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Can air pollution increase the risk of COVID-19?

OPINIONS

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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 (NO_2) 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).

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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.

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