

## COVER STORY

# Validity and accuracy of a risk calculator in predicting periodontal disease

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For most of the 20th century, dental caries and periodontal diseases were prevalent in the United States and many other countries, and dental practice consisted mostly of dealing with the ravages of these diseases and their pathological consequences. Until 15 to 20 years age, knowledge about these diseases was limited, and the pathogenesis and etiology were not well-understood.

**Use of the risk assessment tool over time may result in more uniform and accurate periodontal clinical decision-making.**

Management was based on the "repair" model of care, and the clinician's goal was to diagnose the problem and resolve it via treatment. Dentistry was essentially a surgical discipline. Therapy was empirical and basically the same treatments were administered to all patients. The idea that host factors are a major determinant of disease onset and progression and that risk and susceptibility vary greatly from one

person to another had not been conceived. Disease prevention was neither understood nor practiced. Consequently, repairs were made, but caries and periodontitis generally recurred or progressed unabated.

Within the last two decades, our understanding has grown greatly. As a consequence, management of the major dental diseases is undergoing a transition from the repair model to the medical or wellness model of patient care. (The wellness model guides the clinician and patient toward a health care strategy based on risk reduction and disease prevention.) It is clear that the risk of periodontal disease varies greatly from one

**Background.** Research on the pathobiology of periodontal diseases has increased our knowledge of these diseases and is fostering a transition from the repair model to the medical or wellness model of periodontal care. Successful application of the wellness model depends on an accurate and valid assessment of disease risk, as well as institution of risk reduction as an integral part of prevention and treatment. A computer-based risk assessment tool has been developed.

**Methods.** The authors reviewed clinical records and radiographs of 523 subjects enrolled in the Veterans Affairs Dental Longitudinal Study to evaluate the validity of risk prediction using the computer-based tool. Data from baseline examinations was entered into the risk calculator, and a risk score on a scale from 1 (lowest risk) to 5 (highest risk) was calculated for each subject to predict periodontal deterioration. Actual periodontal status in terms of alveolar bone loss (determined from digitized radiographs) and tooth loss (determined from clinical records) was assessed at years 3, 9 and 15. The authors determined the statistical strength of the association between risk prediction and actual outcome.

**Results.** The risk scores were strong predictors of periodontal status, as measured by alveolar bone loss and loss of periodontally affected teeth. Risk scores consistently ranked risk score groups from least to most bone loss and tooth loss. Compared with a risk score of 2, the relative risk of tooth loss was 3.2 for a risk score of 3, 4.5 for a risk score of 4 and 10.6 for a risk score of 5.

**Conclusions and Practice Implications.** Use of the risk assessment tool over time may result in more uniform and accurate periodontal clinical decision-making, improved oral health, reduction in the need for complex therapy, reduction in health care costs and a hastening of the transition from a repair model to a wellness model of care.

TABLE 1

BASELINE POPULATION CHARACTERISTICS.					
AGE (YEARS)	NUMBER OF SUBJECTS	MEAN ( $\pm$ SD*) BONE LOSS (mm $\dagger$ )	SMOKER (n)	GINGIVAL TREATMENT (n)	DIABETES $\ddagger$ (n)
$\leq 34$	20	2.75 ( $\pm$ 0.53)	10	0	0
35-39	60	2.90 ( $\pm$ 1.05)	12	2	0
40-44	101	3.28 ( $\pm$ 1.12)	28	10	1
45-49	122	3.21 ( $\pm$ 0.83)	25	8	1
50-59	183	3.56 ( $\pm$ 1.11)	24	18	7
60-74	37	3.70 ( $\pm$ 1.00)	2	4	0
<b>TOTAL</b>	<b>523</b>	— $\S$	<b>101</b>	<b>42</b>	<b>9</b>

\* SD: Standard deviation.  
 $\dagger$  mm: Millimeters.  
 $\ddagger$  Subjects with blood glucose levels greater than 126 milligrams per deciliter were judged to be diabetic.  
 $\S$  Not applicable.

person to another,<sup>1-9</sup> and many characteristics and factors have been identified that place people at enhanced risk.<sup>10-14</sup> Development and use of the concept of risk, as well as identification of various risk factors and indicators, are providing the basis for the transition from the repair model to the wellness model.

