

# ***1256 Effect of Periodontal Disease Severity on Altered Neutrophil Response in Diabetic Patients***

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Diabetes mellitus (DM) is a metabolic disorder characterized by hyperglycemia due to defective secretion or activity of insulin. Periodontitis presents a challenge in the control of DM, due to the infectious burden.

**Objective:** The aim of the present study was to evaluate superoxide production and protein kinase C activity in neutrophils of diabetic patients with periodontal disease.

**Methods:** Fifty diabetics were included and grouped into various degrees of periodontitis severity based on clinical and radiographic evaluation. Forty five healthy volunteers with no signs of periodontitis or diabetes were used as controls. Superoxide production and the localization of protein kinase C (PKC) were compared in resting and FMLP or PMA-stimulated neutrophils. Superoxide production was evaluated by reduction of cytochrome C, while PKC activity was measured by the phosphorylation of histone with radiolabeled ATP.

**Results:** Both resting and FMLP or PMA stimulated neutrophils from diabetic patients with severe periodontitis produced significantly more superoxide than diabetics without periodontitis and diabetics with mild or moderate periodontitis ( $p < 0.001$ ). The severity of periodontal disease positively correlated with control of diabetes as assessed by glycated hemoglobin levels ( $r = 0.71$ ). Total Ca-dependent PKC activity is increased in diabetics compared to healthy controls. The cellular distribution of Ca-dependent PKC is altered in diabetics with various severities of periodontitis (membrane associated). DM patients with severe periodontitis exhibited higher neutrophil PKC activity in the membrane fraction compared to diabetics without periodontitis and diabetics with mild or moderate periodontitis ( $p < 0.001$ ).

**Conclusions:** These findings suggest that the severity of periodontal disease correlates with poor diabetic control, increased superoxide production by neutrophils, and the altered subcellular distribution of Protein Kinase C. Supported by USPHS Grants DE13191 and DE14478.