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Editorial

Health Effects of Urban Atmospheric Aerosols

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The impact of atmospheric particulate matter (PM), including $PM_{2.5}$, PM_{10}, and ultrafine particles (UFPs), on public health has become of great concern, particularly in urban locations. Epidemiological and toxicological studies have shown associations between both chronic and long-term exposure to urban PM and a plethora of adverse health effects, including airway damages, and cardiopulmonary disorders [1,2]. A recent review of epidemiological cohort studies has also highlighted a positive association between long-term concentrations of NO_2 (nitrogen dioxide) and mortality, but limited evidence for an association between O_3 (ozone) and mortality [3]. Nonetheless, a high level of heterogeneity between these cohort studies estimates has also been reported, which was considered a shortcoming for determining the precise nature (magnitude and linearity) of the associations of NO_2 and O_3 and mortality [3]. The body of existent literature has also shown that the different chemical and physical properties of urban PM may elicit different types and degrees of detrimental health effects [4]. Nevertheless, additional research is needed to identify the specific PM characteristics (e.g., size, emission sources, atmospheric concentration, and chemical characteristics) that contribute the most to its adverse health effects. Establishing the nature of the relationships between these aerosol/gases and health effects will pave the way for an in-depth knowledge and better estimation of the burden of disease due to these atmospheric constituents and provide a basis for designing effective air quality control strategies.

Within this context, the Special Issue "Health Effects of Urban Atmospheric Aerosols" is motivated by the need to address important issues concerning the various links between the oxidative potential and inflammatory effects of urban PM and its physicochemical properties, as well as the potential health risks of gaseous pollutants (namely, NO_2 and O_3). It comprises five peer-reviewed, open access articles spanning the main topics of the field. For example, Olstrup et al. [5] explores the seasonal variations in daily mortality associated with increases in the concentrations of PM_{10}, $PM_{2.5–10}$, BC (black carbon), NO_2, and O_3 in Stockholm, from 2000 to 2016. This study showed the existence of excess risks associated with PM_{10} and $PM_{2.5–10}$ during springtime, most possibly explained by a larger amount of road dust during this seasonal period. The excess risks associated with BC were unclear, whereas those for NO_2 were negative throughout the year, which was explained by the prevailing low atmospheric concentrations during the sampling period (average values < 20 $\mu g\ m^{-3}$) [5]. On the other hand, this study also showed that O_3 and its oxidative potential were particularly important in terms of daily mortality, namely during summer and autumn, indicating a higher degree of exposure during the warm seasons [5]. The acquired data also suggested the need for developing effective mitigation policies for non-exhaust emission, namely those of road dust particles. In another study, Xia et al. [6] performed a comparative assessment of the adverse health effects of $PM_{2.5}$ on acute exacerbation of chronic obstructive pulmonary disease (AECOPD) in two typical Chinese cities (Beijing and Shenzhen) with different levels of $PM_{2.5}$. According to the authors, $PM_{2.5}$ had non-negligible health effects on AECOPD in both cities, implying that adverse consequences in areas with relative low pollution levels cannot be overlooked [6].

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Xia et al. [6] also highlights that targeted policy actions are needed in both high-pollution and low-pollution locations aimed at mitigating the consequences of $PM_{2.5}$ on people with chronic obstructive pulmonary disease.

Nonetheless, it is well known that the health effects of fine particulate matter go beyond its mass, and all $PM_{2.5}$ constituents may not be equally toxic. There are additional air quality metrics (e.g., primary organic pollutants, secondary organic and inorganic aerosols, and trace metals, as well as more integrated metrics, such as the oxidative potential) that are also important when assessing the health effects of $PM_{2.5}$. In this regard, Shi et al. [7] investigated the contents of potentially toxic elements (V, Cr, Mn, Co, Ni, Cu, Zn, As, Cd, and Pb) and emission characteristics of $PM_{2.5}$ in soil fugitive dust (SFD) in six cities in the Yunnan–Guizhou Plateau region in China. The authors also assessed the associated health risks for the potentially toxic elements, aiming at prompting the development of emission control policies in the region. According to Shi et al. [7], the $PM_{2.5}$ in SFD in cities with relatively developed economies and industries (namely, metal smelting activities) had the highest levels of potentially toxic elements. In terms of health risks, Shi et al. [7] reported non-carcinogenic risks for children in all six cities (mostly associated with As, but also with Cr and Pb to a lesser extent), with ingestion being the main way to induce these risks. For carcinogenic risks of $PM_{2.5}$ in SFD, the authors reported a positive response associated with Cr in just two of the six cities, with inhalation as the main exposure route [7]. Overall, this study sheds light on the importance of considering the toxicity of fine particles in SFD attributable to potentially toxic elements, and the need to develop effective emission control policies for these $PM_{2.5}$ constituents. On a smaller geographical scale, Pachoulis et al. [8] also assessed the risk of exposure of a population of an industrialized residential area (Elefsina, Greece) to atmospheric pollutants (i.e., polycyclic aromatic hydrocarbons (PAHs) and toxic metals (Pb, Cd, As, Ni, Hg, Cu, Zn) and volatile organic compounds (VOCs)) originating mainly from industrial activities. The authors employed different mathematical approaches and took into consideration different parameters, such as age, gender, and daily average exposure time for assessing the health effects of the targeted pollutants. The results indicated that the acceptable limits for cancer risk provided by the Office of Environmental Health Hazard Assessment (OEHHA) of the California Environmental Protection Agency, the U.S. Environmental Protection Agency (EPA), and the World Health Organization (WHO) were not exceeded. Pachoulis et al. [8] also highlighted the need to employ more comprehensive mathematical models since the risk for potential carcinogenic and non-carcinogenic effects varied depending on the applied methodology.

There is also growing evidence that atmospheric PM can induce a variety of effects at the cellular level, such as inflammation and DNA damage, as well as genomic instability [9]. In this regard, Estonilo et al. [10] investigated the genotoxicity of $PM_{2.5}$ and $PM_{1.0}$ on human peripheral blood lymphocytes in Manila, Philippines. The authors reported that cells treated with $PM_{2.5}$ displayed a significantly higher micronucleus count (typically used to screen DNA damage in various tissues) compared to the cells treated with $PM_{1.0}$. These results were associated with the higher content of Cd, Ca, Pb, K, Na, and Zn in the $PM_{2.5}$ samples compared to those of the $PM_{1.0}$ samples [10].

Overall, the studies and the results discussed in this Special Issue will be of interest to the atmospheric research community, namely those interested in air quality outdoors and indoors, air particle toxicity, composition, and sources. It is expected that the research presented here will inspire new research questions to untangle the strong connections between air particles and their impact on human health.

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