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Long-term effects of PS and PET nanoplastics in lung cell lines

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The exponential production of plastics, along with an inefficient recycling system, have resulted in an alarming accumulation of plastic waste. Degradability of these polymers in the environment results in the so-called micro and nanoplastics (MNPLs). In humans, besides ingestion which has already been pointed as an important route of exposure to MNPLs, attention is now paid to inhalation, considered also as a major source of exposure. Although MNPLs have recently been identified in the lungs of living people, very few is known about their chronic effects on health. This study tries to identify the long-term adverse effects of two of the most common plastic wastes: polystyrene (PS) and polyethylene terephthalate (PET) nanoplastics, (NPLs). For that, we have used two cell lines representing the proximal and distal lung epithelia: Beas-2B (bronchial cell line) and A549 (alveolar cell line) exposed to long-term treatments (up to 15 weeks). By performing a battery of in vitro assays, testing the ability of PS and PET to reach cell cytoplasm, their genotoxic potential, and some cell transformation hallmarks (proliferation rate, anchorage independent growth, migration potential, invasion ability or tumorsphere generation), we have observed the high ability of PS and PET NPLs to penetrate the cell, although no genotoxic nor carcinogenic potential have been anticipated. Transcriptomic data for both cell lines exposed to PS and PET under 24 h exposure and chronic exposure (15 weeks) was analysed to distinguish altered genes and pathways. Briefly, from these results we can highlight that A549 cell line reveals an upregulation of immune response and TNFA pathways after chronic exposure to both PS and PET, and a downregulation of mitotic spindle pathway in all the conditions. In Beas-2B cells, although the list of deregulated genes is higher, there is no clear pattern regarding specific altered pathways.

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