

CURRENT STATUS OF PERIODONTAL RISK ASSESSMENT

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ABSTRACT

Chronic periodontitis is a destructive chronic inflammatory disease of bacterial etiology. Mounting evidence confirms that not all patients are susceptible to inflammatory periodontal disease, and further, that the extent and severity of its clinical manifestation varies as a function of individual risk. Risk assessment models are needed to target treatment effectively. Contemporary risk assessment, as applied to periodontal disease, represents an innovative approach to managing periodontitis. The central intent of this paper is to review the current view of risk assessment as it relates to the diagnosis and management of chronic periodontitis, as well as to consider a number of such applications that can be incorporated into daily practice.

INTRODUCTION

Periodontal disease is a heterogeneous group of disorders affecting the periodontium, the most common of which are gingivitis and chronic periodontitis. Within the past 2 decades, substantial evidence indicates that susceptibility to periodontal disease (1) varies among patients and (2) is a function of both acquired and intrinsic risk factors.¹⁻³ These conclusions are the result of key epidemiological studies that suggest the prevalence of chronic periodontitis in an adult population is 35% to 50%.^{4,5} Coupled with epidemiologic evidence, a better understanding of the pathogenesis of periodontitis has emerged.⁶ Accordingly, more recent efforts related to risk assessment have been focused on identifying new risk factors and, more importantly, developing a viable algorithm to assess risk in the clinical setting.⁷ Our primary objective is to review the current state of risk assessment as it relates to the diagnosis and management of chronic periodontitis, of identifying a practical means for clinicians to effectively develop a risk profile for each patient.

Indeed, notwithstanding the publication of numerous studies implicating tobacco use and diabetes as significant risk factors for periodontitis,^{1,8-10} as well as the application of sophisticated methodologies to profile specific bacterial species¹¹ implicated in its pathogenesis, a universally accepted objective method of calculating risk of developing or worsening periodontal disease at a future date does not exist; however, several risk assessment methods have been described.¹²⁻¹⁸ In general, these algorithms take the form of a series of patient-specific data entries representing the constellation of accepted risk factors for periodontal disease, which are then subjected to some form of data analysis. The difference in output, ie, risk profile, is then largely a function of the individual processing of the data, from a functional graphical representation of the patient's risk (Fig. 1),¹⁴ to a more sophisticated assessment,

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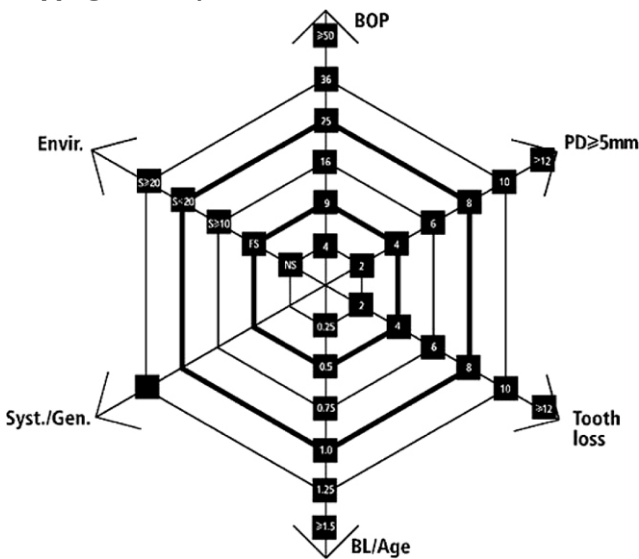
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Figure 1. Functional diagram to evaluate the patient's risk for recurrence of periodontitis. Each vector represents one risk factor or indicator with an area of relatively low risk, an area of moderate risk, and an area of high risk for disease progression. All factors have to be evaluated together and, hence, the area of relatively low risk is found within the center circle of the polygon, whereas the area of high risk is found outside the periphery of the second ring in bold. Between the 2 rings in bold, there is the area of moderate risk. (Originally published in: Lang NP, Tonetti MS. Periodontal risk assessment (PRA) for patients in supportive periodontal therapy (SPT). *Oral Health & Preventive Dentistry*. 2003;1:7-16. Reprinted with permission from Quintessence publishing. Copyright 2003.)



including a quantification of disease severity commonly associated with a specific diagnosis, a general prognosis, and treatment interventions typically associated with a periodontal condition as modified by risk (Fig. 2).¹⁹

To better understand using risk in the management of patients with periodontal disease, it would be useful to first review the current thinking regarding risk. Risk is defined as “the probability that an event will occur in the future, or the probability that an individual develops a given disease or experiences a change in health status during a specified interval of time.”²⁰ A risk factor is defined as “any characteristic, behavior or exposure with an association to a particular disease. The relationship is not necessarily causal in nature.”²¹

What are the risk factors for periodontal disease? As noted above, perhaps the initial, and possibly most significant risk factors thus far identified, are smoking²² and diabetes.²³ In addition, systemic, genetic, and tooth-related local factors have been reported.²⁴ Of these, local factors typically include, but are not limited to, gingival inflammation, prior attachment

loss, calculus deposits, furcations, pocket formation, and defective restorations. Systemic factors include conditions that result in suppression of the immune system, alterations in endocrine status, and certain medications that specifically affect the gingiva. In addition, several studies have linked specific genetic markers to susceptibility to periodontitis,²⁵⁻²⁸ although their results conflict.^{29,30} Accordingly, this review discusses the various risk models that have been developed thus far, the studies that validate these models, implications for the clinician, and future directions in risk assessment.

