

ADHESION MOLECULES, HSP 70 AND AUTOPHAGY IN HUMAN MONOCYTIC CELL LINE EXPOSED TO CIGARETTE SMOKE.

P. Szoka, A. Lukaszewicz, M. Cwiklinska and A. Holownia

Department of Pharmacology, Medical University of Bialystok, Poland

Cigarette smoke (CS) is considered as a critical factor in the progression and pathogenesis of a chronic obstructive pulmonary disease (COPD). Development of COPD is associated with activation of immune response, inflammation and tissue remodelling in which monocytes/macrophages are essential proinflammatory cells. Our study aimed to investigate the effects of acute CS exposition of monocyte/macrophage cell line (THP1 cells) on their integrin adhesion molecules, HSP70 levels, and autophagy. CS-conditioned medium (CSM) was prepared using full-strength Marlboro cigarettes. Cells were grown in CSM for 2h. In some experiments, cells were pretreated for 24h with redox-active compounds: L-buthionine-(S, R)-sulfoximine (BSO; 100 μ M) or n-acetylcysteine (NAC; 5 mM). Effects of CS on adhesion molecules (CD11b; CD18), HSP70, and autophagy (LC3) were assayed using epitope-specific monoclonal fluorescent antibodies and flow cytometry detection. CS-induced a significant increase in the expression of CD11b (about 60-fold) and CD18 (about 2.5-fold). We have also observed overexpression of HSP70 (about 4-fold increase) and elevated LC3 protein (about 4-fold). Similar effects were observed in cells pretreated with BSO only, which suggest that CS-induced changes can depend on oxidative stress. NAC partially attenuated effects of CS and normalized CD18, HSP70 and LC3 expression. Our results demonstrated that short exposure to CS provoked stress-response and activation of adhesion in THP-1 cells.