Identifying risk factors and indicators, as well as undertaking measures that maximally reduce risk, are the hallmarks of the wellness model of care. The ultimate goal is to maintain oral health and to prevent the onset of any form of periodontal disease. The wellness model is new to dentistry and to periodontics. While application of the model requires an accurate and valid assessment of risk, most general dentists and periodontists are not experienced in assessing risk or in using interventions aimed at reducing risk in regard to periodontal diseases.

We have developed a computer-based tool—the periodontal risk calculator, or PRC (Dental Medicine International Inc., Philadelphia)—for assessing risk and predicting periodontal deterioration. The PRC is based on mathematically derived algorithms that assign relative weights to the various known risks that enhance patients' susceptibility to develop periodontitis. The PRC determines the patient's level of risk on a scale from 1 (lowest risk) to 5 (highest risk) and generates suggested treatment options to guide the clinician and patient toward a health care strategy based on risk reduction. It is user-friendly and requires only information that is gathered during a routine periodontal examination.

We report the results of a study designed to test the following hypothesis: using information gathered during a routine periodontal examination, the PRC can calculate risk scores that predict with high accuracy and validity changes in periodontal status, as determined by alveolar bone loss and tooth loss.

**SUBJECTS AND METHODS**

**Study population.** The study population consisted of men enrolled in the Veterans Affairs, or VA, Dental Longitudinal Study, an ongoing closed-panel study of aging and oral health begun in 1968.<sup>15</sup> Table 1 presents characteristics of this population. Comprehensive medical and dental examinations were performed on enrollment and were repeated at intervals of approximately three years.

Of the 1,231 subjects enrolled in this retrospective study, 1,157 were dentate at baseline. Of these, 523 were present at all examinations through the 15-year follow-up and for whom we had records with complete data. These 523 men made up our study population. The subjects were not VA patients, but were recruited from the greater Boston area and received their medical and dental care in the private sector. Additional details about the VA Dental Longitudinal Study, including training and calibration of the clinical examiners, have been published elsewhere.<sup>15-18</sup>

**Baseline and subsequent examinations.** Clinical examinations consisted of charting caries and restorations on all teeth, and assessing peri-

odontal status by probing with a periodontal probe.<sup>18</sup> The clinicians measured probing pocket depths at multiple sites around all teeth. A single ordinal score was assigned to each tooth based on the deepest probed site. We converted the ordinal scores to millimeters for use in the risk calculator analysis (for example, the highest ordinal score of 3 was equivalent to 5 mm or greater probing depth).

We recorded medical and dental histories and obtained full-mouth radiographs with bitewings at baseline and at each subsequent examination. At each of the examinations during the 15-year study, we asked each subject to respond “yes” or “no” to the question, “Have you had any gum treatments or gum surgery since your last examination?” We used these responses as a measure of treatment. Baseline radiographs were read to identify the presence of molar furcations, vertical bone lesions and root calculus. We assessed subgingival restorations during the clinical examinations.

To determine the risk scores at baseline, we used digitized periapical films to calculate the distance in millimeters from the cemento-enamel junction, or CEJ, to the alveolar bone crest at mesial and distal sites of all teeth present.<sup>19</sup> We entered data obtained at the baseline examination (box, “Information Required by the PRC for Determination of Risk Scores”) into the PRC, and calculated a risk score (on a scale of 1 to 5) for each subject. A detailed description of risk factors and their derivation is in preparation (J.M., R.P., unpublished data, 2002).