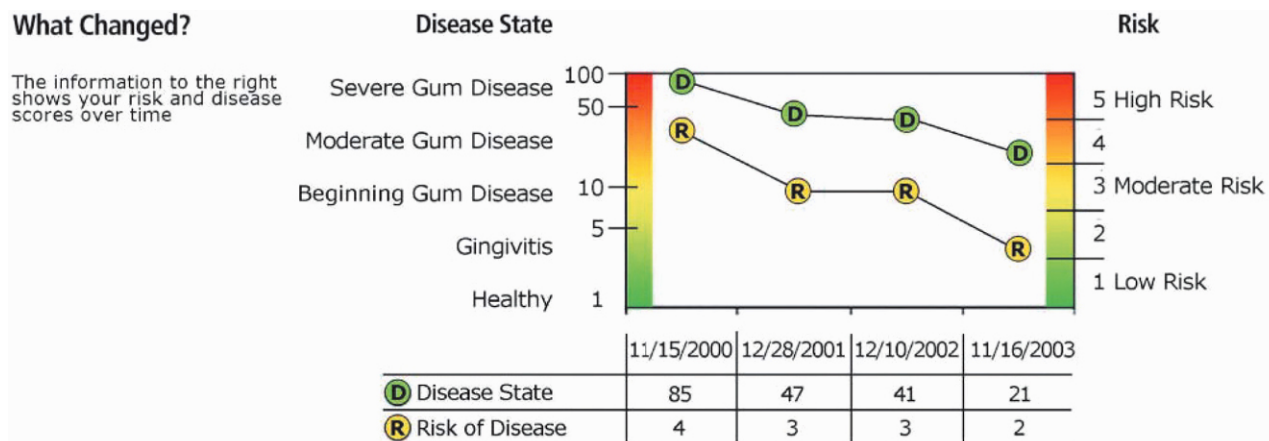
Risk Models

Although the most recognized sign of gingival inflammation is bleeding in response to mechanical challenge,³¹ its indication of current and future disease activity, ie, ongoing attachment loss, has not been established.³²⁻³⁵ In fact, only retrospectively is it possible to ascertain the presence of an “active site.”³⁶ Hence, using the single risk factor, bleeding on probing, is insufficient to accurately determine risk.

In 2008, the American Academy of Periodontology defined risk assessment as “the process by which qualitative or quantitative assessments are made of the likelihood for adverse events to occur as a result of exposure to specified health hazards or by the absence of beneficial influences.”³⁷ Indeed, clinical or laboratory measures that could accurately predict future disease progression would allow clinicians to better prevent recurrent periodontal destruction.³⁸ Unfortunately, traditional clinical parameters of periodontal diseases, eg, probing depth (PD), attachment loss, and alveolar bone level, are simply cumulative measures of past disease and do not accurately predict current (or future) disease activity. In spite of this, most clinicians will often equate periodontal risk with the extent and severity of periodontal status. That is, patients with little or no periodontal breakdown are assumed to be at low-risk for future disease, whereas patients presenting with more severe tissue destruction are considered to be at higher risk for future disease.

Nonetheless, it must be understood that risk and diagnosis are vastly different entities. Risk predicts the disease status at some future point in time, including the rate at which an existing disease condition is likely to progress. Diagnosis, by contrast, is an expression of a current disease status.³⁹ Consistent with these definitions and the importance of risk in periodontal care, the American Academy of Periodontology has stated that “the clinical use of risk assessment will become a component of all comprehensive dental and periodontal evaluations as well as part of all periodic dental and periodontal examinations.”³⁷ Although previous clinical attachment loss is certainly a risk factor for future periodontal breakdown, as noted previously, at present there does not exist a reliable measure for predicting either current or future disease activity. How then, can we determine if a patient is, in fact, at risk? To meet the objective of incorporating

Figure 2. Section of the clinical report showing risk and disease scores and their change over time, along with treatment recommendations. (Reprinted with permission from the *Journal of Dental Education*. Page RC, Martin JA, Loeb CF. The Oral Health Information Suite (OHIS): its use in the management of periodontal disease. *J Dent Educ* 2005;69(5):509-20. Copyright 2005 by the American Dental Education Association. www.jdentaled.org)



Active Intervention You May Need

Generally most effective May be effective Less likely to be effective

REDUCE POCKETS 5-7 mm



Pockets in the 5 to 7 mm depth range cannot be cleaned with a toothbrush and floss, and professional tooth cleaning tools don't always reach the bottom of the pocket. Incomplete removal of plaque and calculus results in deeper pockets and tooth loss.

- Deep cleaning
- Professional cleaning
- Antibacterial Medication (special circumstance)
- Bone surgery
- Flap surgery
- Bone graft (Special circumstances)
- Extraction (Special circumstances)

POCKETS <5 mm



Pockets that are less than 5 mm deep can be thoroughly cleaned of bacteria and calculus most easily. However, all deep pockets were at one time less than 5 mm, so one should always watch for advancing disease. The optimal pocket depth is 3 mm or less.

- Deep cleaning
- Professional cleaning
- Antibacterial Medication (Special circumstances)
- Bone surgery
- Flap surgery
- Bone graft (Special circumstances)
- Extraction (Special circumstances)

VISIT THE DENTIST

Symptoms are warning signs that are frequently too late in the disease process for the simplest, most predictable, least costly treatment. Regular visits to detect disease in the early stages can prevent more complex and expensive treatment. Better still are regular visits for preventive care targeted to your risk factors.

- Two times per year
- One time per year
- More than two times per year or less than one time per year

risk assessment into the diagnostic process, numerous risk assessment models have been introduced during the past decade^{12,14-18,40} (Table I).

In 2002, Page and colleagues¹⁷ introduced the Periodontal Risk Calculator (PreViser), a component of the Oral Health Information Suite, that evaluates 11 key risk parameters: patient's age, smoking, diagnosis of diabetes, history of

periodontal surgery, PD, bleeding on probing (BOP), furcation involvement, subgingival restorations, root calculus, radiographic bone height and the presence of vertical bone lesions (Fig. 3). Based on these parameters, "numeric risk and disease severity scores"¹⁹ are calculated that establish both an assessment of risk as well as a quantification of disease severity. These, in turn, are coupled with suggested treatment options for the clinician (see Fig. 2).

