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Genotoxic risk for workers from São Paulo city, Brazil, due to occupational exposure to traffic air pollution

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Introduction

- Exposure to particulate matter fraction with aerodynamic diameter $\leq 2.5 \mu m$ (PM$_{2.5}$) contributes to increased mortality and morbidity, inducing stress in the lung and causing aggravation of pre-existing diseases.

- Thousands of people exposed continuously to PM$_{2.5}$ the long term effects of this ubiquitous pollutant causes major problems to global public health.

- Cytogenetic studies reporting the frequency of Micronuclei (MN) in subjects exposed to polluted air during their workday provide a direct measure of the genotoxic impact on individual health.
Objective

- The aim of the present study was to evaluate the genotoxic effects of occupational exposure to vehicular PM$_{2.5}$ in healthy male subjects working in different locations of São Paulo city.
Casuistic

- Data collected between April 2010 and February 2012.

17 traffic controllers (TC)
26 taxi drivers (taxi)
15 workers from the Forestry Institute (FI)
Exposure Assessment Methods

Individual sampler particulate matter (PM$_{2.5}$)

Major components of fine particulate matter sampler with a flow rate of 4 Lpm, with impactor developed by the School of Public Health at Harvard, adapted for individual use at LPAE.
Micronuclei analysis

Peripheral blood collection → Lymphocyte isolation → Cell culture → Cytokalasin block

cytological smear

[Images of cells with micronuclei]
Statistical Analysis

- Qui square
- Regression analysis
- P values less than 0.05 were considered statistically significant.
Results

Casuistic

PM$_{2.5}$ load

Lowest
<25 µg.m$^{-3}$ .24h$^{-1}$

Middle

Upper
>39.6 µg.m$^{-3}$ .24h$^{-1}$

Genotoxicity Evaluation

Micronuclei frequency

Lowest

Middle

Upper
Results
Results

- No correlation was found between PM$_{2.5}$ load and Micronuclei frequency.
- Regardless of the professional group, we found individuals with low PM$_{2.5}$ exposure with high frequency of Micronuclei and others who, after high exposure, did not respond with the same intensity.
- MN lymphocyte correlates strongly with MN buccal mucosa.
Conclusion

- The genotoxicity observed in this and other studies following exposure to particulate matter can be explained by the toxicokinetics of inhaled particles and by epigenetics.
- Additional studies should be conducted to clarify the weight that each factor has on genotoxicity due to exposure to particulate matter in the air from large urban centers.

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