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Association Between $PM_{2.5}$ Induced Diseases and COVID-19: A Systematic Review

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ABSTRACT

A novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) caused a global pandemic that started in China (Wuhan, Hubei region) in December 2019, called Coronavirus disease. This systematic review intends to evaluate the correlation of pre-existing particulate matter ($PM_{2.5}$) induced comorbidities along with COVID-19 spread and mortality. A search was operated to report the association between $PM_{2.5}$ and COVID-19 outbreak and evaluating the $PM_{2.5}$ related disease affected by COVID-19 infection. The research was conducted in consent with the criteria of PRISMA (Preferred Reporting Items for Systematic Reviews, and Meta-Analyses). We filtered the review and research articles published only in the English language and selected these keywords: air pollution, particulate matter, COVID-19, health impact. We obtained a total of 27 appropriate published articles in their final version. Additional articles were rectified by searching through Scopus, PubMed and Google Scholar. We concluded that the values of coagulation biomarkers in all SARS-CoV-2 patients were considerably higher as compared with healthy people. It was noted that Hypertension, Diabetes, COPD, CVD, Asthma and Cancer possess an evident relation with COVID-19 severity. Globally, air pollutants affect the body's immunity, leading to people being more susceptible to pathogens. In addition, transmission from person-to-person dynamic of the new respiratory virus was considered the environmental factors' role in accelerating coronavirus spread and its lethality. COVID-19 patients with pre-existing comorbidities induced by particulate matter show a high risk of mortality as compared to COVID-19 patients without these comorbidities.

INTRODUCTION

A novel (COVID-19) pandemic caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-COV- 2) emerged from Wuhan, China in December 2019 and spread all over the world (Han et al. 2020). The diameter of Coronavirus is approx.70–90 nm, single-stranded RNA viruses (He et al. 2020). The life cycle of SARS CoV-2 in human lung cells is completed in several stages including infiltration, inhalation, in the upper respiratory tract (Mallapaty 2020). Therefore, it is necessary to examine the various correlated issues related to particulate matter that are being introduced in this section.

$PM_{2.5}$ and COVID-19

Air pollution exposure has become a main public health issue due to increasing sources of pollutants (Kim et al. 2015). The particulate matter ($PM_{0.1}$, $PM_{2.5}$, and PM_{10}) can stick to viruses. If the virus carrying particulate matter inhaled, may transport the virus in the alveolar and tracheobronchial region and may weaken the immunity (Qu et al. 2020, Chauhan & Singh 2020). The Italian Society of Environmental Medicine launched a discussion paper on 16 March, for the first time presumed a possible correlation between the higher particulate matter concentration ($PM_{2.5}$ and PM_{10}) and the impact of COVID-19 pandemic in Northern Italy, in association with its weather conditions. These observations revealed that ambient particulate matter could not serve as a "boosting factor" but at least imitate as an indicator for COVID-19 severity in terms of health outcomes seen in Northern Italy (Setti et al. 2020, Badawi & Ryoo 2016).

Impact of Climate on COVID-19 Spread

The local environment such as weather, particle source, microenvironments influence PM exposure (Kim et al. 2015). It seems that air pollution plays a role in the airborne spread of SARS-CoV-2 and in the severity of coronavirus (Dehghani et al. 2017, Deepak et al. 2020). In this context, it may be assumed that a high level of air pollutants in an atmosphere together with some climatic parameters might

encourage longer stability of the virus in the air. It would support an indirect dispersal of SARS-CoV-2 with the direct physical spreading of the virus in person to person (Copat et al. 2020). Ho et al. reported that long-term $PM_{2.5}$ exposure affects the cardiovascular and respiratory system, and some COVID-19 symptoms may increase the chance of mortality in COVID-19 patients (Ho et al. 2020, Comunian et al. 2020).

Particulate matter is regulated in the environment, but PM less than 2.5µm is of main concern because of its greater penetration into the alveoli of the lungs and high reactive surface area. It causes cardiovascular disease including thrombosis and endothelial dysfunction (Raji et al. 2020, Hampel et al. 2015). Emerging evidence established correlation between environmental factors (climate, air pollution, chemical exposures, built environment) and transmission of SARS-CoV-2 susceptibility and severity (Weaver et al. 2022, Wang et al. 2022, Diffenbaugh 2022).

Effect of Particle Size and Particle Components

After inhalation, the deposition of particles in the respiratory tract depends on the pattern of breathing and particle size. A particle diameter of more than 10 µm (PM_{10}) can pass through the nose but too broad to reach the respiratory tract. But fine particles ($PM_{2.5}$) and ultrafine particles ($PM_{0.1}$), can deeply penetrate the human lungs and then deposited in alveolar regions at the time of mouth breathing (Dehghani et al. 2017). That's why the researchers and the government of countries should estimate exposure of $PM_{2.5}$ in areas exposed to the high levels of particulate pollution, at the time of framing policies to overcome the rate of COVID-19 (Dehghani et al. 2017, Kamf et al. 2020, Leikauf et al. 2020).