TABLE 1. Characteristics of various risk assessment models

Author(s)/Year	Risk model	Risk variables	Notes
Page et al (2002)	Periodontal Risk Calculator (PRC)*	11 factors: Age, smoking history, DM, history of periodontal surgery, BOP, furcation involvements, subgingival restorations, vertical intrabony defects, root calculus, PD, radiographic bone loss	Only the deepest PD and greatest bone loss per sextant are entered for PD and radiographic bone levels.
Lang and Tonetti (2003)	Periodontal Risk Assessment (PRA)	6 factors: Full-mouth BOP %, PD ≥ 5mm, tooth loss, radiographic bone loss-to-age ratio, systemic and/or genetic conditions, smoking	All sites of BOP and PD ≥ 5mm must be entered. Alveolar bone loss is limited to the most severe posterior site. Binary designation for "systemic and/or genetic conditions" category. Six-point scale for each factor
Chandra (2007)	Modified PRA	8 factors: Full-mouth BOP %, PD ≥ 5mm, tooth loss, CAL to age ratio, smoking, DM, dental status - systemic factors interplay, psychosocial factors	Modified PRA model (see above). DM is separated from systemic conditions. Alveolar bone loss is not evaluated. Five-point scale for each factor.
Leininger et al (2010)	Periodontal Risk Assessment Diagram Surface (PRAS)	6 factors: Full-mouth BOP %, PD ≥ 5mm, tooth loss, radiographic bone loss-to-age ratio, systemic status, smoking	Modified PRA model (see above). Identical to PRA except uses 5-point scale for each factor.
Trombelli et al (2009)	UniFe	5 factors: BOP, PD ≥ 5mm, radiographic bone loss-to-age ratio, smoking, DM	All sites of BOP and PD ≥ 5mm must be entered. Alveolar bone loss included for one interproximal site of each tooth.
Lindskog et al (2010)	DentoRisk†	20 factors: Systemic Predictors: Age in relation to history of chronic periodontitis, family history of chronic periodontitis, systemic disease and related diagnoses, result of skin provocation test, patient cooperation and disease awareness, socioeconomic status, smoking, clinician experience Local Predictors: bacterial plaque (oral hygiene), endodontic pathology, furcation involvements, vertical intrabony defects, radiographic marginal bone levels, PD, BOP, marginal dental restorations, increased tooth mobility, missing teeth, abutment teeth, presence of purulence	

BOP, bleeding on probing; CAL, clinical attachment loss; DM, diabetes mellitus; PD, probing depth

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†DentoSystem Scandinavia AB, Stockholm, Sweden.

Likewise, the Periodontal Risk Assessment (PRA)¹⁴ model is based on a multifactorial graphic, ie, the Periodontal Pentagon Risk Diagram (see Fig. 1). This functional diagram is composed of 6 vectors representing a combination of 6 clinical, systemic, and environmental factors to predict the risk of recurrence of periodontitis, and patients are classified as either low-, moderate-, or high-risk profile. The diagram includes (1) percent BOP, (2) number of residual periodontal pockets ≥ 5 mm, (3) number of lost teeth, (4) percent alveolar bone loss in relation to the patient's age, (5) systemic and/or

genetic predispositions, ie, interleukin (IL)-1 gene polymorphism, diabetes mellitus, or cardiovascular disease, and (6) environmental factors, such as tobacco use. The aggregate sum of these factors provides an individualized total risk profile for the patient. However, in contrast to the Periodontal Risk Calculator (PRC), which is calculated at the onset of treatment, the PRA provides an assessment of risk for patients during the supportive, posttreatment phase, after active therapy has been completed.

Figure 3. Screenshot of Periodontal Risk and Disease Assessment Input Form. (Reprinted with permission from PreViser Corporation, Mount Vernon, WA.)

Periodontal Risk and Disease Assessment Input Form

Prepared By

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 Perio Exam Date 11/16/2004

Prepared For

Name: John Smith
 Date Of Birth 1949-05-05
 PreViser ID 7caa59de-c85c-43c2-9c52-1e0c6427c598

Patient History and Clinical Data

Dental Care

- a) Unknown
- b) Never / First Visit
- c) 1 - 2 per year
- d) > 2 per year**
- e) Irregular
- f) Emergency

Smoking History

- a) Unknown
- b) Never Smoked Cigarettes
- c) Former Cigarette Smoker**
- d) Smokes less than 10 per
- e) Smokes 10 or more per d

Diabetic

- a) Unknown
- b) Not Diabetic**
- c) Good Diabetic Control
- d) Fair Diabetic Control
- e) Poor Diabetic Control

- Periodontal Surgery for Pockets Has Been Done
- Furcation Involvements
- Subgingival Restorations
- Vertical Bone Lesions
- Calculus on Radiographs or Below the Gingival Margin

Oral Hygiene:

- Excellent: improvement not possible**
- Acceptable: slight improvement possible
- Unacceptable: substantial improvement needed

