

Important Role of Air Pollution in the Ontopathogeny of Respiratory and Other Disorders

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Our interest in ecologic problems started with endocrine disruptors [1] and continued later in the field of nanotoxicology [2]. However, there is some overlap in classification of various types of particulate matter, including nanoparticles. In fact, environmental toxicology differentiates coarse, fine and ultrafine particles with aerodynamic diameter less than 10, 2.5 and 0.1 μm respectively, whereas nanotoxicology describes the effects of nanoparticles with dimensions less than 100 nm, i.e. coinciding with ultrafine particles. Therefore, nanotoxicologists add “engineered” or “manufactured” to the term of nanoparticles, in order to delineate better their object of research.

On the other hand, there exists some similarity in the properties and effects of asbestos fibers and carbon nanotubes [3], since the latter have only the diameter in nanoscale range, whereas their length attains several micrometers. If to compare these dimensions with the average size of human or animal cells (approximately 10 μm), it becomes clear that ambient and engineered particles and at least some fibers are able to interfere with biological functions of cells and tissues.

What for the area of pulmonology and respiratory medicine, it is already known that both fine and ultrafine ambient particles are respirable, but especially ultrafine particles are able to reach distal parts of airways and even penetrate through alveolar air-blood barrier. Moreover, they appear to be distributed with blood circulation to various organs, having the capacity to penetrate also through hematoencephalic barrier and placenta [4]. That's why, although the lungs are important targets for ultrafine ambient particles and engineered nanoparticles, the ecology problems caused by them are much more vast, extending to cardiovascular and neuropsychiatric areas and to possible adverse effects during gestation.

However, there is a difference in the importance of ambient and engineered particles. As a matter of fact, fine and ultrafine particles generated during incomplete combustion of fossil fuels in car traffic are principal agents of air pollution in modern great cities like Sao Paulo in Brazil, together with New York, Mexico-City and Santiago (Chile) [5]. Especially dangerous are diesel exhaust particles produced by tracks and autobus engines. Therefore, the extent of air pollution by particulate matter tightly correspond to health problems in respiratory, cardiovascular and neuropsychiatric areas, including bronchial asthma, lung cancer, ischemic heart disease, stroke and others [6,7].

On the other hand, engineered nanoparticles and carbon nanotubes have only potential toxicologic importance, but since the volumes of their production and use are rapidly increasing worldwide, it is essential just now to study their effects on animals and humans.

Some time ago we have offered to use a term “ontopathogeny” in the framework of developmental origins of health and disease (DOHaD) [8]. It means that the main focus should be made on long-term and cumulative effects of etiologic and risk factors, along the whole scale of pre- and postnatal development, continuing to adult state, middle age and senescence. So, our last research efforts were mainly directed to studying the age-related dynamics and gender differences of respiratory, cardiometabolic and other disorders, especially as referred to the role of stress hormones and proteins in the mechanisms of programming/imprinting and embedding phenomena.

However, literature data clearly show that the main ecologic problems are generally man-made and at least partially related to air pollution. So our previous idea to differentiate pharmacotoxicologic programming/imprinting and embedding [9] should be largely refined, considering chronic effects of ambient particulate matter that may be described as toxicologic phenomena, without any pharmacologic component, although the last one may be important later on, during the stage of long-term treatment, e.g. with inhaled corticosteroids.

What for the mechanisms of adverse actions of particulate matter on the lungs, in the first place proinflammatory effects and oxidative stress are considered [6,7,10]. However, carbon nanotubes may interact with lipopolysaccharide and interfere in the clearance of bacteria by alveolar macrophages [11,12], thus adding new dimensions to quite complex problems of nanotoxicology.

In conclusion, air pollution may be considered as ecologic stress [13,14] and in this regard, future studies should be devoted to clarifying further the mechanisms of its long-term impact in the ontopathogeny of respiratory and other disorders, including the essential role of toxicologic programming/imprinting and embedding [9,15].

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