0834 Protein Kinase C Activity in Neutrophils from Diabetic Patients With and Without Periodontitis

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Diabetes mellitus (DM) is a heterogeneous group of metabolic disorders characterized by altered glucose tolerance, and impaired lipid and carbohydrate metabolism. Periodontitis presents a challenge in the control of DM, due to the infectious burden, and periodontitis is also a complication of DM. Studies on the neutrophil response in diabetic patients in association with the presence of periodontitis are limited.

Objectives: The aim of the present study was to evaluate the protein kinase C activity and localization within neutrophils of diabetic patients with and without periodontal disease.

Methods: Twenty diabetics were included in this study and grouped into 10 diabetics without periodontitis and 10 diabetics with periodontitis based on clinical and radiographical evaluation. Twenty age-, sex-, and race-matched healthy volunteers with no signs of periodontitis or diabetes were used as controls. The localization of protein kinase C (PKC) was compared in unstimulated and FMLP and PMA-stimulated neutrophils. PKC activity was measured by the phosphorylation of histone with radiolabeled ATP ([γ-^{32}P] ATP).

Results: In normal cells, PKC (pmole) was primarily found in the cytosol of unstimulated cells (64.2±17.1) versus membrane (25.4±9.0) but translocated to the membrane-bound fraction after treatment with PMA (cytosolic: 29.2±9.3, membrane: 59.5±17.1). The enzyme translocation to the membrane was greater upon stimulation of the cells with PMA compared to FMLP. Examination of diabetic neutrophils revealed that total protein kinase C activity was higher in resting neutrophils from diabetics without periodontitis (150.2±58.9) and diabetics with periodontitis (186.7±58.7) compared to resting controls (89.6±23.2) ($p=0.002$) and that most of the PKC activity was associated with the membrane fraction.

Conclusions: These findings suggest that there is increased PKC activity in diabetic patients independent of periodontal disease status, and that this is due to activation of the cells in situ as evidenced by the translocation of PKC activity to the membrane.