CYTOTOXICITY, OXIDATIVE STRESS AND AUTOPHAGY IN HUMAN ALVEOLAR EPITHELIAL CELLS (A549) EXPOSED TO URBAN AIR PARTICLES

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Exposure to urban airborne particulate matter (PM) has been associated with several adverse health effects but the mechanisms are not clear. In this work, we focused on cytotoxicity (MTT), oxidative stress (DCF/FC), DNA damage (PI/FC), necrosis/apoptosis (FC), and autophagy (LC3 expression; WB/FC) triggered by standardized urban dust (UD) in naïve A549 cells and in A549 cells with reduced glutathione (GSH) levels. The cells were grown in F12K/FCS media supplemented with coarse carbon black (CB; Huber 990; 260 nm diameter; 200 $\mu g \cdot mL^{-1}$) or urban dust (UD; Standard Reference Materials; 200 $\mu g \cdot mL^{-1}$). To deplete intracellular glutathione (GSH) l-buthionine-(*S*, *R*)-sulfoximine (BSO; 100 mM; 24 h) was used. Naïve or BSO-pretreated A549 cells were grown with CB or UD for 24 h and then cell viability and proliferation were measured with MTT and cell cycle/DNA damage and necrotic/apoptotic pathways and autophagy were assayed using WB and FC.A549 cells pretreated with BSO had depleted GSH by almost 30% and similar effect was observed after UD. CB was without significant effect on almost all parameters tested, except for LC3 expression (autophagy) which increased by about 2 fold. On the other hand, UD affected cell viability (decrease by 27%), significantly decreased cell proliferation in BSO pretreated cells, increased ROS production, and increased by about 2 fold both HSP70 and LC3 levels but most changes were not related to ROS-mediated GSH depletion. Our results indicate that UD-induced oxidative stress is important in PM toxicity but other mechanisms are also involved.