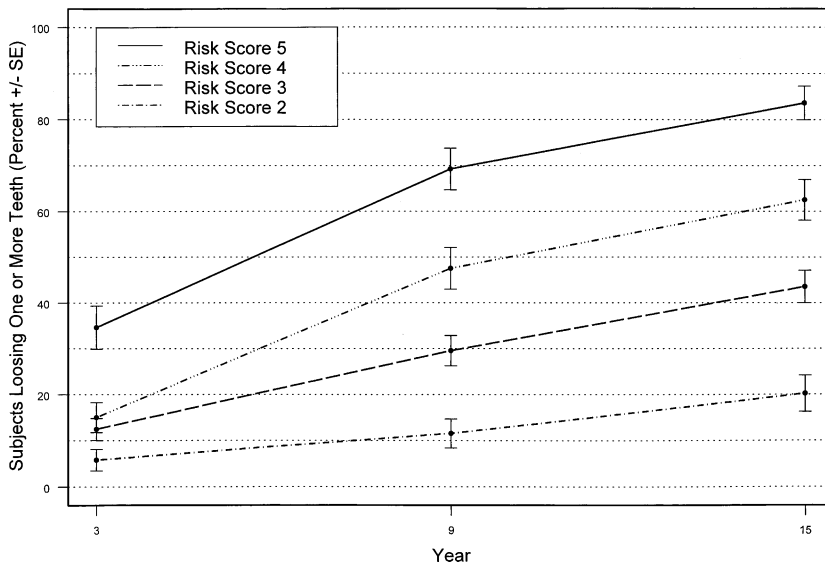


Fig. 4. Percent of subjects with SE who lost one or more teeth that were periodontally affected at baseline for risk groups 2–5 at years 3, 9 and 15.

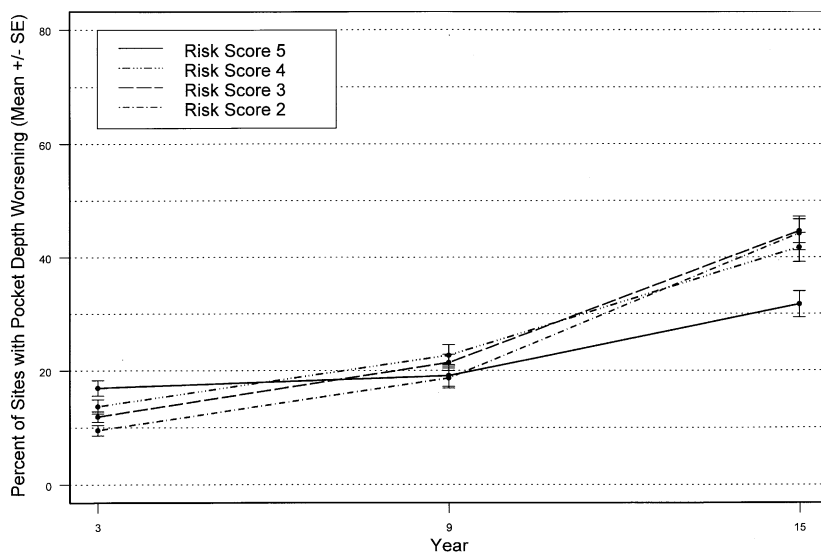


Fig. 5. Mean values with SE for percent of sites with pocket depth worsening from baseline for risk groups 2–5 at years 3, 9 and 15.

baseline was 22.7-fold greater for group 5 than for group 2. Regardless of the disease characteristic measured (except for pocket depth), the risk groups were in rank order at year 3 with risk for group 5 > 4 > 3 > 2, and this rank order was maintained for the entire 15-year study period. At year 15, 83.7% of subjects in risk group 5 had lost one or more periodontally affected teeth compared to only 20.2% of subjects in risk group 2. Our data show that risk scores determined using the PRC and information gathered during a routine examination are accurate and valid predictors of

future periodontal deterioration as determined by actual changes in periodontal status measured as alveolar bone loss and tooth loss that occurred up to 15 years later.

Risk scores determined by the PRC on the basis of baseline data were not a reliable predictor of future increases in pocket depth in our study population. In populations of periodontitis patients, loss of attachment and loss of alveolar bone height generally increase with aging, but pocket deepening does not parallel these changes because of gingival recession (Yoneyama et al. 1988).