Deepest Pocket Per Sextant

Upper Right

- a) <5 mm**
- b) 5-7 mm
- c) >7 mm
- d) No Teeth

Bleeding

Upper Anterior

- a) <5 mm**
- b) 5-7 mm
- c) >7 mm
- d) No Teeth

Bleeding

Upper Left

- a) <5 mm**
- b) 5-7 mm
- c) >7 mm
- d) No Teeth

Bleeding

Lower Right

- a) <5 mm**
- b) 5-7 mm
- c) >7 mm
- d) No Teeth

Bleeding

Lower Anterior

- a) <5 mm**
- b) 5-7 mm
- c) >7 mm
- d) No Teeth

Bleeding

Lower Left

- a) <5 mm**
- b) 5-7 mm
- c) >7 mm
- d) No Teeth

Bleeding

Xray: Distance from CEJ to Bone Crest

Upper Right

- a) <2 mm**
- b) 2-4 mm
- c) > 4 mm
- d) No Teeth

Upper Anterior

- a) <2 mm**
- b) 2-4 mm
- c) > 4 mm
- d) No Teeth
- e) No X-Ray

Upper Left

- a) <2 mm**
- b) 2-4 mm**
- c) > 4 mm
- d) No Teeth

Lower Right

- a) <2 mm**
- b) 2-4 mm
- c) > 4 mm
- d) No Teeth

Lower Anterior

- a) <2 mm**
- b) 2-4 mm
- c) > 4 mm
- d) No Teeth
- e) No X-Ray

Lower Left

- a) <2 mm**
- b) 2-4 mm**
- c) > 4 mm
- d) No Teeth

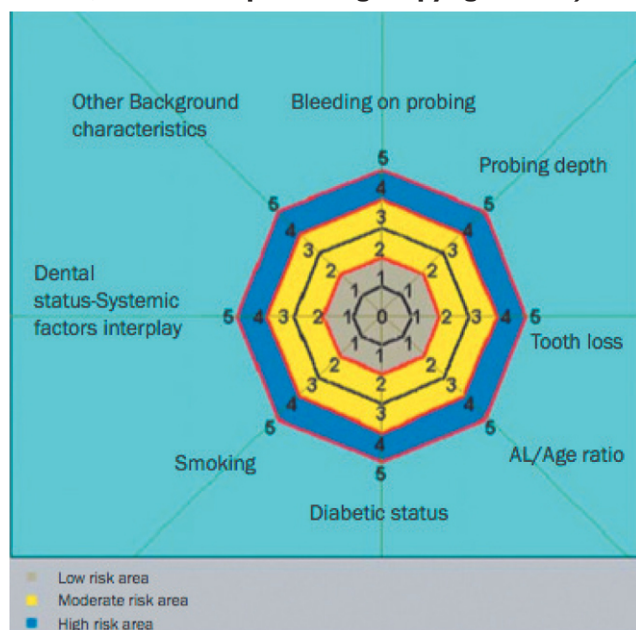
Choose the greatest measurement per sextant (not the average)

Several adaptations to the PRA have been proposed as alternatives.^{12,40} Suggesting that the PRC was “too complicated for the practitioner to implement in clinical practice,” one model¹² included both retrospective and current data, and used a simplified format that retained 4 of the original 6 parameters, with the addition of, specifically, local-systemic factors (tooth-related, immunosuppression, genetic), stress, and diabetic and socioeconomic status. In summary, the 8 parameters were as follows: (1) percentage of sites with BOP, (2) number of sites with PD ≥ 5 mm, (3) number of teeth lost, (4) attachment loss/age ratio, (5) diabetic status, (6) smoking, (7) dental status–systemic factors interplay, and (8) other background characteristics. The ease of interpretation, relative to the PRC, was embodied in the format of the risk diagram itself, which was color-coded into low-, medium-, and high-risk zones (Fig. 4).

Moreover, in contrast to the PRC, which assessed risk prospectively, this model was based on cumulative and retrospective data. That is, according to the author, the model was “primarily a retrospective one where information is gathered to assess the current risk for a patient, unlike other models where current status is assessed and future risk is predicted.”¹² The Periodontal Risk Assessment Diagram Surface⁴⁰ appears to be very similar, if not identical, to the PRA, although in a retrospective study of 30 subjects, no comparison data to the PRA model was cited.

Trombelli and colleagues¹⁸ proposed a simplified risk assessment model (UniFe) using 5 key parameters: (1) smoking status, (2) diabetic status, (3) number of sites with PD ≥ 5 mm, (4) BOP score, and (5) bone loss/age. A numeric value for each parameter was calculated, based on its extent or

Figure 4. The proposed model, which considers the cumulative periodontal status, risk factors, and risk determinants under 8 parameters and with clearly demarcated low-, medium-, and high-risk zones. (Originally published in Chandra RV. Evaluation of a novel periodontal risk assessment model in patients presenting for dental care. *Oral Health Prev Dent* 2007;5:39-48. Reprinted with permission from Quintessence publishing. Copyright 2007.)



severity, and patients were assigned to 1 of 5 risk categories derived from the sum of those values, ie, 1 (low), 2 (low-medium), 3 (medium), 4 (medium-high), or 5 (high).

Most recently, Lindskog and coworkers^{15,16,41} developed a computerized risk assessment and prognostication program (DentoRisk) that is used in conjunction with a skin test for inflammatory reactivity (DentoTest). This model differs from others in that an assessment is first calculated for the patient's overall dentition (Level I). If an elevated risk is detected, a prognosis for annualized attachment loss for each individual tooth (Level II) is then computed. This information can then be used during the treatment planning appointment, and provide the patient and clinician with a current and the *future* prognostication (based on completion of successful therapy).

In general, these models share several common attributes. They all compute risk based on an assessment of current and past findings that have been identified as contributing in some manner to risk for future disease. Each then assigns relative risk values and assigns patients into one of an array of categories that suggest specific approaches to therapy. And, finally, as described below, in each case, attempts have been made to validate the underlying premise, with varying degrees of success.

Validation Studies

Long-term studies⁴²⁻⁴⁴ strongly suggest that clinicians can achieve success in establishing and maintaining periodontal health using conventional therapeutic modalities coupled with empirical, if not frankly subjective, guidelines to estimate risk for future disease. Notwithstanding best efforts, however, tooth loss is invariably seen in a small percentage of patients. The question therefore remains: Might a more quantitative approach to risk assessment significantly enhance the ability to deliver therapy more rationally? Recent studies suggest that such an approach, indeed, may be the case.

