EVIDENCE FOR ACTIVE HOST CONTROL OF THE PERIODONTAL MICROFLORA

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INTRODUCTION

Many adults have encountered suspected periodontal pathogens and they have low levels of circulating antibodies as evidence of the encounter. But most of these adults do not have severe periodontal diseases and they also do not carry high numbers of these organisms in their mouths. In contrast, most periodontal patients have elevated levels of circulating antibodies to these suspected periodontal pathogens and they carry appreciable numbers of these organisms in their mouths. Decades of research involving viable culture of periodontal organisms have demonstrated association of specific marker organisms with periodontal diseases.

Smoking is a known risk factor associated with increased severity of periodontal diseases. Our study examined the relationship between infection with specific marker organisms *Porphyromonas gingivalis, Actinobacillus (Haemophilus) actinomycetemcomitans* and *Prevotella intermedia*, as detected with the Evalusite™ immunoassay (Kodak Canada Inc.), and the known risk factor of smoking in patients attending our specialist periodontal practices.

We anticipated that a diagnostic technology based on demonstration of specific microbial markers in samples from periodontal patients would provide measurements directly related to host control of the oral microflora. The absence or presence of members of the oral microflora would represent an outcome measure of the ability of the host to control the oral microflora in response to the factors of periodontal therapy and smoking.

MATERIALS & METHODS

Our study examined the relationship between infection with specific marker organisms *Porphyromonas gingivalis, Actinobacillus (Haemophilus) actinomycetemcomitans* and *Prevotella intermedia*, as detected with the Evalusite™ immunoassay (Kodak Canada Inc.), and the known risk factor of smoking in patients attending our specialist periodontal practices. We report results for the first 55 patients of F-ME (non-smokers 24f, 18m; F-ME smokers 6f, 7m) and the first 179 patients of MHM (non-smokers 60f, 49m; MHM smokers 48f, 22m) who were assessed with the immunoassay. With its internal positive controls, the commercial immunoassay provides a reliable measurement of sites negative for all 3 marker organisms at the threshold level set for the assay. Aside from a clinically-identified need for periodontal assessment and therapy and the ability and interest of patients to pursue such diagnostic and treatment needs, there was no other process of selection of patients. General dentists had not referred patients specifically for microbiological assessment. The study underwent ethical review at the University of Alberta. Patients were mostly aged 35 to 55 years (F-ME mean 45.6, SD 13.0, median 41, range 16 to 73; MHM mean 43.5, SD 10.3, median 43, range 12 to 70).

Standard clinical assessment involved both the Periodontal Screening and Recording System PSR (American Academy of Periodontology & American Dental Association) and detailed periodontal probing as indicated by the PSR scores according to AAP & ADA Guidelines. We used mouth average PSR as an index of intraoral habitat favourable to growth of oral anaerobes.

Patients were identified as belonging to a Recall treatment group on the basis of a history of regular periodontal maintenance treatment (at least every 6 months) and absence of clinically detectable subgingival calculus or masses of subgingival plaque. Patients who did not meet these criteria, or who had no history of active periodontal maintenance were identified as belonging to a New treatment group (F-ME 19 New, 36 Recall; MHM 100 N, 79 R; EWM 7 N, 12 R). Mouth average PSR values were higher for new patients (PSR mean 3.1, median 3.2) than for recall patients (PSR mean 2.8, median 2.7).

There was no significant difference in the proportion of female and male non-smokers *vs* smokers in either practice. The distribution of ages was closely similar in non-smokers and smokers. For smokers in both practices mouth average PSR values peaked in the PSR range of 3 to 4, while for non-smokers this peak came in the PSR 2 - 3 range with a shoulder in the 3 - 4 range.
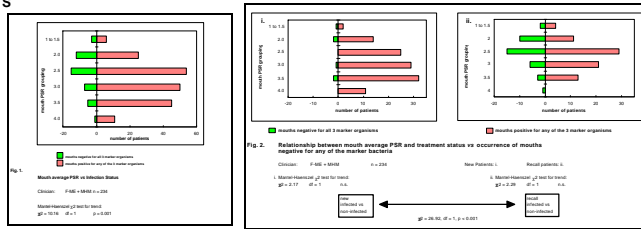
RESULTS

Fig. 1.

Fig. 2.

Fig.1. Infection Status *vs* Mouth average PSR
Fig. 2. Infection status *vs* Treatment Status and average PSR

Mouths negative for all 3 marker organisms are plotted in a negative direction along the horizontal axis. mouths positive for any marker organisms are plotted in a positive direction along the horizontal axis. groupings of mouth average PSR are plotted along the vertical axis.
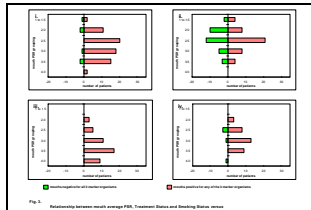Fig 1. With increasing periodontal health (average PSR) there is a significant increasing proportion of marker-negative mouths (combined groups n = 234).
Fig. 2. Patients undergoing regular periodontal maintenance treatment exhibit a significantly higher proportion of marker-negative mouths than untreated patients. There is no association of infection with increasing disease severity in patients undergoing regular treatment.

