

PHOSPHORYLATION OF P53 PROTEIN IN LUNG ALVEOLAR EPITHELIAL CELLS (A549) PRETREATED WITH CISPLATIN AND EXPOSED TO URBAN DUST OR CARBON NANOPARTICLES

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Particulate matter (PM) triggers an inflammatory response and lung cancer. The tumor suppressor protein p53 plays a role in DNA repair. Our study aimed to examine the effect of coarse carbon black (CB), urban dust (UD), and nanoparticle carbon black (NPCB) on DNA damage and p-phosphorylation in A549 cells (alveolar epithelial cells). DNA integrity was assessed by propidium iodide (PI) DNA staining and flow cytometry (FC). Cell cycle-specific subpopulations were quantified and divided into resting (G₀) and proliferating (G₁/M) cells. Phosphorylated p proteins at Ser and Thr were quantified in both fractions using binary FC. A similar experiment was done on cells pre-treated overnight with 10 μg/mL cisplatin (CPT). Untreated A549 cells had constitutive p-Ser p53 and p-Thr p53 in interphase and mitotic nuclei. CPT increased p-Ser p53 by about 2-fold, but p-Thr p53 was increased only in quiescent cells. PMs produced several significant changes. The most relevant difference in phosphorylated p levels between naive and CPT-treated cells was observed in UD and NPCB-treated cells, where lower p-Ser p53 and p-Thr p53 response was observed in CPT-pretreated cells.