WG1 (doc version 07042020)

RESEARCH QUESTIONS

What is the influence of the atmospheric aerosol, including bioaerosol (pollen/bacteria vs virus), and more in general air pollution, in eliciting indirect systemic effects (linked to pro-inflammation and oxidation mechanisms of the lungs, and immunology alteration processes) increasing the vulnerability of the population to COVID-19? What are the factors discriminating indoors from outdoors (e.g., UV solar radiation, humidity, viral load)?

RATIONALE

Air pollution has been linked to acute respiratory inflammation, asthma attack, and death from cardiorespiratory diseases by various studies. In urban areas, in particular, fine particulate matter (particles with aerodynamic diameter less than 2.5 μm, PM2.5) is considered the leading environmental health risk factor globally, causing several million deaths per year. The atmospheric aerosol can be a concause of indirect systemic effects within the human body associated to pro-inflammation and oxidation mechanisms in the lungs and extra pulmonary organs, as well as to immune system alteration processes. In particular, the inflammation in the lung can interfere with mechanisms that clear pathogens (including viruses) from our respiratory tracts.

Although uncontrolled confounding effect might have biased the results, the possibility of a detrimental effect of air pollution and atmospheric aerosols on the prognosis of patients affected by the COVID19 deserves further investigation. A 2003 study on victims of the coronavirus SARS found that patients in regions with moderated air pollution levels (Air pollution index, API) were more likely to die than those in regions with low APIs. Although the interpretation between SARS data and APIs remained uncertain, the possibility that exposure to air pollutants may contribute to increase the vulnerability of the population to COVID-19 should be investigated with suitable epidemiological and toxicological approaches.

An important point deserving further attention is related to the understanding of the physicochemical properties of aerosol particles involved in the generation of inflammation within the human body. It is unclear what are the toxicological mechanisms associated to aerosols possibly eliciting adverse cellular effects, such as cytotoxicity through oxidative stress, oxygen-free radical-generating activity, DNA oxidative damage, mutagenicity, and stimulation of proinflammatory factors. In general, the evaluation of most toxicological studies shows that the smaller the size of PM the higher the toxicity through mechanisms of oxidative stress and inflammation and biodistribution on inhalation. This points out at the importance to investigate ultrafine particles (particles with aerodynamic diameter smaller than 100 nm, UFPs). However, their role has still to be clarified. Existing studies do provide evidence for short-term health effects linked to UFP exposure like pulmonary and systemic inflammation, autonomic tone and blood pressure, which may be at least partly independent of other pollutants concomitant exposure. Another important aspect if the chemical composition that influences in different way ecotoxicity, cytotoxicity, and genotoxicity so that different biological outcomes are expected even in cases of similar number and mass concentrations. In this regard, therefore, caution should be put when translating high values of conventional aerosol metrics such as PM2.5 and PM10 mass concentrations (which do not represent UFPs but include bioaerosol, pollen and fungal spores) into an inflammatory condition, potentially increasing vulnerability of the population. Finally, technological shortcomings should be considered, as current measurement techniques are hampered by the difficulty to capture spatiotemporal scales relevant for the considered processes without inducing any artifacts. Only a strong collaboration between traditionally different aspects of science, and in particular virologists, epidemiologists, toxicologists, aerosol scientists and technologists, and meteorologists, can properly answer the question.

REFERENCES

Bates et al: Asthma attack periodicity: a study of hospital emergency visits in Vancouver. Environ Res 1990, 51:51-70. 4. Schwartz j, Dockery DW: Particulate air pollution and daily mortality in Steubenville, Ohio. Am J Epidemiol 1992, 135:12-9. 5.

Dockery et al : Acute respiratory effects of particulate air pollution. Annu Rev Public Health 1994, 15:107-32. 6.

Schwartz et al: Particulate air pollution and hospital emergency room visits for asthma in Seattle. Am Rev Respir Dis 1993, 147:826-31.

World Health Organization, 2016. Ambient Air Pollution: A Global Assessment of Exposure and Burden of Disease. World Health Organization 2016, pp. 1–121 ISBN9789241511353.

Cohen, A.J.; Brauer, M.; Burnett, R.; Anderson, H.R.; Frostad, J.; Estep, K.; Balakrishnan, K.; Brunekreef, B.; Dandona, L.; Dandona, R. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015. The Lancet 2017, 389, 1907-1918.

GBD 2016 Risk Factors Collaborators. Global, regional, and national comparative risk assessment of 84 behavioral, environmental and occupational, and metabolic risks or clusters of risks, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. The Lancet 2017, 390, 1345–1422.

Gilles S. et al. Pollen exposure weakens innate defense against respiratory viruses. Allergy 2020, 75(3):576-587. doi: 10.1111/all.14047

Lelieveld,et al. The contribution of outdoor air pollution sources to premature mortality on a global scale. Nature 2015, 525, 367–371.

Health Effects Institute. Understanding the health effects of ambient ultrafine particles. HEI perspectives 3. Health Effects Institute, Boston, MA, 2013.

Cui, Y et al.: Air pollution and case fatality of SARS in the People's Republic of China: an ecologic study. Environmental Health: A Global Access Science Source 2003, 2:15

Costabile et al.: First Results of the “Carbonaceous Aerosol in Rome and Environs (CARE)” Experiment: Beyond Current Standards for PM10. Atmosphere 2017 8, 249

Gualtieri et al: . Is it the time to study air pollution effects under environmental conditions? A case study to support the shift of in vitro toxicology from the bench to the field. Chemosphere 2018, 207, 552–564.

Costabile et al. Evidence of association between aerosol properties and in-vitro cellular oxidative response to PM1, oxidative potential of PM2.5, a biomarker of RNA oxidation, and its dependency on the combustion aerosol Atmos. Env. 2019, 213, 444-455.

Lionetto et al.: Correlation of Oxidative Potential with Ecotoxicological and Cytotoxicological Potential of PM10 at an Urban Background Site in Italy, Atmosphere 2019, 10, 733; doi:10.3390/atmos10120733.

Cesari D. et al: Source Apportionment of PM2.5 and of its Oxidative Potential in an Industrial Suburban Site in South Italy, Atmosphere 2019, 10, 758; doi:10.3390/atmos10120758