

# Contribution of Aerosol Sources to Health Impacts

Daniele Contini <sup>1,\*</sup>, Ying-Hsuan Lin <sup>2</sup>, Otto Hänninen <sup>3</sup> and Mar Viana <sup>4</sup>

<sup>1</sup> Institute of Atmospheric Sciences and Climate (ISAC), National Research Council (CNR), 73100 Lecce, Italy

<sup>2</sup> Department of Environmental Sciences, University of California, Riverside, CA 92521, USA; ying-hsuan.lin@ucr.edu

<sup>3</sup> Department of Health Security, Finnish Institute for Health and Welfare (THL), P.O. Box 95, FI-70701 Kuopio, Finland; otto.hanninen@thl.fi

<sup>4</sup> Institute of Environmental Assessment and Water Research (IDAEA-CSIC), C/Jordi Girona, 18-26, 08034 Barcelona, Spain; mar.viana@idaea.csic.es

\* Correspondence: d.contini@isac.cnr.it

Atmospheric aerosol is one of the major leading environmental risk factors for human health worldwide, potentially causing several million premature deaths per year [1,2]. Several epidemiological studies evidence associations between exposure to PM and the onset of cardiovascular and respiratory diseases [3] as well as cardiopulmonary diseases and other adverse health effects [4]. Exact mechanisms leading to PM toxicity are still not completely understood; however, it has been observed that adverse health effects of atmospheric particles depend on their size and their chemical–physical properties, which are strongly linked to their emission sources. Specific components of aerosol, such as metals, organics, and black carbon, are believed to have relevant toxicological effects. Population exposure and inhaled doses play an important role so that spatial and temporal distributions of aerosols have a relevant influence on health effects [5]. Developing effective control strategies to reduce the environmental health risks associated with aerosols is one of the most challenging areas in current research. Therefore, it is becoming more and more important to gather information regarding the contribution to health indicators of specific air pollution sources and metrics to support robust environmental planning and enforcement of mitigation strategies [6–9].

This Special Issue includes 10 research papers [10–19] discussing recent advances in the studies on the influence of particulate matter from different sources and related health effects. The Special Issue covers analyses of air quality [10], health impact assessment [11,16], exposure [12,16], emission sources [13], epidemiology [14,17], toxicology [15] and other topics related to health effects of air pollution [18,19].

Dimitrova and Velizarova [10] used the high-resolution Atmospheric Dispersion Modelling System (ADMS)-Urban to investigate particulate matter pollution sources in the area of Sofia (Bulgaria) taking into account four main emission sources: point industrial, domestic heating, roads, and unorganised transport. The approach allowed the investigation of different scenarios useful for decision making. Results showed that the input of the domestic heating sources was the most significant, followed by the contribution of transport from the main road arteries, which was more relevant for PM in comparison with PAH, and area transport sources. The contribution of the domestic heating from wood and coal burning was highest in the city's outskirts and lower in the centre. The maximum of daily simulated concentrations appeared in clusters, regardless of the various meteorological conditions investigated. The highest concentrations of particulate matter occurred at in the north-western and southern parts of the city.

Matkovic et al. [11] estimated mortality, life expectancy, and years of life lost attributed to long-term ambient PM<sub>2.5</sub> pollution in two cities of Bosnia and Herzegovina: Tuzla and Lukavac. In Tuzla, the main PM source was the coal power plant, with 776 Mg/y of emissions. In Lukavac, several emitters contributed to air pollution: ammonia soda



**Citation:** Contini, D.; Lin, Y.-H.; Hänninen, O.; Viana, M. Contribution of Aerosol Sources to Health Impacts. *Atmosphere* **2021**, *12*, 730. <https://doi.org/10.3390/atmos12060730>

Received: 31 May 2021

Accepted: 4 June 2021

Published: 7 June 2021

**Publisher's Note:** MDPI stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.



**Copyright:** © 2021 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<https://creativecommons.org/licenses/by/4.0/>).

manufacturing (160 Mg/y), coke manufacturing, fertiliser, and water treatment facility (55 Mg/y), and cement production (27 Mg/y). Authors estimated that 16.20% and 22.77% of premature deaths were due to ambient PM<sub>2.5</sub> pollution in Tuzla and Lukavac, respectively. They also studied possible gains in life expectancy if current air quality legislation were implemented and a second scenario with PM pollution reduction to the WHO guidelines values. Calculations were performed with WHO's AirQ+ software, showing that life expectancy could increase by 2.1 and 2.4 years for those cities.

