Oxidative Potential of PM obtained at T0 and T1: An evaluation by EPR and DNA degradation

Raul Quintana Belmares, Instituto Nacional de Cancerología; Virginia Gómez Vidales, Instituto de Química, UNAM; Inés Vázquez López, Instituto Nacional de Cancerología; Geraldine Flores Rojas, Instituto Nacional de Cancerología; Yazmín Segura García, Instituto Nacional de Cancerología; Yesenia Sánchez Pérez, Instituto Nacional de Cancerología; Irma Rosas Pérez, Centro de Ciencias de la Atmósfera - Facultad de Medicina, UNAM; Ernesto Alfaro Moreno, Instituto Nacional de Cancerología; Claudia García Cuellar, Instituto Nacional de Cancerología; Javier Miranda Martín del Campo, Instituto de Física, UNAM; Andrea de Vizcaya, CINVESTAV, IPN; Violeta Mujica, UAM-Azcapotzalco; Benjamin de Foy, Department of Earth and Atmospheric Sciences, St Louis University; Luisa T. Molina, Molina Center for Energy and the Environment; Álvaro R. Osornio Vargas, Instituto Nacional de Cancerología, PUMA, UNAM.

Álvaro R. Osornio-Vargas, Instituto Nacional de Cancerología, PUMA, UNAM.
aosornio_06@servidor.unam.mx; aosornio@yahoo.com

Current working hypothesis on the mechanisms that mediate particulate matter (PM) toxicity reside on their capacity to induce oxidative stress on cells. This could be the result of direct PM effects on cells or the result of particle-cell interactions that trigger secondary intracellular signaling mechanism. Some PM metals and organics have been identified as components with the oxidative potential to cause those effects. However, further understanding of the relationships between particle composition and cellular effects is needed. As part of the MILAGRO campaign we sampled PM10 and PM2.5 at T0 and T1 in order to compare PM composition, oxidative potential, cellular effects and the plume ventilatory pattern with the intention to identify links among them. PMs were sampled on cellulose nitrate membranes using High Vol samplers, for 24 to 72 hrs, to be removed physically from the membranes and stored by day, size and site under desiccation in the dark at 4°C. Oxidative potential from each sample is measured by Electron Paramagnetic Resonance (EPR) using 3 mg dry samples and 1 mg/mL samples using the OH• trapper DMPO (0.2 M) and by evaluating the redox activity through the determination of the ability of PM to catalyze the consumption of dithiothreitol (DTT). Elemental analysis will be done on each sample by PIXE and organics by GC/MS. Generation of intracellular reactive oxygen species will be determined by fluorometry in macrophages exposed to PM. Cellular oxidative effects will be assessed on DNA evaluating its degradation by electrophoresis on agarose gels, in the presence and absence of H2O2. Ventilatory patterns will be calculated using FLEXPART and MM5 meteorological simulations. We are expecting to use complementary PM composition data from other groups (CENICA, CCA-UNAM) to further correlate with PM composition. Principal component multivariate analysis will be used to test for correlations. At the present time we have thirty one 24 hrs PM10 and PM2.5 samples collected at T0 and sixteen PM10 and PM2.5 samples collected at T0. Most of the samples at T1 were collected during 48-hrs period. We have recovered from the T1 membranes an average of 22 ±19.1 mg of PM10 and 1.9 ± 1.7 mg of PM2.5. EPR evaluation indicates that dry samples show a broad signal at g = 2.2 with $f´H$= 64 ±15

mT related to transition metals paramagnetic spices. Inside this broad signal, we identified a smaller one at g= 2.0031 ± 0.0002 mT and fnf'H= 0.5 ± 0.1 mT, related to organic components, probably to semiquinone free radicals. Signal intensities varied by day. At the present time we have identified in the PM mixture a couple of chemical species with the potential to induce oxidative stress. Experimentation using DMPO will give us the next set of data towards characterization of PM pro-oxidative potential in biological systems.