



MEETING ABSTRACT

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Smoking alters the antigenicity and infectivity of *Porphyromonas gingivalis*

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Background

Cigarette smokers are more susceptible to periodontal diseases and are more likely to be infected with *Porphyromonas gingivalis* than non-smokers. Furthermore, smoking is known to alter the expression of *P. gingivalis* surface components and to compromise IgG generation. The aim of this study was to evaluate if the overall IgG response to *P. gingivalis* is suppressed in smokers in vivo and if previously established in vitro tobacco-induced phenotypic *P. gingivalis* changes would be reflected in vivo.

Materials and methods

We examined the humoral response to several *P. gingivalis* strains as well as specific tobacco-regulated outer membrane proteins (FimA and RagB) by ELISA in biochemically-validated (salivary cotinine) smokers and non-smokers with chronic (CP, n = 13) or aggressive (AP, n = 20) periodontitis. We also monitored the local and systemic presence of *P. gingivalis* DNA by PCR.

Results

Smoking was associated with decreased total IgG responses against clinical (10512, 5607, and 10208C; all $p < 0.05$) but not laboratory (ATCC 33277, W83) *P. gingivalis* strains. Smoking did not influence IgG produced against specific cell surface proteins, although a non-significant pattern towards increased total FimA-specific IgG in CP subjects, but not AP subjects, was observed. Seropositive smokers were more likely to be

infected orally and systemically with *P. gingivalis* ($p < 0.001$), as determined by 16S RNA analysis.

Conclusions

Smoking alters the humoral response against *P. gingivalis* and may increase *P. gingivalis* infectivity, strengthening the evidence that mechanisms of periodontal disease progression in smokers may differ from non-smokers with the same disease classification.

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