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 A 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 G G and proliferating G M cells Phosphorylated p proteins at Ser and were quantified in both fractions using binary FC A similar experiment was done on cells pre-treated overnight with g mL cisplatin CPT Untreated A cells had constitutive p Ser P p Ser P p Ser P and p Ser P in interphase and mitotic nuclei CPT increased by about - p Ser P p Ser P but p Ser P but p Ser P was increased only in quiescent cells PMs produced several significant changes The most relevant difference in phosphorylated p levels between nave and CPT-treated cells was observed in UD and NPCB-treated cells where lover p Ser P and p Ser P response was observed in CPT-pretreated cells.