Consequently, pocket depth measurement is not a good indicator of deteriorating periodontal status past middle age. Furthermore, the method of measurement used to record pocket depth clouds interpretation of the data. Pocket depth measurements were recorded for each tooth as ordinal numbers, with the highest number on the scale assigned to pockets with depths ≥ 5 mm. Thus, pockets deeper than 5 mm at baseline were not recorded and increases in the depth of pockets to values > 5 mm after the baseline measurement could not be recorded. These factors and the loss of teeth with the greatest pocket depth in risk group 5 and to a lesser extent in group 4 could account for an apparent slower increase in pocket depth in these two risk groups as seen in Fig. 5.

A major strength of this study was the nature of the subject population. All data required by the PRC for making risk predictions, including radiographs, were available and actual periodontal outcomes were known for a period of 15 years. The size of the population was sufficiently large to have adequate numbers of subjects present in each risk group at baseline and over the entire 15-year period. The population consisted of males only. However, study outcome should be independent of gender since we measured the strength of the association between risk prediction and actual outcome, and that would be expected to be comparable for both genders. Since the onset and progress of periodontitis in males is known to be greater than in females, the use of males in contrast to females or both genders was a benefit in that the scale of disease onset and progression would be expected to be greater.

Our data set did not include the measurement of loss of connective tissue attachment. Since loss of attachment measurements are generally not performed in day-to-day clinical practice as a part of standard periodontal examination, such measurements are not required by the PRC. Periapical radiographs with bitewings were available and they were of sufficient quality to permit analysis of change using digitized films (Jeffcoat et al. 1984). For alveolar bone loss change, we required change greater than a threshold value of 2% in order to reduce the potential effect of technical error.

We defined the criteria for identity of periodontally affected teeth at baseline as teeth that were present and mani-

fested either ≥ 5 mm pocket depth or alveolar bone height of > 2 mm from the CEJ. Although these criteria could result in an under- or over-count, they appear to be valid since their use permitted us to demonstrate that risk scores were very strong predictors of loss of teeth periodontally affected at baseline but not teeth that were unaffected. The validity of the criteria is also supported by the observed parallel relationship between increasing bone loss and tooth loss over time, especially in the high-risk groups, and the remarkable difference in loss of periodontally affected and unaffected teeth.

The VA Normative Study of Aging did not include a dental treatment component. Subjects enrolled in the study received treatment only if they chose to do so. Based on the self-reports of treatment, the proportions of subjects who had no or only one gum treatment during the 15-year course of the study for risk groups 2–5 were 98%, 94%, 94% and 80%, respectively. Because of the small numbers of subjects who received treatment, our observations are essentially for an untreated population. We do not know the effects of treatment on the outcome of the risk predictions. It is now important to conduct comparable studies on subjects who have had therapy.

It is notable that the increase in mean alveolar bone loss over the 15-year period of our study did not appear to increase linearly. Instead, for groups 3–5, the apparent rate for alveolar bone loss was highest from baseline to year 3, lower from years 3 to 9, and lowest from years 9 to 15. This was especially true for group 5 and to a lesser extent for group 4. This apparent slowing of the rate of bone loss in the absence of therapy was unexpected, and it is inconsistent with knowledge about the natural history of periodontitis. In contrast to mean alveolar bone loss, tooth loss occurred at a constant rate for all risk groups over the 15-year period, and most of the teeth extracted (74.6%) were periodontally affected at baseline. It is reasonable to expect that the periodontally affected teeth extracted were those with the most advanced bone loss. Since extraction removes these teeth from the mean alveolar bone loss measurement, increases in mean alveolar bone loss could have occurred but would have been masked by tooth extraction. This observation will take on added importance in the interpretation of results

of future studies of the effects of therapy on change in periodontal status and risk.

In summary, our data show that highly valid and accurate predictions of risk for future periodontal deterioration as measured by change in alveolar bone status and tooth loss can be made using the PRC and information gathered during a traditional dental/periodontal examination. No laboratory test results are required. The association between the assigned risk prediction and the actual periodontal deterioration observed over a period of 15 years was unusually strong with probability values < 0.0001 . The PRC will provide dentists with a new tool for accurately assessing risk, and it also generates suggested treatment options for minimizing future risk and for repair of existing damage. Use of the PRC and suggested treatment options over time may be expected to result in more uniform decision-making about periodontal disease, a reduction in disease incidence, improved oral health and a significant reduction in the need for complex periodontal treatment and the cost of care (Axelsson et al. 1991, 2000, Fors & Sandberg 2001).

Acknowledgements

Supported in part by Dental Medicine International, Inc. (Philadelphia, PA).

