## Chemical composition and *in vitro* short-term exposure to air pollution fine and ultrafine particles induced cytotoxicity and metabolic activation of organic compounds in a human lung cell line

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Despite their little contribution to air particulate matter (PM) mass, ultrafine particles (UFP) are very abundant in the atmosphere (Terzano *et al* (2010)) and are currently emerging as the most abundant particulate pollutants in urban and industrial areas because of their numerous anthropogenic sources (Keogh *et al* (2009), Morawska *et al* (2008), Zhu *et al* (2002)). It has been shown that the smaller the size of the particles is, the more important their health effects are (WHO, 2013). However, the process involved in the PM toxicity, especially concerning UFP remains poorly understood.

Therefore, fine particles (FP) and UFP were collected at urban and rural coastal sites in Lebanon using a high volume Staplex<sup>®</sup> model 236 cascade impactor and were studied for their chemical composition and toxicological effects.

UFP were found more enriched in trace elements (Ag, Cd, Ni, Pb, Sb, Sn and V), secondary inorganic ions  $(SO_4^{2^-} \text{ and } NH_4^+)$ , total carbon and organic compounds (PAHs, PCDDs and PCDFs) when compared to FP collected at the same site.

For toxicological analysis, human bronchial epithelial cells (BEAS-2B) were exposed for 24, 48 and 72 h to increasing concentrations of FP, water-UFP suspension (UFP<sub>w</sub>) and UFP organic extract (UFP<sub>org</sub>). Our findings showed that UFP caused earlier alterations of mitochondrial metabolism and membrane integrity from the lowest particle concentrations. Moreover, a significant induction of CYP1A1, CYP1B1 and AhRR genes expression was showed after cells exposure to UFPorg (independently to particle concentration) and to a lesser extent to UFPw and FP samples (dose-dependent gene expression) indicating the induction of metabolizing enzymes expression which may increase the formation of PAHs-reactive metabolites, leading to an increase in DNA damages by adduct formation.

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