HUMAN EXPOSURE TO ENVIRONMENTAL XENOBIOTICS AND ITS INTERRELATION WITH OXIDATIVE DAMAGES AND CARDIOVASCULAR FUNCTION

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*Doutoranda: Início 2010/1

Introduction: Air pollution has been associated with significant adverse health effects leading to increased morbidity and mortality. In 2006, the World Health Organization (WHO) recommended a strict control of air quality, recognizing that global pollution is responsible for more than two million deaths per year. Epidemiological and experimental data have shown that exposure to air pollutants lead to increased cardiovascular events, so further studies are needed relating to human health and toxic effects of environmental pollutants. Inflammation due to oxidative stress may be one responsible mechanism, as suggested by both animal and human studies. The exact mechanisms linking the inhalation of ambient air particles to an acute exacerbation of cardiovascular disease are not completely understood. Alveolar inflammation induced by particles may either directly or via oxidative stress lead to systemic inflammation. The mechanisms linking air pollution and cardiovascular system have been attributed to oxidative stress.
Objective: Evaluate the possible relation between exposure to environmental particulate matter 2,5 µm and benzopyrene on oxidative damage and cardiovascular function in subjects with chronic exposure to environmental pollution. Environmental monitoring and quantification of biomarkers of exposure will be essential to show relation between pollutants and damage.
Materials and Methods: Exposed-control study with a group occupationally exposed to environmental pollutants, taxi drivers (n = 100) and nonoccupational exposed subjects to environmental pollutants (n = 50). The environmental monitoring will be conducted to quantify the levels of particulate matter 2,5 µm, benzopyrene, benzene, toluene and xylene. The biological monitoring will be accomplished through the quantification of urinary biomarkers cotinine and 1-hydroxypyrene. Oxidative stress will assessed through the blood quantification of biomarkers of lipid and protein damage (malondialdehyde and protein carbonyl, respectively), by the levels of reduced glutathione, as well as, by the antioxidant enzyme activities. The cardiovascular damage will be assessed by clinical and laboratory analysis. Cardiovascular clinical analysis will be conducted, for example, pulse oximetry and carotid ultrasonography. Inflammatory markers will be evaluated such as interleukins (IL-6 and IL-8), c-reactive protein, tumor necrosis factor. Additionally it will also be quantification carboxyhemoglobin.

Acknowldgements: Financial support from CNPq/Universal (Nº 479613/2009-5) and CNPq/MCT (Nº 479613/2009-5).