Figure 3. There was a significant relationship between prior periodontal treatment and increased proportions of marker-negative mouths.
There was also a significant difference between the proportion of marker-negative mouths in treated non-smokers *vs* treated smokers.

Table 1.
Logistic Regression to Examine the Effect of Smoking on Presence of Marker Bacteria

| F-ME + MHM		odds ratio	95% CI	P-value
Univariate analyses	Smoker *vs* non-smoker	5.2	2.0-13.9	0.0009
	New *vs* recall patient	8.9	3.6-22.2	0.0001
	Unit change in average PSR	2.5	1.4-4.4	0.0018
Multivariate model 1	Smoker *vs* non-smoker	5.6	2.0-15.5	0.0008
	New *vs* recall patient	9.4	3.7-23.7	0.0001
Multivariate model 2	Smoker *vs* non-smoker	5.0	1.7-14.4	0.0027
	New *vs* recall patient	8.7	3.4-22.4	0.0001
	Unit change in average PSR	1.3	0.6-2.6	0.4499

Table 1. Multivariate analysis helps to confirm the very different patterns of infection in periodontal patients undergoing regular treatment *vs* untreated patients. Fig. 2 shows that treatment is associated with increased clearance of marker organisms relative to untreated patients.

The analysis also helps to confirm that smoking inhibits the treatment-associated clearance of organisms to the extent that smokers are 5x more likely to be infected above immunoassay threshold levels than non-smokers. Periodontal therapy and smoking are associated with opposing effects on the occurrence of marker organisms at levels above the detection thresholds of the immunoassay.

Clinician	Patients	Status	number	Negative for all 3 markers *vs* any marker % -'ve	χ² df = 1	*P.i.* present in mouth *vs* not present in mouth % +'ve	c2 df = 1	*P.g.* present in mouth *vs* not present in mouth % +'ve	c2 df = 1	*A.a.* present in mouth *vs* not present in mouth % +'ve	c2 df = 1
Eggert	all	non-smoker	42	38%	2.5	57%	0.9	48%	2.36	14%	4.2*
		smoker	13	8%		77%		77%		46%	
McLeod	all	non-smoker	109	20%	7.0**	70%	10.5***	40%	1.24	30%	3.2
		smoker	70	6%		9%		50%		17%	
Eggert & McLeod	all	non-smoker	151	25%	12.4***	66%	13.6***	42%	2.6	26%	0.3
		smoker	83	6%		89%		54%		22%	

*** p < 0.001
** p < 0.01
* p < 0.05

Table 2.

Non-Smoking is Associated with an Increased Proportion of Marker-negative Mouths
There is no consistent pattern of association between smoking and infection with any of the 3 specific marker organisms
percentage values refer to proportion of total patients in a category
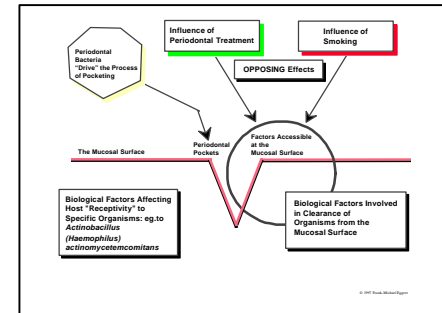
DISCUSSION

1) The significant trend toward increasing marker-negative mouths with decreasing disease severity (mouth average PSR) indicates that the natural trend in the periodontally-healthy dentition is toward marker-negative mouths (Fig 1. & 2.) below thresholds of the immunoassay.

2) There is a marked association between periodontal maintenance treatment and the occurrence of marker-negative mouths in periodontal patients (Fig. 2). The effect of periodontal therapy is to facilitate the development of marker-negative mouths (Fig. 2, Table 1) below detection thresholds of the immunoassay.

3) Smoking is associated with a significant inhibition of the clearance of marker organisms from periodontal patients undergoing treatment (Fig. 2, Table 1).

4) There is no consistent pattern of infection with any of these marker organisms with respect to smoking (Table 2). Other work indicates that specific monitoring of *A.a.* is important in a biological/clinical context distinct from the biological system affected by smoking (Eggert *et al.*, unpublished results).

CONCLUSIONS:

Immunoassay results identify problems with host defences at two different biological levels:
1) Smoking inhibits the ability of a periodontal patient to suppress the 3 marker organisms following conventional periodontal therapy and thereby complicates periodontal treatment through inhibiting its beneficial effects. Smoking interferes with defence mechanisms accessible at or within the mucosal surface.
2) Which of the three marker organisms are present at periodontal sites is determined by factors that appear to operate at a second level within host defences that is not accessible to the effects of smoking.
The opposing effects of smoking *versus* periodontal therapy reveal a dynamic balance between host mechanisms that lead to mucosal integrity and factors affecting growth of the mucosal microflora in the periodontal environment.

References: ‡Snyder, B, Ryerson, CC, Grogan, EA, Reynolds, HS, Contestable, PB, Boyer , BP, Mayer, K, Mangan, T, Norkus, N, Zambon, JJ, Genco RJ. Analytical performance of an immunologic-based periodontal bacterial test for simultaneous detection and differentiation of *Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis* and *Prevotella intermedia. J. Periodontology* 1996;67:497-505.