**Determination of changes in periodontal status.** We determined changes in periodontal status over time by comparing the clinical and historical medical and dental records and radiographs obtained at years 3, 9 and 15 with the baseline medical and dental records and radiographs. We measured alveolar bone height on mesial and distal tooth surfaces from digitized films, according to the method developed by Jeffcoat and colleagues.<sup>19</sup> (Bone height around periodontally healthy teeth was counted as 100 percent. Measured values were subtracted from 100 to yield a percentage loss of alveolar bone height.) Radiographic data were not available for all sites for all years. We used the clinical records to identify teeth that were extracted during the 15-year period. For analysis of tooth loss, we defined periodontally affected teeth as those teeth that had a pocket depth of 5 mm or more or a loss of alveolar bone height of greater than 2 percent.

## BOX

### INFORMATION REQUIRED BY THE PRC\* FOR DETERMINATION OF RISK SCORES.

**Patient Age**

**Smoking History**

**Diabetes Diagnosis**

**History of Periodontal Surgery**

**Pocket Depth**

**Bleeding on Probing**

**Restorations Below the Gingival Margin**

**Root Calculus**

**Radiographic Bone Height**

**Furcation Involvements**

**Vertical Bone Lesions**

\* PRC: Periodontal risk calculator.

*Bone loss.* We defined disease severity as the mean percentage reduction in bone height at sites that experienced bone loss greater than the threshold of 2 percent for all sites that could be compared. (Some sites could not be compared because of technical difficulties such as overlapping images of teeth in the interproximal areas, absence of a landmark and missing radiographs.) The extent of disease within subjects was defined as the percentage of sites that experienced a decrease in bone height. We calculated the extent of disease within subjects by dividing the number of sites with alveolar bone height loss that exceeded 2 percent by the total number of sites that could be measured; the result was expressed as a percentage.

*Tooth loss.* We defined “percentage tooth loss” as the percentage of teeth present at baseline that subsequently were extracted. We determined loss of teeth that were defined at baseline as periodontally affected or unaffected. In addition, we determined changes in the percentage of subjects in each risk group who lost one or more teeth at years 3, 9 and 15.

## STATISTICAL ANALYSIS

We grouped study subjects on the basis of risk scores calculated by the PRC at baseline (that is,

TABLE 2

PERCENTAGE INCREASE IN DISEASE EXTENT AT YEARS 3, 9 AND 15.				
YEAR	RISK SCORE AT BASELINE	NO. OF SUBJECTS	MEAN* (SE†) PERCENTAGE OF SITES	ANOVA P VALUE‡
3	1	2	11.1 (11.1)	< .00001
	2	104	17.2 <sup>a</sup> (1.4)	
	3	193	21.3 <sup>a,b</sup> (1.0)	
	4	120	25.6 <sup>b</sup> (1.7)	
	5	104	35.3 <sup>c</sup> (1.9)	
9	1	2	60.0 (6.0)	< .00001
	2	104	35.2 <sup>a</sup> (2.2)	
	3	193	40.8 <sup>a</sup> (1.6)	
	4	120	50.3 <sup>b</sup> (2.2)	
	5	104	57.0 <sup>b</sup> (2.5)	
15	1	2	15.4 (15.4)	.00082
	2	104	51.9 <sup>a</sup> (2.9)	
	3	193	53.8 <sup>a</sup> (2.2)	
	4	120	60.4 <sup>a,b</sup> (2.7)	
	5	104	63.9 <sup>b</sup> (3.0)	

\* Within each year, the one-way analysis of variance, or ANOVA, P value is for any differences between the risk score groups. If group differences are indicated ( $P < .05$ ), then pairwise comparisons between the groups were performed using a Bonferroni method to adjust the significance level for the multiple comparisons.  
 † SE: Standard error.  
 ‡ Within each year, mean values with different superscripts (for example, A and B) are significantly different at the .05 significance level (Bonferroni  $P < .05$ ).

each year of follow-up. In addition, we calculated bone loss and tooth loss using the risk score of 2 as the referent.

RESULTS

Table 1 lists the characteristics of the study population, which consisted of 523 men aged 25 through 74 years at enrollment. Subjects were well-distributed among the age groups. As determined by the mean bone loss score, periodontitis was present in all groups and generally became more severe with increasing age. Of all subjects at baseline, 101 were smokers, nine were diabetic and 42 reported having had some type of periodontal treatment.

