Air pollution and COVID-19



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Air pollution is an important health issue in Hong Kong and all over the world. Short-term and long-term exposure to particulate matters in ambient air pollution has been shown to be associated with increased mortality from all causes, cardiovascular and respiratory diseases.[1] Increased risk of respiratory viral infections has also been reported with exposure to air pollution.[2] During the period of severe acute respiratory syndrome (SARS) outbreak, ecologic studies showed that air pollution was associated with an increased mortality from SARS.[3] It is timely to examine the relation between air pollution and SARS-CoV2 infection during the current pandemic of COVID-19.

High rates of COVID-19 infection and mortality has been reported to be associated with short-term and long-term exposure to air pollution.[4] Fine particulate matter, PM2.5 of size <2.5, and nitrogen dioxide (NO2) are the most reported air pollutants that contribute significantly to the risk of COVID-19. Exposure to particulate matters has also been suggested to facilitate SARS-CoV-2 transmission with these particulate matters acting as carrier through the aerosol.[5]

However, many ecological studies share methodological shortcomings relying mainly on measures of exposures and outcomes without adequate adjustment for confounding factors.[6] A recent study that examined the association between air pollution exposure and COVID-19 incidence has addressed this potential source of bias by statistical adjustment to neighbourhood-level measures such as racial group, socioeconomic status, risk factors for COVID-19 transmission and community transmission among 140 neighbourhoods in Canada.[7] In this study, reactive oxygen species (ROS) generation in human lung epithelial lining fluid was applied as an indicator of oxidative stress in the exposure metrics.[8] ROS generated by COVID-19 in this system was shown to be attributed by the metals of copper and iron in PM2.5 but not the PM2.5 mass.

Stieb et al found that ROS, proportion of black residents and prevalence of crowded housing as the factors that correlated significantly with the incidence of COVID-19. Spatial patterns of ROS reflected a source of metal-containing particulate matter from railyards and brakewear in Toronto. In subgroup analysis, ROS was found to be linked to sporadic cases but not outbreak cases, in particular young male aged below 50. They postulated that the elderly and female subpopulation may spend more time indoor and, thus, have lower likelihood of exposure to both COVID-19 and outdoor pollution. The finding of higher COVID-19 in neighbourhoods with higher incidence of black residents echoed the higher mortality reported in countries with a larger proportion of Black communities and the authors postulated that the associated lower socioeconomic status and crowded housing created more barriers to self-isolating and social distancing in this subpopulation.[9]

As current literature demonstrated a positive association between incidence and mortality of COVID-19 and high exposure to PM2.5 and NO2 in air pollution, Frontera et al postulated a double-hit hypothesis to explain the susceptibility to SARS-CoV-2 infection.[10] Chronic exposure to PM2.5 leads to increased expression of the angiotensin-converting enzyme (ACE)-2 in the lung which is a receptor for SARS-CoV-2. The binding between spike protein of SARS-CoV-2 and the membrane bound ACE-2 causes downregulation of ACE-2 expression resulting in loss of various protective functions of ACE-2 such as anti-inflammatory effects in immune response. The second hit may be achieved by ambient NO2 in air pollutant which results in severe SAR-CoV-2 infection and worse outcome. With such intriguing hypotheses, more in vitro and in vivo mechanistic studies are required to confirm these postulations so that environmental measures can be taken with the addition goal to prevent major viral infection for populations residing in areas with suboptimal air quality.

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