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Hazard assessment of polystyrene nanoplastics in primary human nasal epithelial cells, focusing on the autophagic effects

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The human health risks posed by micro/nanoplastics (MNPLs), as emerging pollutants of environmental/health concern, need to be urgently addressed as part of a needed hazard assessment. The routes of MNPL exposure in humans could mainly come from oral, inhalation, or dermal means. Among them, inhalation exposure to MNPLs is the least studied area, even though their widespread presence in the air is dramatically increasing. In this context, this study focused on the potential hazard of polystyrene nanoplastics (PSNPLs with sizes 50 and 500 nm) in human primary nasal epithelial cells (HNEpCs), which constitute the first line of cells acting as a physical and immune barrier in the respiratory system. Primarily, cellular internalization was evaluated by utilizing laboratory-labeled fluorescence PSNPLs with iDye, a commercial, textile pink-colored dye, using confocal microscopy, and found PSNPLs to be significantly internalized by HNEpCs. After, various cellular effects, such as the induction of intracellular reactive oxygen species (iROS), the loss of mitochondrial membrane potential (MMP), and the modulation of the autophagy pathway in the form of the accumulation of autophagosomes (LC3-II) and p62 markers (a ubiquitin involved in the clearance of cell debris), were evaluated after cell exposure. The data demonstrated significant increases in iROS, a decrease in MMP, as well as a greater accumulation of LC3-II and p62 in the presence of PSNPLs. Notably, the autophagic effects did indicate the implications of PSNPLs in defective or insufficient autophagy. This is the first study showing the autophagy pathway as a possible target for PSNPL-induced adverse effects in HNEpCs. When taken together, this study proved the cellular effects of PSNPLs in HNEpCs and adds value to the existing studies as a part of the respiratory risk assessment of MNPLs.

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Polystyrene nanoplastics; HNEpCs; oxidative stress; mitochondrial membrane potential; autophagy.