On the basis of the self-reports, the proportion of subjects who underwent only one or no “gum treatments” during the 15-year study was 98 percent, 94 percent, 94 percent and 80 percent, respectively, for risk score groups 2 through 5. The numbers of subjects in groups 2 through 5 were well-distributed (Table 2).

**Disease severity.** Figure 1 shows the change in mean percentage alveolar bone loss for all periodontal sites that exceeded a 2 percent change for each risk group at years 3, 9 and 15.

The results show a strong positive association between risk score at baseline and disease severity at each year ( $P < .0001$ ). The mean percentage bone loss at year 3 ranged from 0.7 percent in group 2 to 2.5 percent in group 5. By year 15, the mean percentage bone loss ranged from 3.3 percent in group 2 to 6.9 percent in group 5. The rank order of change in mean percentage bone loss for groups 2 through 5 (from most to least bone loss) was 5, 4, 3 and 2 at all years. Mean values for bone loss for group 5 differed from those for group 4 at each of the three years; mean values for group 4 differed from those for group 3 at year 9; and mean values for groups 2 and 3 did not differ from each other at any of the three years (.05 Bonferroni-adjusted significance level).

**Disease extent.** Table 2 shows the change in disease extent for all groups at all years. The results show a strong positive association

year 0). Only two subjects had a risk score of 1, and were not included in any of the statistical analyses. We compared disease severity (that is, mean percentage bone loss) and extent (that is, mean percentage of sites with loss in bone height) between the four risk groups at 3, 9 and 15 years using a one-way analysis of variance, or ANOVA.

The incidence of tooth loss was compared between risk groups at 3, 9 and 15 years using Poisson regression by means of generalized estimating equations to allow for overdispersion in the Poisson variance.<sup>20</sup> We compared subjects who lost one or more teeth between risk groups at 3, 9 and 15 years using  $\chi^2$  analysis. When differences between the risk groups were present ( $P < .05$ ), we performed all-pairwise comparisons between risk groups, and used a Bonferroni method to adjust the significance level for the multiple comparisons ( $k = 6$ ) regarding each outcome within

between risk score at baseline and the percentage of affected teeth in all risk groups at years 3 and 9 ( $P < .00001$ ) and at year 15 ( $P = .00082$ ). The mean percentage of affected teeth increased over time within the groups, and by year 15, it ranged from 52 percent in group 2 to 64 percent in group 5. As was true for alveolar bone loss, the rank order of groups (from most to least disease extent) was 5, 4, 3 and 2 at all years.

**Tooth loss.** We found a strong positive association between risk score at baseline and tooth loss at years 3, 9 and 15 ( $P < .0001$ ) (Figure 2). The mean percentage tooth loss increased linearly over time for all four risk groups. At all years, the rank order of change in percentage tooth loss (from most to least) for groups 2 through 5 was 5, 4, 3 and 2. Within each year, group 5 had more tooth loss than did group 4, group 2 had the least tooth loss and groups 3 and 4 did not differ from each other (Bonferroni-adjusted .05 significance level). Compared with a risk score of 2, the relative risk, or RR (95 percent confidence interval, or CI), was 3.2 (2.2 to 4.8) for a risk score of 3, 4.5 (3.0 to 6.6) for a risk score of 4 and 10.6 (7.2 to 15.6) for a risk score of 5.

**Periodontally affected teeth.**

Because teeth can be lost for reasons other than periodontal disease, we calculated tooth loss separately for teeth that had and teeth that did not have periodontitis at baseline (data not shown). Of the 1,250 teeth extracted during the 15-year study, 933 (74.6 percent) met the criteria for having periodontitis at baseline. Since so many of the extracted teeth had periodontitis at baseline, a plot of tooth loss restricted to teeth with periodontal disease at baseline closely resembled that shown in Figure 2 for all extracted teeth.