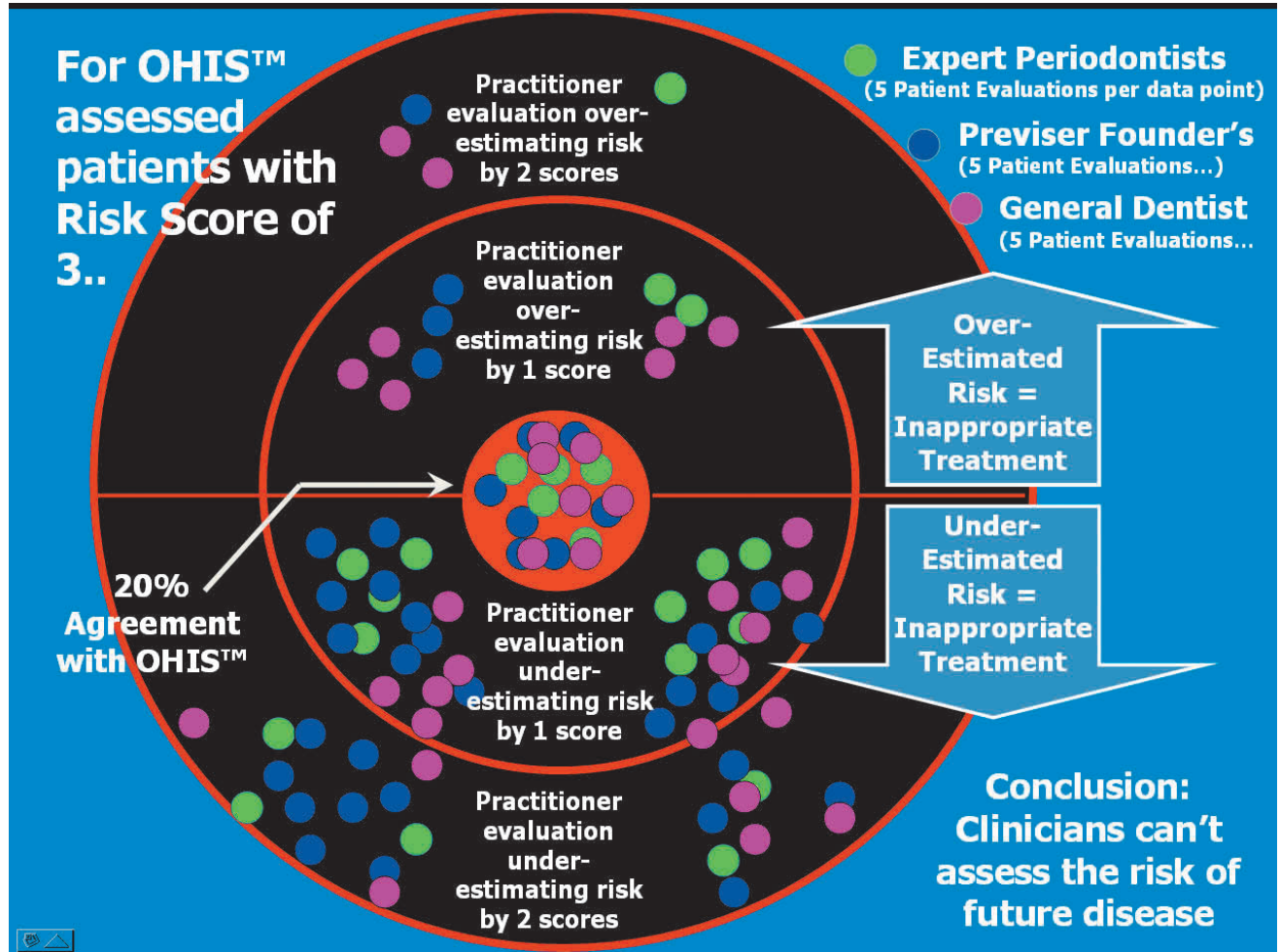
In one study, Persson et al⁴⁵ compared risk estimated by "expert clinicians" to that computed by the PRC. The objectives of their study were actually twofold: (1) to determine "the level of agreement between expert clinician scores and PRC scores, and (2) to determine the "extent of inter-evaluator variation" among the experts. Results of the study showed surprising variation among clinicians' scores and, relative to the PRC scores, a clear tendency to underestimate the risk for future periodontal disease. Overall, among the clinicians, approximately 80% of subject risk was either over- or underestimated (**Fig. 5**). Predictably, in light of the variability of subjective scoring, the authors advocate the use of an objective method to assess risk.

In a related study using the PRA, Persson et al⁴⁶ examined the ability of the IL-1 gene polymorphism to predict the response to regular, follow-up maintenance; following the completion of definitive periodontal treatment, patients were assessed after 4 years of supportive periodontal therapy. In general, if IL-1 status was taken into consideration, PRA scores decreased for IL-1-negative patients, representing a *reduced* PRA-determined risk, whereas scores increased for IL-1-positive patients, indicating an *increased* PRA-determined risk. These findings, therefore, suggested a useful approach for identifying patients who may respond less favorably to maintenance therapy.

More recently, in a blind retrospective study of 107 randomly selected patients seeking periodontal treatment, Trombelli et al¹⁸ compared the Unife and the PRC risk models. For the Unife system, each of 5 separate parameters was scored, and their sum was expressed as an overall measure of risk. Statistical analysis showed complete agreement between the 2 models in approximately 75% of the patients, and although clearly validated in comparison with the PRC, the authors suggested the need for long-term, longitudinal studies to further validate their model.

