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ABSTRACT BOOK

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IN VITRO EVALUATION OF GENOMIC DAMAGE INDUCED BY 2.5 PARTICULATE MATTER ON HUMAN LYMPHOCYTES

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Background. The increased exposure to environmental pollutants has led to the awareness of the necessity for constant monitoring of human populations, especially those living in urban areas. Indeed, the prolonged exposure to high levels of urban pollution was associated with increased risk of cancer, especially lung cancer. In this scenario, biomarker-based studies may represent useful tools providing a better understanding of the contribution of atmospheric pollution to the overall genotoxic burden suffered by urban residents.

Aim. We analysed the effects of urban air fine particles (PM2.5) on human lymphocytes by in vitro Micronuclei (MNi) assay. MNi originate from acentric chromosome fragments or whole chromosomes that fail to segregate properly during mitosis and appear in the cytoplasm of interphase cells as small additional nuclei. Therefore, MNi assay allows the evaluation of both clastogenic and aneugenic effects of different xenobiotics and published studies provided evidences for a relationship between high levels of MNi in peripheral blood lymphocytes and increase of cancer risk.

Study Area. Meteorological-chemical stations were positioned in the urban area of Turin (Italy), a city located in the Po river valley, an area where air exchanges are limited by the surrounding mountains, winds are weak, and air pollutants can accumulate easily. For these reasons, Turin is one of the most polluted European cities and, for many years, the average annual PM2.5 pollution in Turin was higher than limits set by the WHO.

Methods. PM2.5 sampling was performed from January to December 2017, using a sampler, according to directive UNIEN14907. Peripheral venous blood was collected from 5 healthy subjects. Lymphocytes were exposed to four concentrations of PM2.5: 5, 10, 15 and 20 µg/mL. MNi, Nucleoplasmic Bridges (NPBs) and Nuclear Buds (NBDUs) were scored in 2000 binucleated lymphocytes per subject per concentration, whereas the Cytoleukin-Block Proliferation Index (CBPI) was calculated on a total of 1000 cells observed.

Results. PM2.5 significantly increased the frequencies of MNi, NPBs and NBDUs at all concentrations tested, with respect to controls. Vice versa, the CBPI was significantly reduced only at the concentrations of 15 and 20 µg/mL, indicating that the PM2.5 cytotoxicity threshold could be close to 15 µg/mL.

Conclusion. Although simple in vitro experiments cannot accurately mimic the complex in vivo kinetics of xenobiotic compounds, the results we obtained point to the necessity of further investigations in order to establish the adoption of more stringent measures reducing the presence of these pollutants in the environment.