CIGARETTE SMOKE AFFECTS HUMAN ALVEOLAR BASAL EPITHELIAL CELL GROWTH (A-549 CELLS) IN CULTURE

P. Szoka, M. Cwiklinska, A. Lukaszewicz, A. Rybak, U. Baranowska and A. Holownia

Department of Pharmacology, Medical University of Bialystok, Mickiewicza 2C St, 15-222 Bialystok, Poland, Holow_sinai@hotmail.com

Cigarette smoke (CS) induces chronic inflammation of the airways and is considered as a key factor in the development and pathogenesis of chronic obstructive pulmonary disease. It was shown, that acute exposure to CS increases expression of proinflammatory mediators and may lead to chronic inflammation. In this study we aimed to evaluate the effect of CS on oxidative stress, glutathione (GSH) levels and autophagy (AF). AF plays an essential role in the inflammatory response and stress and may be critical for pulmonary inflammation and lung injury. Human alveolar epithelial cells (A549 cell line) were grown for 2 hours in CS-conditioned medium (CSM). Cytotoxicity (MTT test), oxidative stress (DCF fluorescence; FC), GSH, heat shock protein 70 (HSP70) levels and autophagy (LC3 expression; WB/FC) were measured. In cells cultured over 2 hours in CS-conditioned medium there was depleted GSH levels (by more than 30%) and by more than 2-fold increased ROS production. Cell viability was increased by almost 42%. Moreover a considerable overexpression of HSP70 was observed. Oxidative stress was also a major, but not the only one driving force for autophagy. There was a substantial accumulation of LC3 which increased by about 4-fold after CS exposure. Cell treatment with buthionine sulfoximine (BSO) which significantly depleted GSH, exerted similar effects to CS. Our data showed that autophagy is involved in CS-induced stress to alveolar epithelial cell line.