An increasing amount of research has focused on the role of oxidant/antioxidant imbalance occurs in lung pathologies due to exposure to polycyclic aromatic hydrocarbons (PAHs), the main constituent of air pollutants (1). The inert PAHs, (benzo(a)pyrene (BaP) and benzo(e)pyrene(BeP)), are metabolized locally in the lung epithelia (2) leading to the generation of harmful intermediates and reactive oxygen species (ROS) released by the inflammatory leukocytes, both neutrophils and macrophages (3). These ROS degrade polyunsaturated lipid, particularly of the cell membrane, forming malondialdehyde (MDA) (4). The traditional role for MDA is reacting with deoxyadenosine and deoxyguanosine in DNA, forming DNA adducts, which is mutagenic (5). The guanidine group of arginine residue condenses with MDA to give 2-aminopyrimidines, causing peroxidative injury through increased oxidative burden (6). The production of this aldehyde is used as a biomarker to measure the level of oxidative stress in an organism (7). The aim of this pulmonary study is spatial assessments of MDA production and to determine the sustainability of any observed effect in PAH exposure.

**Keyword:** intratracheally, MDA, oxidative stress, PAHs