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CALCIUM SIGNALING IN NEUTROPHILS AND LYMPHOCYTES AND ITS MODIFICATION BY INSULIN

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The goal of the study was to evaluate the process of neutrophil and lymphocytes activation via Ca mediated transduction signal into those cells and its modification by insulin. The studies was performed with the use of isolated peripheral blood neutrophils and lymphocytes from 30 healthy volunteers. Neutrophils were activated by bacterial peptide fMLP. Lymphocytes T were activated by anti CD3 antibodies. Intracellular Ca^{2+} concentration kinetics was assessed by flow cytometry with the use of Fluo3 and Fura Red fluorescent dyes. Data were collected in histograms displaying Fluo3 fluorescence vs. time and Fura Red fluorescence vs. time and mean channels of fluorescence intensity were used for calculation. fMLP induced highly significant Ca^{2+} mobilization in granulocytes ($p<0.0001$).Insulin increased both free and bounded calcium level ($p<0.05$). fMLP induced stimulation of insulin pretreated granulocytes resulted in the increase of free calcium ions but not bounded calcium. Proportion of free to bounded calcium increased significantly ($p<0.001$). Anti CD3 antibodies induced significant increase of free calcium ions in T lymphocytes ($p<0.0001$). Bounded calcium level remained unchanged. Insulin increased both free and bounded calcium concentration in T lymphocytes ($p<0.01$). We conclude that insulin is a potent immunomodulator and its signaling pathways are mediated by calcium ions concentration changes. Derangements in the concentration of intracellular calcium may represent a link between the mechanisms of insulin resistance in diabetes.