The VA Dental Longitudinal Study, a component of the Massachusetts Veterans Epidemiology Research & Information Center, is supported by the VA CSP/ERIC program and by VA Medical Research Service, U.S. Department of Veterans Affairs. Dr. Garcia was recipient of a Career Development Award from the VA HSR&D Service and is supported by NIDCR Grant K24 DE00419. We acknowledge the assistance of Mohamed Hamed (BDS, DMedSc, MPH) in the development of the analytic data set.

Zusammenfassung

Langzeit-Validierung eines Risikorechners für Parodontale Erkrankungen

Grundlagen: Die Risikobestimmung und die Verwendung ihrer Ergebnisse sind wichtige Komponenten der Prävention, Diagnose und Behandlung von parodontalen Erkrankungen. Die Risikobestimmung ist für die Zahnmedizin relativ neu. Gegenwärtig wird das Risiko durch eine subjektive Evaluation bestimmt und die Ergebnisse zeigen zwischen den Klinikern eine große Variation. Für eine objektive und quantitative Beurteilung haben wir ein Computergestütztes Risikoberechnungsprogramm en-

twickelt, den Periodontal Risk Calculator (PRC). Der Zweck der Studie über die hier berichtet wird, war es, die Genauigkeit und Validität dieses Werkzeugs zu evaluieren.

Methoden: Von 523 Personen, welche an der VA zahnmedizinischen Langzeitstudie zur oralen Gesundheit und Krankheit teilnahmen, die eine Periode von 15 Jahren umfaßte, wurden klinische Befunde und Röntgenbilder verwendet. Die Informationen der Ausgangsuntersuchung wurden in den Risikorechner eingegeben und für jede Person wurde ein Risikowert auf einer Skala von 1 bis 5 für die parodontale Verschlechterung berechnet. Der aktuelle Parodontalstatus wurde bezüglich des Alveolarknochenabbaus mittels digitaler Röntgenaufnahmen bestimmt und der Zahnverlust wurde über die klinischen Befundaufzeichnungen nach 3, 9 und 15 Jahren bestimmt. Die Stärke der Assoziation zwischen der Risikovorhersage und dem aktuellen Ergebnis wurde statistisch bestimmt.

Ergebnisse: Die Risikowerte waren starke vorhersagende Faktoren für den zukünftigen Parodontalstatus, was an der sich vergrößernden Schwere und dem Ausmaß des Alveolarknochenabbaus und dem Zahnverlust, insbesondere der parodontal befallenen Zähne, gemessen wurde. Während der kompletten 15-jährigen Periode bildeten die Risikowerte übereinstimmend Gruppen von geringsten zum größten Knochenabbau und Zahnverlust. Die Risikogruppen unterschieden sich stark untereinander. Im 3. Jahr war die Inzidenzrate für Knochenabbau bei der Gruppe-5 3.7-fach höher als bei Gruppe-2 und im 15. Jahr war der Verlust von parodontal befallenen Zähnen 22.7-fach höher, als für Gruppe-2 ($p < 0.001$). Im 15. Jahr hatten 83.7% der Personen der Risikogruppe-5 einen oder mehrere parodontal befallene Zähne verloren, verglichen mit 20.2% der Personen in Gruppe-2.

Schlussfolgerungen: Risikowerte die mit dem PRC berechnet werden und Informationen, die während einer parodontalen Standarduntersuchung erhoben werden erlauben mit einem hohen Niveau an Genauigkeit und Validität die Vorhersage des zukünftigen Parodontalstatus. Man könnte erwarten, dass die Verwendung des Risikorechners, im Laufe der Zeit, in einer einheitlicheren und genaueren klinischen parodontalen Entscheidungsfindung, verbesserter Mundgesundheit, Reduktion der Notwendigkeit für komplexe Therapie und einer Reduktion der Krankheitskosten resultiert.