Wang et al. [12] investigated the changes in the network of mass rapid transit (MRT) systems and the number of automobiles in the Taipei metropolitan area (Taiwan). To gather information on the effects of these changes on PM<sub>2.5</sub> exposure for the residents using different modes of transportation, portable PM<sub>2.5</sub> sensors were used by volunteers to measure PM<sub>2.5</sub> concentrations while commuting in different modes of transportation. Results showed that exposure to PM<sub>2.5</sub> was higher when walking along the streets, compared to travelling in buses or cars. PM<sub>2.5</sub> concentrations were higher in underground MRT stations and inside MRT cars running in underground sections, compared to those in elevated MRT stations and inside MRT cars running in elevated sections. Riding motorcycles also was associated with high PM<sub>2.5</sub> exposure. Even with some limitations of the study arising from the use of optical PM<sub>2.5</sub> detectors and the number of volunteers involved, results allowed concluding that residents in the Taipei metropolitan area may still be exposed to high PM<sub>2.5</sub> during some forms of commuting, including riding underground MRT.

The investigation of the health effects of road traffic continued in the work of Crosignani et al. [13]. Here, the authors studied the nitrogen oxides (NO<sub>x</sub>) emitted by diesel vehicles in the town of Milan (Italy) and their health effects in terms of premature mortality, focusing on the year 2018. Diesel cars accounted for 32% of total circulating vehicles; 34% were petrol cars; 11% were light commercial vehicles; 3% were heavy commercial vehicles; 2% were buses; and 12% were L-category vehicles (two-wheelers, microcars, and similar). Results showed that diesel cars contributed 50% to the total car urban mileage and 86% to total car NO<sub>x</sub> emissions. A modelling dispersion system able to take into account chemical transformation was applied in two scenarios: actual emissions and Diesel Emission Standards Compliance (DESC) with EURO standard scenario. The average NO<sub>2</sub> reduction in the DESC scenario was 6.6 µg/m<sup>3</sup>. The exposure to this extra-concentration led to an attributable fraction of 4.4% of all natural deaths in the area studied.

Atmospheric particulate matter pollution can lead to an increase in the morbidity and mortality of cardiovascular and cerebrovascular diseases. The work of Fu et al. [14] studied this aspect in 16 districts of Shijiazhuang (China), considering the role of PM<sub>2.5</sub> pollution on mortality for cardiovascular and cerebrovascular diseases between 2014 and 2016. They used generalised additive models (GAMs), taking into account several confounding factors such as meteorology, seasonal effects, and holiday effects; the authors found an increase in relative risk of death from circulatory system diseases by 0.33% (95% CI: 1.0025–1.0041). The increase in relative risk had a spatial variability that followed the variability of PM<sub>2.5</sub> concentrations with the highest value 1.19% (95% CI: 1.0071–1.0168) in the eastern plain, more polluted and heavily populated, and the lowest value 0.21% (95% CI: 0.9981–1.0062) in the western mountainous area, having relatively less pollution and lower population density.

Lionetto et al. [15] investigated the toxicity of water-soluble PM<sub>10</sub> collected at an urban background site (Environmental–Climate Observatory; (ECO)) in Lecce (southern Italy) by using both in vitro and acellular tests. Specifically, they determined cytotoxicity (using MTT test on A549 cells), genotoxicity (using the comet assay), and intracellular oxidative stress on A549 cells exposed for 24 h to aqueous extracts of PM<sub>10</sub> samples. In addition, carbon content (OC and EC) of PM<sub>10</sub> and acellular determination of oxidative potential with DTT assay were performed to compare results of acellular and cellular biological assays as well as the influence of combustion sources. Cellular (oxidative stress and MTT results) and acellular determination of oxidative potential with the DTT assay, normalised by volume, were well correlated with carbon content, suggesting that combustion sources play an important role in determining cellular oxidative stress and cytotoxicity of PM<sub>10</sub>.

Even if the number of data was limited, genotoxicity results were well correlated with intracellular stress and MTT results. Results suggested that the outcomes of the different toxicity tests were influenced by chemical compositions and, consequently, by sources with combustions sources, mainly road traffic and biomass burning at this site, having a relevant role. In addition, PM-induced oxidative stress could be an important mechanism in determining some adverse health effects.

Indoor environments are very important in estimating pollution-related health risks because people spend most of their time indoors. Ouyang et al. [16] carried out a quantitative investigation of PM<sub>2.5</sub>-bound PAHs in a middle school classroom in the Xicheng (China) district from October 2016 to March 2017 to estimate the health risk for adolescents. Results showed that indoor PM<sub>2.5</sub> and PHAs concentrations correlated with outdoor concentrations and diagnostic ratios; moreover, the application of the positive matrix factorisation (PMF) source apportionment approach indicated that coal combustion was the main source of PAHs in the classroom environment. The average value of incremental lifetime cancer risk (ILCR) was estimated to be  $1.49 \times 10^{-6}$ , which indicated a potential health risk to students according to USEPA standards. Modelling of future scenarios indicated that this risk could decrease by 2021–2022, but further studies will be necessary to confirm this result.