We found a strong positive association between risk score at baseline and loss of teeth that were periodontally affected at baseline for all years ( $P < .0001$ ), and the rank order of tooth loss (from most to least) was always group 5, group 4, group

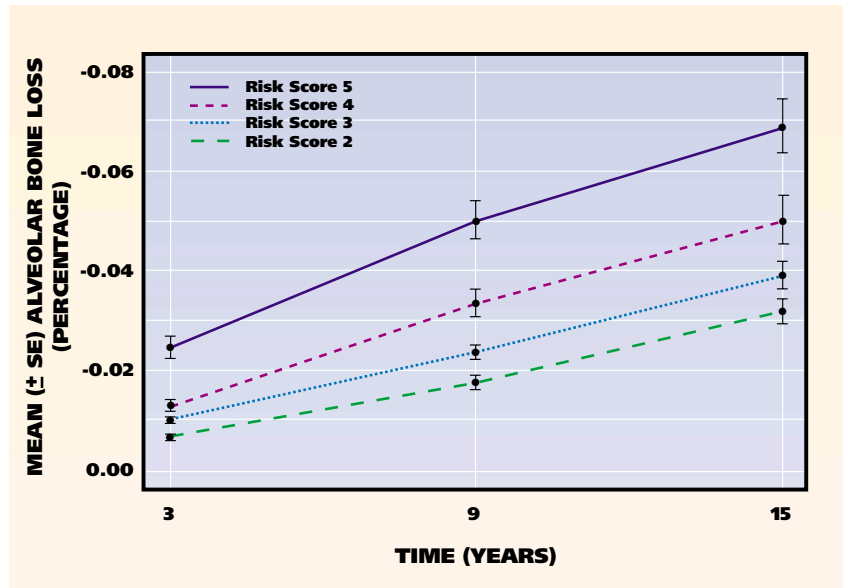


Figure 1. Mean (± standard error) alveolar bone loss from baseline for risk groups 2 through 5, at sites exceeding the threshold of 2 percent loss of alveolar bone height for all sites that could be compared.

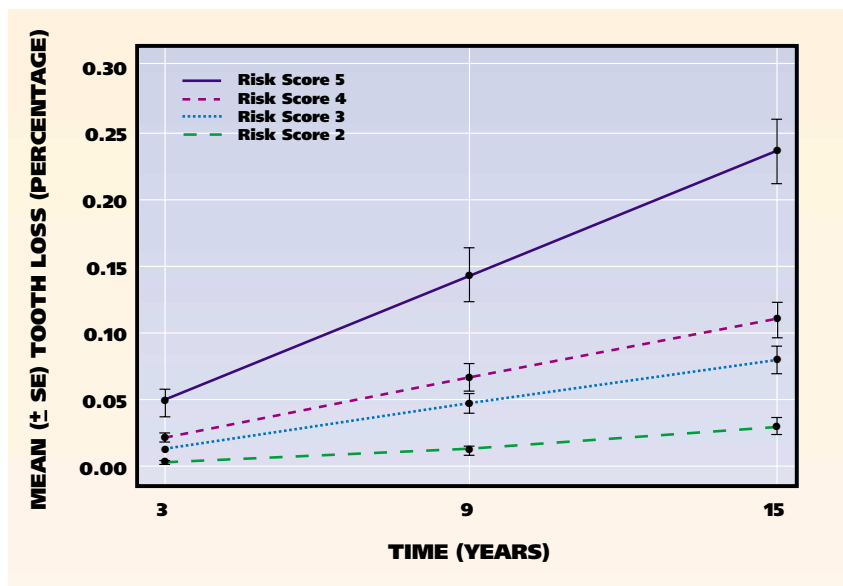


Figure 2. Mean (± standard error) tooth loss from baseline for risk groups 2 through 5, defined as the percentage of teeth present at baseline that were subsequently extracted.

3 and group 2. Compared with a risk score of 2, the RR for loss of teeth that were periodontally affected at baseline (95 percent CI) was 5.5 (2.7 to 11.0) for a risk score of 3, 8.1 (4.2 to 15.7) for a risk score of 4 and 22.7 (11.8 to 43.7) for a risk score of 5. Loss of teeth not periodontally affected at baseline was small, and the risk score was not a predictor of loss of these teeth.