Résumé

Validation longitudinale d'un calculateur de risque pour la maladie parodontale

L'évaluation du risque et l'utilisation des résultats sont des composants importants de la prévention, du diagnostic et du traitement de la maladie parodontale. Cette évaluation est relativement nouvelle en médecine dentaire. Actuellement le risque est estimé par une évaluation subjective et les résultats varient énormément parmi les cliniciens. Un outil d'évaluation du risque basé sur ordinateur, le Periodontal Risk Calculator (PRC) pour l'évaluation objective et quantitative du risque a été

développé. Le but de l'étude présente a été de mesurer la valeur et la précision de cet outil. Des données cliniques et radiographiques de 523 sujets enrôlés dans une étude sur la santé et la maladie buccale longitudinale chez des vétérans de l'armée couvrant une période de quinze années ont été utilisées. L'information de départ a été rentrée dans le calculateur de risque et un score de risque sur une échelle de un à cinq pour la détérioration a été calculé pour chaque sujet. L'état parodontal actuel en terme de perte osseuse alvéolaire a été déterminé par radiographies et la perte dentaire déterminée par des données cliniques qui ont été relevées après trois, neuf et quinze années. La force de l'association entre la prédiction du risque et l'état actuel a été déterminée statistiquement. Les scores du risque étaient des signes forts de l'état parodontal futur mesurés en tant que dégradation et étendue des pertes osseuses alvéolaires et dentaires, spécialement les dents touchées par la parodontite. Sur l'entièreté de la période de quinze années les scores de risque classaient constamment les groupes des plus faibles au plus fortes perte dentaires. Les groupes à risque étaient très différents les uns des autres. Après trois années, le taux d'incidence de perte osseuse du groupe 5 était de 3.7 fois plus important que pour le groupe 2, et après 15 années la perte des dents touchées par la parodontite 22.7 fois supérieure à celle trouvée dans le groupe 2 ($p < 0.001$). Après quinze années 83.7% des sujets du groupe à risque 5 avaient perdu une ou plusieurs dents touchées par la parodontite comparé à 20.2% des sujets du groupe 2. Les scores de risque calculés par PRC et l'information obtenue durant un examen parodontal standard prédisent l'état parodontal futur avec des niveaux de précision et de valeur élevés. L'utilisation de l'outil d'estimation du risque avec le temps semble apporter en une décision clinique plus uniforme et plus précise, augmente la santé buccale et réduit la nécessité de traitements complexes et donc le coût des soins de santé.