Exposure to atmospheric pollution could influence respiratory and cardiovascular pathologies as well as mental health and circadian rhythms that are 24 h oscillations driven by a hypothalamic master oscillator that entrains peripheral clocks in almost all cells, tissues, and organs. Kim et al. [17] studied the long-term consequences of air pollution on mental health, using a natural experiment in Indonesia. Even if with some limitations of the study discussed by the authors and the need for further studies in different conditions, authors provided evidence of significant and persistent negative effects of air pollution on mental health even 10 years after exposure. An extra standard deviation in the pollution index raised the probability of clinical depression measured 10 years past exposure by almost 1%. Women, in particular, seemed to be more affected, with an increase in the likelihood of clinical depression and severity of symptoms, but some effects persisted for men as well. On a similar topic, Benedusi et al. [18] investigated circadian misalignment, triggered by industrialisation and modern lifestyles that have been linked to several pathological conditions, with possible impairment of the quality of life. Current studies on pollution-induced tissue damage correlated to circadian biology focus on how exposure to air pollutants disturbs the molecular clock and on how circadian rhythms influence susceptibility to external stressors. In this framework, Benedusi et al. [18] hypothesised that chronodisruption can exacerbate cell vulnerability to exogenous damaging agents such as ozone and particulate matter, two of the principal air pollutants. They suggested that the intersection of the circadian clock and environmental pollution susceptibility could be traced back to the efficiency of the antioxidant system in free radical detoxification processes.

Finally, the article of Sreenonchai et al. [19] dealt with the perception of risks related to air pollution investigating willingness to pay for self-protection and haze pollution management using face-to-face interviews in 250 householders in one urban and four rural areas of Chiang Mai Province (Northern Thailand). Results showed that risk perception of urban and rural respondents staying in lower areas demonstrated greater familiarity with health effects due to haze pollution. However, urban respondents had the least trust in local authorities' roles to deal with the serious haze. Respondents in all areas were mainly willing to pay for a mask for self-protection. The highest average price of willingness to pay was found in the urban area.

In summary, this collection of articles provides a valuable update on research concerning the influence of aerosol sources on health effects and toxicity indicators in different conditions, showing both how far the research community has arrived nowadays, and how much research work is still needed to advance further on this topic.

**Funding:** This research received no external funding.

**Institutional Review Board Statement:** Not applicable.

**Informed Consent Statement:** Not applicable.

**Data Availability Statement:** No new data were created or analysed in this study.

**Acknowledgments:** The editors would like to thank the authors for their contributions to this Special Issue, and the reviewers for their constructive and helpful comments to improve the manuscripts.