Page and coworkers¹⁷ reported that the PRC risk score accurately predicted future periodontal status and tooth loss of a population that received routine dental care but typically not periodontal care. In a subsequent study⁵² using the same population, it was determined that tooth loss was more precisely

Figure 5. The extent of agreement for subjects assigned a risk score of 3 by the risk calculator and risk scores assigned by expert evaluator Groups A (blue), B (green), and C (red). Each circle represents 5 risk assessments. For agreement, circles are located on the bull's eye; when scores of 4 or 2 instead of 3 were assigned, circles are located in the inner circle; when 1 or 5 were assigned instead of 3, circles are located in the outer circle. (Originally published in Page RC, Martin JA, Loeb CF. Use of risk assessment in attaining and maintaining oral health. *Compend Contin Educ Dent* 2004;25(9):657-670. Copyright 2004 to AEGIS Publications, LLC. All rights reserved. Reprinted with permission from the publisher.)



and accurately predicted by the combination of risk and severity scores than by either score alone. In contrast, Martin and coworkers⁴⁷ reported on tooth loss observed during periodontal therapy in patients who were assessed using the PRC. Nearly all of the 776 patients were classified as having moderate or severe periodontitis and a total of 980 teeth were lost. Regression analysis showed that the average tooth loss rate (TLR) can be accurately predicted when a combination of the model's disease and risk scores is used. They concluded that tooth loss could be minimized, if not prevented, if periodontal therapy is instituted before the periodontal condition becomes severe. In a subsequent study,⁴⁸ premised on the notion that timely intervention would result in decreased tooth loss, the TLR of 2 patient populations was compared. One population received only routine dental care, whereas the second population received definitive periodontal therapy. For each group of

subjects with the same risk and severity category, comprehensive periodontal therapy was associated with a lower TLR and more subjects who lost no teeth. This study provided clear evidence that a patient with periodontal disease can retain more teeth if comprehensive periodontal therapy is performed in addition to routine dental care. However, because care was administered without use of objectively determined risk, the study shows only the stratification of tooth loss by risk and disease severity. The hypothesis that remains to be proven is that the use of risk assessment will result in better outcomes.

Clinical Implications

Risk assessment provides the clinician with the opportunity to develop a risk-based treatment plan that incorporates the level of risk along with the severity of periodontal disease. Including risk in treatment planning means that the intensity

or frequency of treatment typically associated with a specific condition would be ratcheted up when risk is high and down when risk is low.^{1,19} For example, a risk-based treatment plan for a high-risk patient with severe chronic periodontitis may be surgery and periodontal maintenance 4 times per year; whereas a treatment plan for a low-risk patient with slight chronic periodontitis may be scaling and root planing and 2 periodontal maintenance visits per year. Treatment planning in this manner means that severity alone is not the sole criterion of treatment complexity, which apparently has been an important reason to refer.⁴⁹ Accordingly, guidelines for referral have been developed based on risk.⁵⁰

Risk assessment also provides the opportunity to develop a treatment plan that targets the risk factors, such as periodontal pocket depth, bacteria, tobacco use, and diabetic control for the purpose of reducing risk.^{1,19,24} A typical consequence of periodontal treatment is pocket depth reduction. However, pocket depth is an indicator of disease severity that is used to determine risk.^{12,14,15,17,18,40} Although risk may be lowered as a result of pocket reduction, the clinician could include pocket-reducing treatment as a risk-increasing factor. The clinical importance relates to a key use of risk, which is to prevent worsening of periodontal status by periodontal maintenance. Hence, periodontal maintenance frequency should be based in part on risk. But absent a history of periodontal status that has remained stable for a significant time period, a risk level unadjusted for pocket reduction may be associated with a periodontal maintenance frequency that is too low. For example, treatment that results in a risk reduction from high to low may be interpreted to mean periodontal maintenance needs to be twice instead of 4 times per year. Over time, clinical evidence coupled with a clinician's experience are factors to verify or change periodontal maintenance frequency.

A frequency of 4 times per year is a common recommendation for periodontal maintenance.⁵¹ Although this may have been a reason for inclusion in guidelines, it may be because referred patients typically have a severity of disease typically associated with high risk.^{19,45,49,51} But not every patient is high risk^{47, 52} and not every patient has severe disease.^{4,47,52} As demonstrated by Axelsson,⁵³ customizing the frequency of preventive care means that low-risk patients could be scheduled once a year; whereas high-risk patients may need to be scheduled every 3 months.⁵¹ Customizing recall frequency to risk level means that fewer appointments may be needed, which could increase access for care.

In addition to the individual clinician and patient, a public health organization and dental insurance company could use periodontal disease risk models for periodontal disease surveillance and insurance benefit plan design, respectively. However, the risk model (including its risk factors) would not necessarily be the same for the clinician, the public health organization, and the dental insurance company. Of primary

interest to the clinician is the inclusion of risk factors that are affected by treatment, such as periodontal pocket depth, bacteria, tobacco use, and diabetic control. In contrast, factors not affected by treatment (eg, socioeconomic status, race, ethnicity, gender, age) have limited value for a clinician in the development of a risk-based treatment plan but may be useful for a public health organization or dental insurance company to determine treatment needs over time.

The methods of risk assessment range from the individual clinician's subjective opinion to standardized computer models that use assessments that are more objective in nature.^{12,14,15,17,18,40,45,54} The evidence is clear that the former method has a wide range of variation that could result in the misapplication of treatment for some patients.^{45,54} Merely accurately assessing risk may be insufficient to manage periodontal disease, because treatment needs to account for risk.^{1,19} And a risk-based treatment plan requires that the clinician understands risk and communicates issues about risk to the patient so that they are understood. Unfortunately, understanding and communicating risk is difficult.⁵⁴⁻⁵⁹ Expertise in literacy and numeracy is not universal. Furthermore, words do not have a precise meaning. For example, each of the phrases "very common," "very rare," "very high risk," and "very low risk" can be interpreted differently. And even well-educated individuals may be challenged with concepts such as probability. Although numbers convey risk better than words and visual aids are available to illustrate the meaning of numbers, a patient's assessment of his or her own risk is also influenced by how the information is framed and his or her emotions. Hence, although risk models may lead to better-informed patients and clinicians, how risk is explained to the patient may be just as important as the risk model itself.⁶⁰

Future Directions

Validated risk assessment models are expected to result in better therapeutic outcomes at a lower cost. However, risk assessment models have not yet been validated in longitudinal studies and remain an important issue to be examined.⁶⁰ An economic analysis of savings realized by reducing the incidence or progression of periodontitis would likely provide valuable cost-benefit information that could be used to determine most effective treatment. Additionally, clinical research is needed to determine the most effective way to incorporate risk assessment in patient education.

