
Can air pollution increase the risk of COVID-19?

OPINIONS

JØRN A. HOLME

Jørn A. Holme, chief scientist at the Department for Environment and Health at the Norwegian Institute of Public Health.

The author has completed the ICMJE form and declares no conflicts of interest.

MARIT LÅG

Marit Låg, senior researcher and adviser at the Department for Environment and Health at the Norwegian Institute of Public Health.

The author has completed the ICMJE form and declares no conflicts of interest.

JOHAN ØVREVIK

Johan Øvrevik, chief scientist at the Department for Environment and Health at the Norwegian Institute of Public Health and adjunct professor at the Department of Biosciences at the University of Oslo.

The author has completed the ICMJE form and declares no conflicts of interest.

KARAN GOLESTANI

E-mail: karan.golestani@fhi.no

Karan Golestani, specialist in infectious diseases and senior consultant at the Department of Infection Control and Preparedness at the Norwegian Institute of Public Health.

The author has completed the ICMJE form and declares no conflicts of interest.

Air pollution is a well-known risk factor for various diseases, and perhaps also for COVID-19.

Air pollution is one of the most critical environmental health risk factors worldwide, and a contributory factor to cardiovascular disease, impaired pulmonary development and function, COPD, and increased incidence and exacerbation of asthma [\(1\)](#). Fine particulate matter appears to cause most damage to health, but gases and larger road dust particles also contribute.

Air pollution probably also increases the risk of acute infections of the upper and lower respiratory tract [\(2, 3\)](#).

Increased risk of COVID-19

Several studies have reported an association between concentrations of particulate pollutants and COVID-19 incidence and mortality rates [\(4–7\)](#).

However, one should be cautious in interpreting such associations. The results of a recently published study indicate that the relationships may be due to confounders [\(8\)](#).

A number of viruses and bacteria from aerosols and dust particles have been identified by means of DNA and RNA sequencing, including SARS-CoV-2 RNA that has recently been found on particulate matter [\(9\)](#). Because of dilution effects, however, it is improbable that particulates in outdoor air would constitute a real means of transmission.

Increased susceptibility to SARS-CoV-2

Chronic exposure to particulate pollutants and elevated levels of nitrogen dioxide (NO₂) are associated with hypertension, cardiovascular disease, chronic pulmonary disease and diabetes, all of which are well-known risk factors for severe disease in COVID-19 [\(10\)](#).

SARS-CoV-2 uses the cellular receptor ACE2 (angiotensin-converting enzyme 2) for viral entry into cells. ACE2 is present on type II pneumocytes in pulmonary alveoli, but also in myocardium, kidneys and vascular endothelial cells [\(11\)](#). Exposure to cigarette smoke has been shown to upregulate the ACE2 receptor. Similarly, it has been shown that inhalation of particulate pollutants increases ACE2 receptor levels in rats and in human epithelial cells in vitro [\(12\)](#). Increased expression of the ACE2 receptor is believed to be a risk factor for severe clinical course in COVID-19 [\(11\)](#).

«Several studies have reported an association between concentrations of particulate pollutants and COVID-19 incidence and mortality rates»

Air pollution may also increase susceptibility to respiratory diseases via other mechanisms. It has been shown that air pollution promotes proinflammatory responses, reduces the functionality of pulmonary epithelial barriers, reduces mucociliary transport and leads to immune deficiencies, including a reduced

ability to recognise and eliminate pathogens (2). Air pollution may lead to a more pro-allergic Th2 response (type 2 T-helper cell) and delayed tissue restitution. Imbalance in the Th2 versus Th1 response is assumed to be an immunological factor that coincides with a more severe clinical course in SARS, MERS and COVID-19 (2, 3, 13). Hypercoagulability is central in the pathogenesis and clinical presentation of COVID-19, and there are indications that chronic exposure to air pollution may lead to a procoagulant state (14).

In summary, it appears that air pollution may be a contributory factor to increased incidence and risk of COVID-19 in the most polluted cities.

LITERATURE

1. Landrigan PJ, Fuller R, Acosta NJR et al. The Lancet Commission on pollution and health. *Lancet* 2018; 391: 462–512. [PubMed][CrossRef]
2. Horne BD, Joy EA, Hofmann MG et al. Short-term elevation of fine particulate matter air pollution and acute lower respiratory infection. *Am J Respir Crit Care Med* 2018; 198: 759–66. [PubMed][CrossRef]
3. Brugha R, Grigg J. Urban air pollution and respiratory infections. *Paediatr Respir Rev* 2014; 15: 194–9. [PubMed]
4. Fattorini D, Regoli F. Role of the chronic air pollution levels in the Covid-19 outbreak risk in Italy. *Environ Pollut* 2020; 264: 114732. [PubMed][CrossRef]
5. Wu X, Nethery RC, Sabath BM et al. Exposure to air pollution and COVID-19 mortality in the United States: A nationwide cross-sectional study. *medRxiv* 2020; 2020.04.05.20054502. [PubMed]
6. Bashir MF, Ma BJ, Bilal et al. Correlation between environmental pollution indicators and COVID-19 pandemic: A brief study in Californian context. *Environ Res* 2020; 187: 109652. [PubMed][CrossRef]
7. Magazzino C, Mele M, Schneider N. The relationship between air pollution and COVID-19-related deaths: An application to three French cities. *Appl Energy* 2020; 279: 115835. [PubMed][CrossRef]
8. Cox LA, Popken DA. Should air pollution health effects assumptions be tested? Fine particulate matter and COVID-19 mortality as an example. *Glob Epidemiol* 2020; 2: 100033. [PubMed][CrossRef]
9. Setti L, Passarini F, De Gennaro G et al. Searching for SARS-COV-2 on particulate matter: A possible early indicator of COVID-19 epidemic recurrence. *Int J Environ Res Public Health* 2020; 17: 2986. [PubMed][CrossRef]
10. Ambient air pollution: a global assessment of exposure and burden of disease. Geneva: World Health Organization, 2016.
<https://www.who.int/phe/publications/air-pollution-global-assessment/en/>
Accessed 11.11.2020.

11. Chung MK, Karnik S, Saef J et al. SARS-CoV-2 and ACE2: The biology and clinical data settling the ARB and ACEI controversy. *EBioMedicine* 2020; 58: 102907. [PubMed][CrossRef]
 12. Tung NT, Cheng PC, Chi KH et al. Particulate matter and SARS-CoV-2: A possible model of COVID-19 transmission. *Sci Total Environ* 2021; 750: 141532. [PubMed][CrossRef]
 13. Zhang YY, Li BR, Ning BT. The comparative immunological characteristics of SARS-CoV, MERS-CoV, and SARS-CoV-2 coronavirus infections. *Front Immunol* 2020; 11: 2033. [PubMed][CrossRef]
 14. Robertson S, Miller MR. Ambient air pollution and thrombosis. *Part Fibre Toxicol* 2018; 15: 1. [PubMed][CrossRef]
-

Publisert: 14 December 2020. Tidsskr Nor Legeforen. DOI: 10.4045/tidsskr.20.0843

Received 20.10.2020, accepted 11.11.2020.

Copyright: © Tidsskriftet 2025 Downloaded from tidsskriftet.no 26 December 2025.