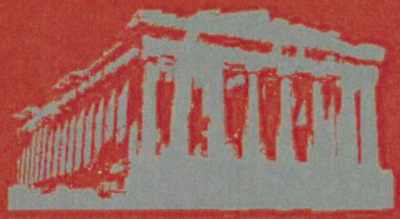

THE ATHENS INSTITUTE FOR EDUCATION AND RESEARCH

Abstract Book

13th Annual International Symposium on **Environment**

28-31 May 2018, Athens, Greece

Edited by
Gregory T. Papanikos

2018

Abstracts
13th Annual International Symposium on Environment
28-31 May 2018
Athens, Greece

Edited by Gregory T. Papanikos

Paola Palestini
Associate Professor, Università degli Studi di Milano-Bicocca, Italy

Oxidative Stress and Inflammation Induced by Acute and sub-Acute UFPs Exposures: Contribution to Neurodegenerative Disease Onset

Particulate matter (PM) is a complex mixture of solid and liquid particles suspended in the air, and this suspension could be formed by a variety of particles of different size and composition depending on their origin. Among the different fractions, ultrafine particles (UFPs) are thought to have the greatest health effects because of different characteristics: their small size, their high surface-to-volume ratio (high reactivity), their prolonged residence time in the lungs because of mitigated clearance efficiency, and their possible translocation rates across epithelial/endothelial barriers into the blood and lymphatic circulation. UFPs derive primarily from combustion processes in urban settings and, in the Lombardy Region, solid biomass burning for residential heating and diesel combustion used for private and public transport represent their major sources. Exposure to PM has been identified as the cause of several health effects including increased hospital admissions, emergency room visits, respiratory symptoms, exacerbation of chronic respiratory and cardiovascular diseases, decreased lung function and premature mortality.

Interestingly, emerging evidences from different studies suggest that neurological diseases, such as AD, PD and stroke, may be strongly associated with ambient PM. It has been demonstrated that continuous exposure to significant levels of airborne PM may result in the direct translocation of pollutants to different extra pulmonary sites, including central nervous system (CNS), or trigger the release of soluble inflammatory mediators from primary entry organs or secondary deposition sites. Systemic inflammation could activate cerebral endothelial cells, alter BBB integrity, or trigger signaling cascades that lead to the activation of mitogen-activated protein kinase (MAPK) and nuclear factor kappa-light-chain-enhancer of activated B cells (NFκB) pathways. Notably, post-mortem examinations of adult humans resident in highly polluted urban areas exhibited significantly higher brain COX-2 expression and accumulation of Aβ42 when compared to subjects living in cities with low pollution levels.

The aim of this presentation is to show the detrimental effect of UFPs exposure, regarding oxidative stress, inflammation and the possible physiopathological correlation between these two mechanisms and AD neurodegeneration, on *in-vitro* and *in-vivo* models of CNS.