Table 3 shows the percentage of subjects with

TABLE 3

PERCENTAGE OF SUBJECTS WITH TOOTH LOSS AT YEARS 3, 9 AND 15.				
YEAR	RISK SCORE AT BASELINE	NO. OF SUBJECTS	NUMBER (PERCENTAGE) OF SUBJECTS WITH TOOTH LOSS*	$\chi^2$ P VALUE†
3	1	2	0 (0.0)	.00002
	2	104	11 (10.6 <sup>a</sup> )	
	3	193	35 (18.1 <sup>a</sup> )	
	4	120	28 (23.3 <sup>a,b</sup> )	
	5	104	39 (37.5 <sup>b</sup> )	
9	1	2	1 (50.0)	< .00001
	2	104	24 (23.1 <sup>a</sup> )	
	3	193	78 (40.4 <sup>b</sup> )	
	4	120	68 (56.7 <sup>c</sup> )	
	5	104	74 (71.2 <sup>c</sup> )	
15	1	2	1 (50.0)	< .00001
	2	104	39 (37.5 <sup>a</sup> )	
	3	193	115 (59.6 <sup>b</sup> )	
	4	120	88 (73.3 <sup>b</sup> )	
	5	104	91 (87.5 <sup>c</sup> )	

\* Within each year, percentages with different superscripts (for example, A and B) are significantly different at the .05 significance level (Bonferroni  $P < .05$ ).

† Within each year,  $\chi^2$  P value for any association between risk score and percentage of subjects with any tooth loss. If group differences are indicated ( $P < .05$ ), then pairwise comparisons between the groups were performed using a Bonferroni method to adjust the significance level for the multiple comparisons.

tooth loss since baseline, a measure of the distribution of advancing disease among subjects in each risk group. Again, there was a strong positive association between risk score and tooth loss ( $P < .00002$ ). At year 3, the rank order of groups (from most to least tooth loss) was 5, 4, 3 and 2, and this order was maintained at years 9 and 15. Compared with a risk score of 2, the RR for any tooth loss by years 3, 9 and 15 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.

**DISCUSSION**

During the last two decades, researchers have conducted extensive research on the pathobiology of periodontal diseases, and our understanding of the nature of these diseases has increased greatly. This is especially true in regard to the important role of the host and risk factors in the

pathobiology of periodontal disease, and the wide range of susceptibility among people.<sup>1-12</sup> This new knowledge is changing our approach to clinical management of these diseases. Dental practice is in transition from the repair model to the medical or wellness model of care.

An example of the wellness model from the field of medicine is cardiovascular disease, for which major risk factors have been identified and are relatively well-understood. Attempts to reduce risk exposure have been highly successful in lowering the incidence of the disease. In dentistry, risk factors for dental caries are reasonably well-defined, and there is a large body of literature in regard to caries risk assessment.<sup>21-25</sup> This is not the case for periodontal diseases.

Transition to the wellness model of care is an important development

because, over time, its use should result in a decreased incidence of periodontitis, a significant reduction in the periodontal disease treatment burden in the population and a reduction in the costs of care. Success of the wellness model depends in large measure on the ability of practitioners to accurately assess risk and institute risk reduction steps as an integral part of prevention, treatment and maintenance. A risk assessment tool somewhat like that developed for breast cancer<sup>26</sup> and those being developed for dental caries<sup>21-23</sup> is badly needed for periodontal disease.

We have developed a risk assessment tool that is user-friendly and inexpensive, requires little dentist or patient time and effort, and requires only information that is gathered during a traditional periodontal examination. No laboratory testing is required for the PRC. The purpose of our study was to test the accuracy and validity of

risk scores calculated using the PRC as predictors of periodontal status.

