MARKER OF NANOPARTICULATE CARBON BLACK-INDUCED DNA DAMAGE IN TYPE II LUNG EPITHELIAL CELLS (A549 CELL LINE)

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Air pollutant nanoparticles trigger DNA damage by direct interaction or via the ROS mechanism. Phosphorylation of histone protein H2A.X at residue Ser-139 (γH2A.X) by PI3K-like kinases is an early cellular response to the generation of DNA double-strand breaks (DSBs) and γH2A.X has emerged as a specific and sensitive molecular marker for DNA damage. The objective of our study was to evaluate markers of DNA damage in type II lung epithelial cells induced by nanoparticle carbon. A549 cells were grown for 24 hours with 200µg/mL NPCB or carbon black. H2A.X (mRNA and protein) and γH2A.X were quantified with WB and flow cytometry and RTPCR. Co-expression of H2A.X and γH2A.X and epitope distribution were also studied. NPCB but not CB increased oxidative stress and decreased cell proliferation. H2A.X mRNA was elevated by more than 2 fold in cells grown with NPCB when compared to cells treated with CB or control cells. H2A.X and γH2A.X were also higher in both WB and FC assays. Our results show that NPCB increases H2A.X mRNA and protein levels, and elevates γH2A.X