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Meeting abstract

PM_{10} from Mexico City (MC) induce senescence-like phenotype and ATM, γH2A.X and p53 activation linked to oxidative stress

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Background

Urban air particulate matter (PM) relates to adverse health effects including lung cancer. Previous work from our group demonstrated that the PM_{10} produce DNA damage, although the mechanisms involved are not known. DNA double-strand breaks (DSBs) are the most deleterious DNA lesions. DSBs arise from exogenous agents such as metals and hydrocarbons, or endogenous agents, such as reactive oxygen species (ROS). We explore the cellular response that senses DNA damage after cell exposure to PM_{10}, evaluating markers such as phosphorylation of ATM (ser1981), γH2A.X (ser139) and p53 (ser15), and senescence-like induction, as well as the identification of ROS co-participation in the response.

Materials and methods

A549 cells were exposed 24 h to 10 μg/cm^2 of PM_{10} from an urban-commercial zone from MC. ROS induction was evaluated by DCFH-DA assay. Senescence was determined by the β-galactosidase assay. ATM, γH2A.X and p53 activation and their co-localization on DNA damage foci were done by immunofluorescence. Experiments in the presence of Trolox as an OH· scavenger were done in parallel.

Results

Senescence-like was induced by PM_{10}. ATM, H2A.X and p53 were activated by PM_{10} and co-localize in DNA damage foci. Both cellular responses were prevented by Trolox.

Conclusion

Cells exposed to PM_{10} had a cellular response directly involved in sensing DSBs. There was a strong co-relation with the presence of ROS that suggests the participation of oxidative stress in the deleterious effects induced by PM_{10}. Senescence-like phenotype and ATM, γH2A.X and p53 activation conform an early cellular response that could evolve into the signaling and recruitment of repair factors, and the activation of enzymes of cell cycle checkpoints in cells exposed to PM_{10}. Further studies must be done to confirm that.

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