DIESEL PARTICLES (DEP) EFFECTS ON AN ENDOTHELIAL CELL LINE (hCMEC/D3) AND HIPPOCAMPAL NEURONS (HT22)

Chiara Milani[1], Francesca Farina[2], Roberta Dal Magro[2], Laura Botto[2], Elena Lonati[2], Giulio Sancini[2], Alessandra Bulbarelli[2], Paola Palestini[2]

[1] PhD program in Neuroscience, Department of Surgery and Translational Medicine, University of Milano-Bicocca, Monza, Italy. [2] Department of Health Science, Polaris Centre, University of Milano-Bicocca, Monza, Italy. [3] Department of Health Science, University of Milano-Bicocca, Monza, Italy.

This research has received a financial support by the CARIPLO Foundation.

Alzheimer's disease (AD) is a neurodegenerative illness affecting the elderly population, characterized by plaques of Aβ42 aggregates, neurofibrillary tangles and neuronal loss (Allsop, 2000). In AD vascular factors could precede the neurodegenerative process (de la Torre, 2002, 2008); Aβ42 accumulation in the cerebral capillary may be a consequence of a local production in the vascular domain (Natté et al., 1999).

Air pollution has been associated with central nervous system (CNS) diseases. Inhaled UFPs (< 100 nm) could easily translocate cross the air-blood barrier, reach the bloodstream and be distributed to the cardiovascular system or the CNS (Oberdorster et al., 2002), thus affecting systemic microvasculature (Nurkiewicz et al., 2011).

hCMEC/D3 and HT22 cells have been exposed to different DEP concentrations for different times. The following parameters have been measured: cell viability, oxidative stress and inflammation markers (HO-1, iNOS, Cyp1b1, COX-2, TNFα, IL1β, IL8, VEGF), tight junction proteins (claudin-5, occludin), an amyloidogenic processing marker (BACE-1), besides an AD marker (Tau).

In both cell lines, none of the concentrations induced cytotoxicity. In hCMEC/D3, DEP caused increases in HO-1, COX-2 and BACE-1 levels; moreover, the lower dose elicit a significant VEGF release. In HT22, after 3h all the concentrations caused an increase in HO-1, iNOS, HSP70 and Cyp1b1, whereas after 24h iNOS and Cyp1B1 return almost to control levels. Finally, after 24h a decrease in Tau levels has been found.

In conclusion, all the parameters, except cytotoxicity, were differently affected in hCMEC/D3 and HT22, confirming the major susceptibility of neurons to toxic insults. Supported by Fondazione Cariplo.