CANONICAL AND NONCANONICAL AUTOPHAGY IN THP1 CELLS EXPOSED TO URBAN DUST;

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Exposure to air particulate matter can trigger inflammatory response and is a risk factor for cardiopulmonary mortality The aim of this study was to examine the effect of urban dust (UD) on autophagy (AF) a disassemby of dysfunctional intracellular components and on phagocytosis (Ph) in monocyte-derived macrophages (THP1 cells). AP and Ph operate in multiple clinical scenarios, are relevant to immune response and may be functionally and structurally interrelated (noncanonical AP; LAP) THP1 cells were grown for 24 hours in RPMI/CS media supplemented with 200 microg/mL-coarse carbon black (CB, Huber) or urban dust (UD; Standard Reference Materials) and then were loaded with a green, FITC-labelled Ph marker (Cayman Co) anti LC3/4 (AP marker) primary antibody and a cyanine Cy5-labelled secondary antibody and were run on flow cytometer or confocal microscope. Moreover, LC3 expression (WB), glutathione (GSH) levels (Cayman Co.) and cytotoxicity (MTT test) were done. Ph was gradually stimulated by increasing CB levels reaching almost two-fold increase vs control with 200 microg/ml CB. UD applied for 2 hours to the cells decreased Ph by about fold irrespectively of the dose. Both compounds were without significant effect on AP (FC and WB) in nave cells but increased LC3 levels in cells with depleted GSH (BSO-pretreated cells. Double staining revealed a heterogeneous, bimodal distribution of Ph/AP cells in BSO+UD-treated THP1 cells with a significant subpopulation of AP+/Ph- cells. Our results indicate that UD may affect canonical, LAP-dependent pathways in responsive THP1 cells with depleted GSH.