**Conflicts of Interest:** The authors declare no conflict of interest.

## References

1. Lubczyńska, M.J.; Sunyer, J.; Tiemeier, H.; Porta, D.; Kasper-Sonnenberg, M.; Jaddoe, V.W.; Basagaña, X.; Dalmau-Bueno, A.; Forastiere, F.; Wittsiepe, J.; et al. Exposure to elemental composition of outdoor PM 2.5 at birth and cognitive and psychomotor function in childhood in four European birth cohorts. *Environ. Int.* **2017**, *109*, 170–180. [[CrossRef](#)] [[PubMed](#)]
2. Lelieveld, J.; Evans, J.S.; Fnais, M.; Giannadaki, D.; Pozzer, A. The contribution of outdoor air pollution sources to premature mortality on a global scale. *Nature* **2015**, *525*, 367–371. [[CrossRef](#)] [[PubMed](#)]
3. Pope, C.A., III; Dockery, D.W. Health Effects of Fine Particulate Air Pollution: Lines that Connect. *J. Air Waste Manag. Assoc.* **2006**, *56*, 709–742. [[CrossRef](#)] [[PubMed](#)]
4. Sun, Z.; Zhu, D. Exposure to outdoor air pollution and its human health outcomes: A scoping review. *PLoS ONE* **2019**, *14*, e0216550. [[CrossRef](#)] [[PubMed](#)]
5. Korhonen, A.; Relvas, H.; Miranda, A.I.; Ferreira, J.; Lopes, D.; Rafael, S.; Almeida, S.M.; Faria, T.; Martins, V.; Canha, N.; et al. Analysis of spatial factors, time-activity and infiltration on outdoor generated PM2.5 exposures of school children in five European cities. *Sci. Total Environ.* **2021**, *785*, 147111. [[CrossRef](#)] [[PubMed](#)]
6. Ahmed, C.M.S.; Yang, J.; Chen, J.Y.; Jiang, H.; Cullen, C.; Karavalakis, G.; Lin, Y.H. Toxicological responses in human air-way epithelial cells (BEAS-2B) exposed to particulate matter emissions from gasoline fuels with varying aromatic and ethanol levels. *Sci. Total Environ.* **2020**, *706*, 135732. [[CrossRef](#)] [[PubMed](#)]
7. Viana, M.; Rizza, V.; Tobias, A.; Carr, E.; Corbett, J.; Sofiev, M.; Karanasiou, A.; Buonanno, G.; Fann, N. Estimated health impacts from maritime transport in the Mediterranean region and benefits from the use of cleaner fuels. *Environ. Int.* **2020**, *138*, 105670. [[CrossRef](#)] [[PubMed](#)]
8. Chirizzi, D.; Cesari, D.; Guascito, M.R.; Dinoi, A.; Giotta, L.; Donato, A.; Contini, D. Influence of Saharan dust outbreaks and carbon content on oxidative potential of water-soluble fractions of PM2.5 and PM10. *Atmos. Environ.* **2017**, *163*, 1–8. [[CrossRef](#)]
9. Cesari, D.; Merico, E.; Grasso, F.M.; Decesari, S.; Belosi, F.; Manarini, F.; De Nuntiis, P.; Rinaldi, M.; Volpi, F.; Gambaro, A.; et al. Source Apportionment of PM2.5 and of its Oxidative Potential in an Industrial Suburban Site in South Italy. *Atmosphere* **2019**, *10*, 758. [[CrossRef](#)]
10. Dimitrova, R.; Velizarova, M. Assessment of the Contribution of Different Particulate Matter Sources on Pollution in Sofia City. *Atmosphere* **2021**, *12*, 423. [[CrossRef](#)]
11. Matkovic, V.; Mulić, M.; Azabagić, S.; Jevtić, M. Premature Adult Mortality and Years of Life Lost Attributed to Long-Term Exposure to Ambient Particulate Matter Pollution and Potential for Mitigating Adverse Health Effects in Tuzla and Luka-vac, Bosnia and Herzegovina. *Atmosphere* **2020**, *11*, 1107. [[CrossRef](#)]
12. Wang, C.Y.; Lim, B.S.; Wang, Y.H.; Huang, Y.C.T. Identification of High Personal PM2.5 Exposure during Real Time Commuting in the Taipei Metropolitan Area. *Atmosphere* **2021**, *12*, 396. [[CrossRef](#)]
13. Crosignani, P.; Nanni, A.; Pepe, N.; Pozzi, C.; Silibello, C.; Poggio, A.; Conte, M. The Effect of Non-Compliance of Diesel Vehicle Emissions with Euro Limits on Mortality in the City of Milan. *Atmosphere* **2021**, *12*, 342. [[CrossRef](#)]
14. Fu, G.; An, X.; Liu, H.; Tian, Y.; Wang, P. Assessment of the Impact of PM2.5 Exposure on the Daily Mortality of Circulatory System in Shijiazhuang, China. *Atmosphere* **2020**, *11*, 1018.
15. Lionetto, M.; Guascito, M.; Giordano, M.; Caricato, R.; De Bartolomeo, A.; Romano, M.; Conte, M.; Dinoi, A.; Contini, D. Oxidative Potential, Cytotoxicity, and Intracellular Oxidative Stress Generating Capacity of PM10: A Case Study in South of Italy. *Atmosphere* **2021**, *12*, 464. [[CrossRef](#)]
16. Ouyang, R.; Yang, S.; Xu, L. Analysis and Risk Assessment of PM2.5-Bound PAHs in a Comparison of Indoor and Outdoor Environments in a Middle School: A Case Study in Beijing, China. *Atmosphere* **2020**, *11*, 904. [[CrossRef](#)]
17. Kim, Y.; Manley, J.; Radoias, V. Air Pollution and Long Term Mental Health. *Atmosphere* **2020**, *11*, 1355. [[CrossRef](#)]
18. Benedusi, M.; Frigato, E.; Bertolucci, C.; Valacchi, G. Circadian Deregulation as Possible New Player in Pollution-Induced Tissue Damage. *Atmosphere* **2021**, *12*, 116. [[CrossRef](#)]
19. Sreenonchai, S.; Arunrat, N.; Kamnoonwatana, D. Risk Perception on Haze Pollution and Willingness to Pay for Self-Protection and Haze Management in Chiang Mai Province, Northern Thailand. *Atmosphere* **2020**, *11*, 600. [[CrossRef](#)]