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Murine Pulmonary Responses after Sub-Chronic Exposure to Environmental Ultrafine Particles

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Abstract : Air pollution is one of the leading causes of premature death worldwide. Among air pollutants, particulate matter (PM) is a major health risk factor, through the induction of cardiopulmonary diseases and lung cancers. They are composed of coarse, fine and ultrafine particles (PM10, PM2.5, and PM0.1 respectively). Ultrafine particles are emerging unregulated pollutants that might have greater toxicity than larger particles, since they are more abundant and consequently have higher surface area per unit of mass. Our project aims to develop a relevant in vivo model of sub-chronic exposure to atmospheric particles in order to elucidate the specific respiratory impact of ultrafine particles compared to fine particulate matter. Quasi-ultrafine (PM0.18) and fine (PM2.5) particles have been collected in the urban industrial zone of Dunkirk in north France during a 7-month campaign, and submitted to physico-chemical characterization. BALB/c mice were then exposed intranasally to 10µg of PM0.18 or PM2.5 3 times a week. After 1 or 3-month exposure, broncho alveolar lavages (BAL) were performed and lung tissues were harvested for histological and transcriptomic analyses. The physico-chemical study of the collected particles shows that there is no major difference in elemental and surface chemical composition between PM0.18 and PM2.5. Furthermore, the results of the cytological analyses carried out show that both types of particulate fractions can be internalized in lung cells. However, the cell count in BAL and preliminary transcriptomic data suggest that PM0.18 could be more reactive and induce a stronger lung inflammation in exposed mice than PM2.5. Complementary studies are in progress to confirm these first data and to identify the metabolic pathways more specifically associated with the toxicity of ultrafine particles.

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