MT1a mRNA Expression in Human Lung Cells (BEAS 2B) After Airborne PM$_{10}$ and Copper Exposure: A Possible Biomarker for Asthma

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**ABSTRACT**

Puerto Ricans have the highest prevalence, morbidity and mortality of asthma than any other ethnic group in the U.S. and can be triggered by different stimuli such as airborne Particle pollution (PM). PM transfers transition metals, including cooper, to the human airways generating reactive oxygen species (ROS). Metallothionein protein (MT1A) is a free radical scavenger expected to be induced in lung cells by PM exposure; however, this has never been proven nor demonstrated. The hypothesis involves the amounts of PM$_{10}$ in 2004, which would be significantly higher in the urban sites during months of March and June, thereby increasing PM metal exposure and inducing MT1A gene at the urban site. PM$_{10}$ filters for Urban (Guaynabo) and rural (Fajardo) site were provided by the PREQB. Organic extracts were prepared from filters by means of Soxhlet Extraction and heavy metals were determined using Atomic Absorption Spectrometry. BEAS-2B cells were cultured and exposed to PM$_{10}$ during 4 hrs. RNA was isolated and relative mRNA levels of MT1A were determined by RT-PCR. Urban PM$_{10}$ concentrations were 35 ug/m$^3$ and 60ug/m$^3$ for the months of March and June, respectively. Concentrations of copper for this urban PM$_{10}$ were 29.6 ppm and 50.3 ppm, for March and June, respectively. Exposure to Cu leads to an MT1A expression in lung cells demonstrating its response to metal exposure. Cellular response for the mRNA expression of MT1A was greater in relative high inflammation markers than in PM$_{10}$ samples.

**Key Words:** asthma, lung cells, particle matter, metallothionein, copper

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