The population used in this study was ideal in that all data required by the PRC, including radiographs, were available and actual outcomes were known for a period of 15 years. The size of the population and the distribution of periodontal status were sufficient to provide adequate numbers of subjects in each risk score group at baseline and during the 15-year period. The population consisted of men only. However, study outcome should be independent of sex since we measured the strength of the association between risk prediction and actual outcome.

The VA Dental Longitudinal Study did not include a dental treatment component. Subjects enrolled in the study were not VA patients and received treatment only if they chose to do so through the private sector. Based on self-reports, the proportions of subjects who received no or only one gingival treatment during the 15-year study were 98 percent, 94 percent, 94 percent and 80 percent, respectively, for groups 2 through 5. Because relatively few subjects reported that they received treatment, our findings may be relevant only to untreated populations. We do not know the effects of treatment on the outcome of the risk predictions. It is important to conduct comparable longitudinal studies of subjects who have had periodontal therapy.

Traditional measurement of periodontal status and its change over time generally includes measurement of loss of periodontal connective-tissue attachment and loss of alveolar bone and teeth. The data set we used in this study did not include measurements of connective-tissue attachment loss, nor are such measurements made as part of a routine clinical periodontal examination. However, sequential periapical radiographs with bitewings were available during the 15-year period. These radiographs were of sufficient quality to permit analysis of change using a computer-assisted method.<sup>19</sup> Such radiographic measurements have far greater sensitivity and specificity as an indicator of periodontal status change than does loss of clinical attachment.

The risk scores accurately predicted alveolar bone loss during the entire 15-year period. Whether assessed as mean percentage bone loss from baseline (a measure of disease severity) or increase in percentage of sites with alveolar bone loss (a measure of disease extent), a strong association existed between group risk scores and

actual bone loss ( $P < .0008$ ), and groups were in rank order of increasing alveolar bone loss throughout the 15-year period. We should note that by year 3, the mean bone loss was 3.1 percent in group 5, while subjects in group 2 did not experience a similar amount of bone loss (2.5 percent) until year 15, at which time mean bone loss was 6.9 percent in group 5. Thus, risk scores are both accurate and valid predictors of alveolar bone loss.

The results show that risk scores were strong predictors of loss of periodontally affected teeth but not of periodontally unaffected teeth during the 15-year period. Whether measured as the percentage of total teeth present at baseline that were lost or the proportion of subjects in each group that lost teeth, tooth loss increased in rank order with higher risk score ( $P < .0001$ ). By year 3, 37.5 percent of subjects in group 5 had lost teeth, while subjects in group 2 did not reach that percentage until year 15, at which time 87.5 percent of subjects in group 5 had lost teeth.

The RR of tooth loss compared with a risk score of 2 was 3.2 for a risk score of 3, 4.5 for a risk score of 4 and 10.6 for a risk score of 5. We should note that 74.5 percent of the total teeth lost were designated at baseline as being periodontally affected. The RRs for tooth loss were approximately double those for teeth that were periodontally affected at baseline. Thus, the PRC is particularly good at detecting patients at high risk of losing teeth that are periodontally affected at baseline.

## CONCLUSION

Our data show that valid and accurate predictions of risk of periodontal deterioration as measured by change in alveolar bone status and tooth loss can be made using information gathered during a traditional periodontal examination and the PRC. No laboratory tests such as the periodontitis susceptibility test for a polymorphism in the interleukin-1 gene family or bacterial culture data are required. Our findings show a strong association between the assigned risk score and the actual periodontal deterioration observed during a 15-year period.

The PRC will provide dentists with a new tool for assessing risk accurately, and it generates suggested treatment options for minimizing risk (such as quitting smoking) and for repairing existing damage (such as scaling and root planing



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or flap surgery). We should expect use of the PRC along with suggested treatments to result in more uniform clinical

decision-making about periodontal disease, a reduction in disease incidence, improved oral health, a significant reduction in the need for complex periodontal treatment and a reduction in the costs of care. The availability of the PRC should foster the transition from the repair model to the wellness model for the prevention and treatment of periodontal diseases. ■

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