CO-EXPRESSION OF HSP70 AND LC3 PROTEIN IN THP-1 AND A549 CELLS EXPOSED TO AIR POLLUTION NANOPARTICLES.

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Airborne particles can cause serious health problems and are a risk factor for cardiopulmonary mortality. The aim of this study was to examine the effect of coarse carbon black (CB), urban dust (UD), nanoparticle carbon black (NPCB) and NPCB + benzo(a)pyrene (NPCB-BaP) on two intracellular proteins involved in cell proteostasis, the 70-kDa heat shock protein (Hsp70), and autophagosome marker protein - LC3B. Protein co-expression was tested in a monocyte/macrophage lineage (THP-1 cells) and human alveolar epithelial cells (A549 cells). The cells were grown for 24 hours in cell-dedicated culture media supplemented with 100 microg/mL of CB, UD, NPCB or NPCB-BaP and then were homogenized and analysed by WB or loaded with a green, FITC-labelled LC3B antibody and an Alexa Fluor 647 - antibody and were run on 2 colour flow cytometer, or on CellInsight CX7 High-Content Screening (HCS) Platform. Cell treatment resulted in different patterns of changes. In THP-1 cells Hsp70 was the highest (more than 3-fold increase) after UD, while LC3B was significantly stimulated by NPCB. UD increased mostly LC3B levels (more than 2 fold) in epithelial cells, while the highest increase in Hsp70 was found after NPCB. Our data indicate that HSP70-dependent autophagy may be relevant to particulate matter (PM) toxicity and possibly to oxidative stress and can be a PM- and cell type-specific.