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Influence of Smoking on Fine And Ultrafine Air Pollution Pm in Their Pulmonary Genetic and Epigenetic Toxicity

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Abstract : In 2013, the International Agency for Research on Cancer (IARC) classified air pollution and fine particles as carcinogenic to humans. Causal relationships exist between elevated ambient levels of airborne particles and increase of mortality and morbidity including pulmonary diseases, like lung cancer. However, due to a double complexity of both physicochemical Particulate Matter (PM) properties and tumor mechanistic processes, mechanisms of action remain not fully elucidated. Furthermore, because of several common properties between air pollution PM and tobacco smoke, like the same route of exposure and chemical composition, potential mechanisms of synergy could exist. Therefore, smoking could be an aggravating factor of the particles toxicity. In order to identify some mechanisms of action of particles according to their size, two samples of PM were collected: PM0.03 2.5 and PM0.33 2.5 in the urban-industrial area of Dunkerque. The overall cytotoxicity of the fine particles was determined on human bronchial cells (BEAS-2B). Toxicological study focused then on the metabolic activation of the organic compounds coated onto PM and some genetic and epigenetic changes induced on a co-culture model of BEAS-2B and alveolar macrophages isolated from bronchoalveolar lavages performed in smokers and non-smokers. The results showed (i) the contribution of the ultrafine fraction of atmospheric particles to genotoxic (eg. DNA double-strand breaks) and epigenetic mechanisms (eg. promoter methylation) involved in tumor processes, and (ii) the influence of smoking on the cellular response. Three main conclusions can be discussed. First, our results showed the ability of the particles to induce deleterious effects potentially involved in the stages of initiation and promotion of carcinogenesis. The second conclusion is that smoking affects the nature of the induced genotoxic effects. Finally, the in vitro developed cell model, using bronchial epithelial cells and alveolar macrophages can take into account quite realistically, some of the existing cell interactions existing in the lung.

Keywords : air pollution, fine and ultrafine particles, genotoxic and epigenetic alterations, smoking
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