The usefulness of risk assessment is not limited to disease risk. Risk also pertains to treatment success and risks associated with treatment. For example, resective periodontal surgery results in marginal gingival recession, which may adversely affect oral health-related quality of life and caries susceptibility. Not only would a risk model of treatment be a valuable aid for a clinician and patient but a risk model(s) that accounts for the full spectrum of diseases, therapy, and outcomes could have enormous clinical utility and value.

CONCLUSIONS

What, then, is the value of risk assessment? Incorporation of risk for oral disease into clinical practice, in the broadest sense, has the potential to substantially alter the traditional approach to oral health care delivery. For example, traditional management of periodontal disease has been based on the repair model of care where a lesion or condition is diagnosed and repaired. The “best” treatment in this model is based on the lesion, regardless of the patient’s risk. In contrast, the wellness model of oral health care incorporates risk in the care algorithm, which emphasizes prevention and treatment targeted to risk factors in addition to reparative treatment that is customized to a patient’s specific risk and prognostic factors. In addition to increasing the well-being of patients, this may also lead to decreased morbidity and reduce the overall costs of health care. In this paper, we have reviewed the current status of risk assessment as it pertains to periodontal disease. As we have described, within the past decade, substantial progress had been made in terms of developing viable models for calculating risk that specifically apply to this pervasive inflammatory condition. The goal of risk assessment is the long-term retention of teeth via an amalgamation of prevention, early intervention, and directed therapy. Given that numbers of risk assessment models that focus on periodontal disease have been validated clinically clearly anticipates the next phase in the process. That is, in parallel to guidelines established for managing dental caries, similar guidelines for managing periodontal disease(s) could well result in the very near future.

REFERENCES

1. Beck JD. Methods of assessing risk for periodontitis and developing multifactorial models. *J Periodontol* 1994;65:468-78.
2. Genco RJ. Current view of risk factors for periodontal diseases. *J Periodontol* 1996;67:1041-9.
3. Kornman KS. Patients are not equally susceptible to periodontitis: does this change dental practice and the dental curriculum? *J Dent Educ* 2001;65:777-84.
4. Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988-1994. *J Periodontol* 1999;70:13-29.
5. Albandar JM. Underestimation of periodontitis in NHANES surveys. *J Periodontol* 2011;82:337-41.
6. Page RC, Kornman KS. The pathogenesis of human periodontitis: an introduction. *Periodontol* 2000 1997;14:9-11.
7. Page RC, Beck JD. Risk assessment for periodontal diseases. *International dental journal* 1997;47:61-87.
8. Genco RJ. Assessment of risk of periodontal disease. *Compendium* 1994;S678-83; quiz S714-7.
9. Genco RJ, Loe H. The role of systemic conditions and disorders in periodontal disease. *Periodontol* 2000 1993;2:98-116.
10. Grossi SG, Zambon JJ, Ho AW, Koch G, Dunford RG, Machtei EE, et al. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *J Periodontol* 1994;65:260-7.
11. Dahlen G. Role of suspected periodontopathogens in microbiological monitoring of periodontitis. *Adv Dent Res* 1993;7:163-74.
12. Chandra RV. Evaluation of a novel periodontal risk assessment model in patients presenting for dental care. *Oral Health Prev Dent* 2007;5:39-48.
13. Cronin AJ, Claffey N, Stassen LF. Who is at risk? Periodontal disease risk analysis made accessible for the general dental practitioner. *Br Dent J* 2008;205:131-7.
14. Lang NP, Tonetti MS. Periodontal risk assessment (PRA) for patients in supportive periodontal therapy (SPT). *Oral Health Prev Dent* 2003;1:7-16.
15. Lindskog S, Blomlof J, Persson I, Niklason A, Hedin A, Ericsson L, et al. Validation of an algorithm for chronic periodontitis risk assessment and prognostication: risk predictors, explanatory values, measures of quality, and clinical use. *J Periodontol* 2010;81:584-93.
16. Lindskog S, Blomlof J, Persson I, Niklason A, Hedin A, Ericsson L, et al. Validation of an algorithm for chronic periodontitis risk assessment and prognostication: analysis of an inflammatory reactivity test and selected risk predictors. *J Periodontol* 2010;81:837-47.
17. Page RC, Krall EA, Martin J, Mancl L, Garcia RI. Validity and accuracy of a risk calculator in predicting periodontal disease. *J Am Dent Assoc* 2002;133:569-76.
18. Trombelli L, Farina R, Ferrari S, Pasetti P, Calura G. Comparison between two methods for periodontal risk assessment. *Minerva Stomatol* 2009;58:277-87.
19. Page RC, Martin JA, Loeb CF. The Oral Health Information Suite (OHIS): its use in the management of periodontal disease. *J Dent Educ* 2005;69:509-20.
20. Albandar JM. Global risk factors and risk indicators for periodontal diseases. *Periodontol* 2000 2002;29:177-206.
21. Brownson RC, Pettiti, D. B. *Applied epidemiology: theory to practice*. New York: Oxford University Press; 1998.
22. Tonetti MS. Cigarette smoking and periodontal diseases: etiology and management of disease. *Ann Periodontol* 1998;3:88-101.
23. Chavarry NG, Vettore MV, Sansone C, Sheiham A. The relationship between diabetes mellitus and destructive periodontal disease: a meta-analysis. *Oral Health Prev Dent* 2009;7:107-27.
24. Nunn ME. Understanding the etiology of periodontitis: an overview of periodontal risk factors. *Periodontol* 2000 2003;32:11-23.
25. Kornman KS, Crane A, Wang HY, di Giovine FS, Newman MG, Pirk FW, et al. The interleukin-1 genotype as a severity factor in adult periodontal disease. *J Clin Periodontol* 1997;24:72-7.
26. McGuire MK, Nunn ME. Prognosis versus actual outcome. IV. The effectiveness of clinical parameters and IL-1 genotype in accurately predicting prognoses and tooth survival. *J Periodontol* 1999;70:49-56.
27. McDevitt MJ, Wang HY, Knobelmann C, Newman MG, di Giovine FS, Timms J, et al. Interleukin-1 genetic association with periodontitis in clinical practice. *J Periodontol* 2000;71:156-63.
28. Michalowicz BS, Diehl SR, Gunsolley JC, Sparks BS, Brooks CN, Koertge TE, et al. Evidence of a substantial genetic basis for risk of adult periodontitis. *J Periodontol* 2000;71:1699-707.
29. Greenstein G, Hart TC. A critical assessment of interleukin-1 (IL-1) genotyping when used in a genetic susceptibility test for severe chronic periodontitis. *J Periodontol* 2002;73:231-47.
30. Kinane DF, Shiba H, Hart TC. The genetic basis of periodontitis. *Periodontol* 2000 2005;39:91-117.