References

- Axelsson, P., Lindhe, J. & Nystrom, B. (1991) On the prevention of caries and periodontal disease. Results of a 15-year longitudinal study in adults. *Journal of Clinical Periodontology* **18**, 182–189.
- Axelsson, P., Paulander, J., Svardstrom, G. & Kaijser, H. (2000) Effects of population based preventive programs on oral health conditions. *Le Journal de Parodontologie et d'Implantologie Orale* **19**, 255–269.
- Beck, J. D. (1998) Risk assessment revisited. *Community Dentistry and Oral Epidemiology* **26**, 220–225.
- Beck, J. D., Koch, G. G. & Offenbacher, S. (1995) Incidence of attachment loss over 3 years in older adults. New and progressing lesions. *Community Dentistry and Oral Epidemiology* **23**, 291–296.
- Beck, J. D., Koch, G. G., Rozier, R. G. & Tudor, G. E. (1990) Prevalence and risk indicators for periodontal attachment loss in a population of older community-dwelling blacks and whites. *Journal of Periodontology* **61**, 521–528.
- Feldman, R. S., Douglass, C. W., Loftus, E. R., Kapur, K. K. & Chauncy, H. H. (1982) Interexaminer agreement in the measurement of periodontal disease. *Journal of Periodontal Research* **17**, 80–89.
- Fleiss, J. L. & Chilton, N. W. (1983) The measurement of interexaminer agreement on periodontal disease. *Journal of Periodontal Research* **18**, 601–606.
- Fors, U. G. H. & Sandberg, H. C. H. (2001) Computer-aided management – a software tool for the Hidep model. *Quintessence International* **32**, 309–320.
- Glass, R. L., Loftus, E. R., Kapur, K. K. & Alman, J. E. (1973) Analysis of components of periodontal disease. *Journal of Dental Research* **52**, 1238–1244.
- Hart, T. C. & Kornman, K. S. (1997) Genetic factors in the pathogenesis of periodontitis. *Periodontology 2000* **14**, 202–215.
- Hirschfeld, L. & Wasserman, B. (1978) A long-term survey of tooth loss in 600 treated patients. *Journal of Periodontology* **49**, 225–237.
- Hujoel, P. P., Isokangas, P. J., Tiekso, J., Davis, S., Lamont, R. J., DeRouen, T. A. & Makinen, K. K. (1994) A re-analysis of caries rates in a preventive trial using Poisson regression models. *Journal of Dental Research* **73**, 573–579.
- Ismail, A. I., Morrison, C. E., Burt, B. A., Caffesse, R. G. & Kavanagh, M. T. (1990) Natural history of periodontal disease in adults: findings from the Tecumseh Periodontal Disease Study, 1959–1987. *Journal of Dental Research* **69**, 430–435.
- Jeffcoat, M. K., Jeffcoat, R. L. & Williams, R. C. (1984) A new method for the comparison of bone loss measurement on non-standardized radiographs. *Journal of Periodontal Research* **19**, 434–440.
- Jeffcoat, M. K., Reddy, M. S., Magnusson, I., Johnson, B., Meredith, M. P., Cavanaugh, P. F., Jr. & Gerlach, R. W. (1996) Efficacy of quantitative digital subtraction radiography using radiographs exposed in a multicenter trial. *Journal of Periodontal Research* **31**, 157–160.
- Jenkins, W. M. M., MacFarlane, T. W. & Gilmour, W. H. (1988) Longitudinal study of untreated periodontitis: (I) Clinical findings. *Journal of Clinical Periodontology* **15**, 324–330.
- Kapur, K. K., Glass, R. L., Loftus, E. R., Alman, J. E. & Feller, R. P. (1972) The Veterans Administration longitudinal study of oral health and disease. *Aging and Human Development* **4**, 125–137.
- Kornman, K. S., Crane, A., Wang, H.-Y., di Giovine, F. S., Newman, M. G., Pirk, F. W., Wilson, T. G., Jr., Higginbottom, F. L. & Duff, G. W. (1997) The interleukin-1 genotype as a severity factor in adult periodontal disease. *Journal of Clinical Periodontology* **24**, 72–77.
- Lang, N. P., Joss, A., Orsanic, T., Gusberti, F. A. & Siegrist, B. E. (1986) Bleeding on probing. A predictor for the progression of periodontal disease? *Journal of Clinical Periodontology* **13**, 590–596.
- Lindhe, J., Haffajee, A. D. & Socransky, S. S. (1983) Progression of periodontal disease in adult subjects in the absence of periodontal therapy. *Journal of Clinical Periodontology* **10**, 433–442.
- Lindhe, J., Okamoto, H., Yoneyama, T., Haffajee, A. & Socransky, S. S. (1989) Longitudinal changes in periodontal disease in untreated subjects. *Journal of Clinical Periodontology* **16**, 662–670.
- McFall, W. T., Jr. (1982) Tooth loss in 100 treated patients with periodontal disease. A long-term study. *Journal of Periodontology* **53**, 539–549.
- Michalowicz, B. S., Diehl, S. R., Gunsolley, J. C., Sparks, B. S., Brooks, C. N., Koertge, T. E., Califano, J. V., Burmeister, J. A. & Schenkein, H. A. (2000) Evidence of a substantial genetic basis for risk of adult periodontitis. *Journal of Periodontology* **71**, 699–1707.
- Page, R. C. & Beck, J. D. (1997) Risk assessment for periodontal diseases. *International Dental Journal* **47**, 61–87.
- Page, R. C., Krall, E. A., Martin, J., Mancl, L. & Garcia, R. I. (2002) Validity and accuracy of a risk calculator in predicting periodontal disease. *Journal of the American Dental Association* **133**, 569–576.
- Page, R. C., Offenbacher, S., Schroeder, H. E., Seymour, G. J. & Kornman, K. K. (1997) Advances in the pathogenesis of periodontitis: summary of developments, clinical implications and future directions. *Periodontology 2000* **14**, 216–248.
- Persson, G. R., Mancl, L., Martin, J. & Page, R. C. (2003) Assessment of risk for periodontal disease by expert clinicians relative to assessment using a computerized risk assessment tool. *Journal of the American Dental Association* **134**, 575–582.
- Salvi, G. E., Lawrence, H. P., Offenbacher, S. & Beck, J. D. (1997) Influence of risk factors on the pathogenesis of periodontitis. *Periodontology 2000* **14**, 173–201.
- Yoneyama, T., Okamoto, H., Lindhe, J., Socransky, S. S. & Haffajee, A. D. (1988) Probing depth, attachment loss and gingival recession. Findings from a clinical examination in Ushiku, Japan. *Journal of Clinical Periodontology* **15**, 581–591.

Address:

Roy C. Page

Regional Clinical Dental Research Center
Box 357480/B-530 Health Sciences Bldg.

University of Washington School of Dentistry

1959 NE Pacific Street

Seattle, WA 98195, USA

Fax: +1 206 685 8024

E-mail: roypage@u.washington.edu