ORCHESTRATED EFFECTS OF CIGARETTE SMOKE AND LIPOPOLYSACCHARIDE ON PHAGOCYTOSIS AND CYTOKINE RELEASE FROM DESIALYLATED THP1CELLS

Adam Holownia, Przemyslaw Wielgat. Jan J. Braszko

Department of Clinical Pharmacology, Medical University of Bialystok, Poland

Sialyl residues on cell membrane may be relevant to airway inflammation. We examined phagocytosis and cytokine release from naïve and desialylated THP1 cells exposed to cigarette smoke (CS) and/or lipopolysaccharide (LPS). Naïve cells or cells grown for 24 hours with neuraminidase (100U/ml) were grown for additional 24 hours in CS-conditioned medium and/or in medium supplemented with LPS (1 µg/mL). Phagocytosis was tested using latex-IgG-FITC complexes and flow cytometry, cytokine levels in culture media was analysed with Cytokine ELISArray Kit. In naïve cells LPS and CS significantly decreased phagocytosis but there was no synergy between both agents. In desialylated THP1 cells phagocytosis was significantly higher than in naïve cells. LPS but not CS increased fluorescent marker uptake, by about 22%, while CS and LPS increased phagocytosis by more than 2 fold. CS significantly increased proinflammatory IL1, while neuraminidase treatment increased almost all measured interleukins but not TNF. LPS produced spectacular increases in several cytokines including proinflammatory IL1 and TNF. This effect was further enhanced by CS. In desialylated THP1 cells treated with LPS and CS all cytokines were very highly elevated. These data indicate that increased neuraminidase activity, especially in smokers may significantly amplify proinflammatory signaling.