31. Greenstein G. The role of bleeding upon probing in the diagnosis of periodontal disease. A literature review. *J Periodontol* 1984;55:684-8.
32. Belibasakis GN, Bostanci N. The RANKL-OPG system in clinical periodontology. *J Clin Periodontol* 2012;39:239-48.
33. Fine DH, Mandel ID. Indicators of periodontal disease activity: an evaluation. *J Clin Periodontol* 1986;13:533-46.
34. Grbic JT, Lamster IB, Celenti RS, Fine JB. Risk indicators for future clinical attachment loss in adult periodontitis. Patient variables. *J Periodontol* 1991;62:322-9.
35. Haffajee AD, Socransky SS, Goodson JM. Clinical parameters as predictors of destructive periodontal disease activity. *J Clin Periodontol* 1983;10:257-65.
36. Goodson JM. Clinical measurements of periodontitis. *J Clin Periodontol* 1986;13:446-60.
37. American Academy of Periodontology statement on risk assessment. *J Periodontol* 2008;79:202.
38. Pihlstrom BL. Periodontal risk assessment, diagnosis and treatment planning. *Periodontol 2000* 2001;25:37-58.
39. Page RC MJ. Quantification of periodontal risk and disease severity and extent using the Oral Health Information Suite (OHIS). *Periodontal Practice Today* 2007;4:163-80.
40. Leininger M, Tenenbaum H, Davideau JL. Modified periodontal risk assessment score: long-term predictive value of treatment outcomes. A retrospective study. *J Clin Periodontol* 2010;37:427-35.
41. <http://www.dentotreat.com>. 3/11/2012.
42. Hirschfeld L, Wasserman B. A long-term survey of tooth loss in 600 treated periodontal patients. *J Periodontol* 1978;49:225-37.
43. Meador HL, Lane JJ, Suddick RP. The long-term effectiveness of periodontal therapy in a clinical practice. *J Periodontol* 1985;56:253-8.
44. Oliver RC, Brown LJ. Periodontal diseases and tooth loss. *Periodontol 2000* 1993;2:117-27.
45. Persson GR, Mancl LA, Martin J, Page RC. Assessing periodontal disease risk: a comparison of clinicians' assessment versus a computerized tool. *J Am Dent Assoc* 2003;134:575-82.
46. Persson GR, Matuliene G, Ramseier CA, Persson RE, Tonetti MS, Lang NP. Influence of interleukin-1 gene polymorphism on the outcome of supportive periodontal therapy explored by a multi-factorial periodontal risk assessment model (PRA). *Oral Health Prev Dent* 2003;1:17-27.
47. Martin JA, Page RC, Loeb CF, Levi PA, Jr. Tooth loss in 776 treated periodontal patients. *J Periodontol* 2010;81:244-50.
48. Martin JA, Page RC, Loeb CF, Kaye EK. Reduction of tooth loss associated with periodontal treatment. *Int J Periodontics Restorative Dent* 2011;31:471-9.
49. Cobb CM, Carrara A, El-Annan E, Youngblood LA, Becker BE, Becker W, et al. Periodontal referral patterns, 1980 versus 2000: a preliminary study. *J Periodontol* 2003;74:1470-4.
50. Krebs KA, Clem DS 3rd. A report from the American Academy of Periodontology. Guidelines for the management of patients with periodontal diseases. *Compend Contin Educ Dent* 2006;27:654-8.
51. Cohen RE. Position paper: periodontal maintenance. *J Periodontol* 2003;74:1395-401.
52. Martin JA, Page RC, Kaye EK, Hamed MT, Loeb CF. Periodontitis severity plus risk as a tooth loss predictor. *J Periodontol* 2009;80:202-9.
53. Axelsson P, Nystrom B, Lindhe J. The long-term effect of a plaque control program on tooth mortality, caries and periodontal disease in adults. Results after 30 years of maintenance. *J Clin Periodontol* 2004;31:749-57.
54. Persson GR, Attstrom R, Lang NP, Page RC. Perceived risk of deteriorating periodontal conditions. *J Clin Periodontol* 2003;30:982-9.
55. Apter AJ, Paasche-Orlow MK, Remillard JT, Bennett IM, Ben-Joseph EP, Batista RM, et al. Numeracy and communication with patients: they are counting on us. *J Gen Intern Med* 2008;23:2117-24.
56. Edwards A, Elwyn G, Mulley A. Explaining risks: turning numerical data into meaningful pictures. *BMJ* 2002;324:827-30.
57. Moore RA, Derry S, McQuay HJ, Paling J. What do we know about communicating risk? A brief review and suggestion for contextualising serious, but rare, risk, and the example of cox-2 selective and non-selective NSAIDs. *Arthritis Res Ther* 2008;10:R20.
58. Paling J. Strategies to help patients understand risks. *BMJ* 2003;327:745-8.
59. Rothman RL, Montori VM, Cherrington A, Pignone MP. Perspective: the role of numeracy in health care. *J Health Commun* 2008;13:583-95.
60. Garcia RI, Nunn ME, Dietrich T. Risk calculation and periodontal outcomes. *Periodontol 2000* 2009;50:65-77.