MATERIALS AND METHODS

We followed a systematic review method to report the association between $PM_{2.5}$ and COVID-19 outbreak and evaluating the $PM_{2.5}$ related disease affected by COVID-19 infection. The research was conducted in consent with the criteria of PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses), and the Flow Diagram is shown in Fig. 1. The research was guided in the Scopus database using Advanced Search Builder, and the keywords were searched in (Abstract OR Title). We have filtered the review and research articles published only in the English language and selected the keywords: air pollution, particulate matter, COVID-19, health impact. We have excluded articles, short surveys, letter, opinion, commentary, or non-relevant articles. We obtained a total of 27 appropriate published articles in their final version. For some of the papers, we select only principal findings that precisely fit the aim of this review.

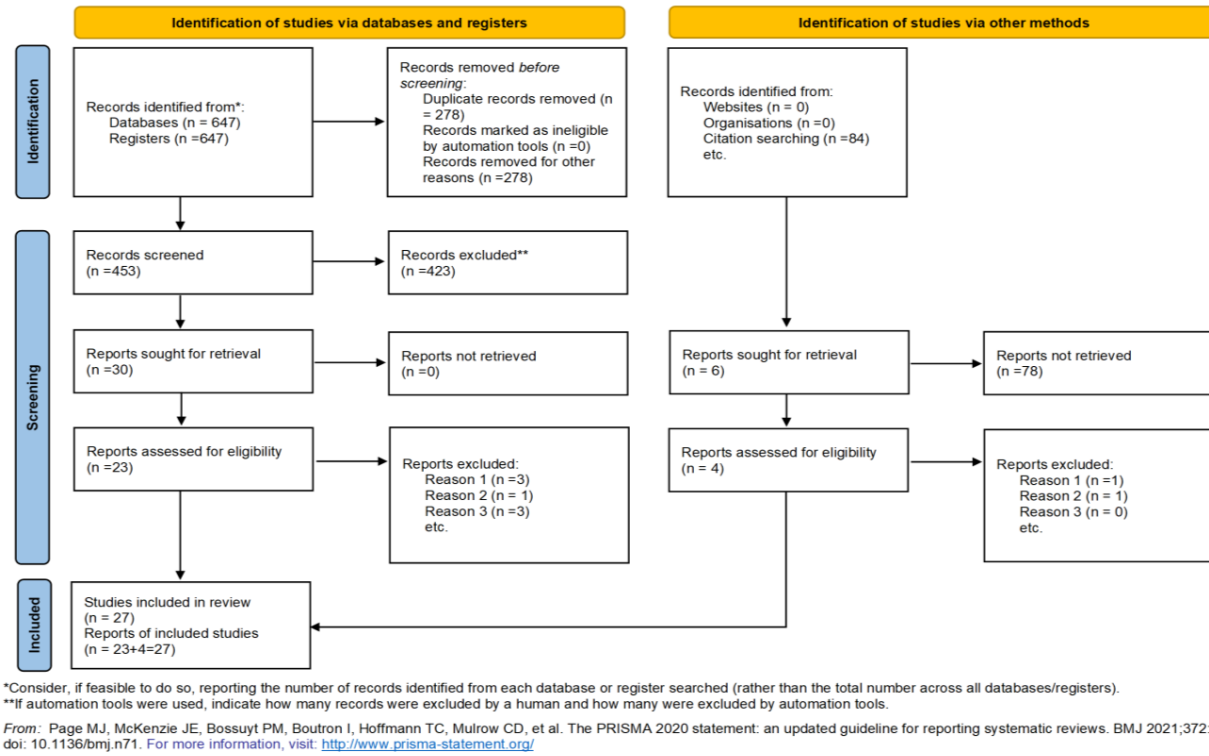

Fig. 1. PRISMA flow diagram of identification, screening, and inclusion of studies.

Morbidity/Mortality Evidence on the Correlation between $PM_{2.5}$ and COVID-19

It can be assumed that SARS-CoV-19 using a species of "highways", made up of atmospheric particulates helps in increasing its indirect viral transmission. The air pollutants exposure can cause oxidative stress, lead to free radicals' production, which in turn might damage the respiratory system, and results in decreasing the resistance towards viral infections. The COVID-19 pandemic is spreading via particulate matters, air, and aerosols which act as a major factor in mediating severity in COVID-19 confirmed cases because the SARS-CoV-2 virus can prolong survival in aerosols for up to 3 hours (Marques & Domingo 2020). Air pollution is positively associated with Angiotensin-Converting Enzyme 2 (ACE-2) receptor (that behaves as a binding receptor for SARS-CoV-2) in respiratory cells of humans and that may speed up the severity in susceptible people from Coronavirus Disease leading to death (Agarwal et al. 2020, Morawska et al. 2020, Zeng et al. 2022).

Significantly, the seriousness of COVID-19 is dependent on the interconnections between viral infection and chronic diseases such as respiratory and cardiovascular diseases which are themselves impacted by environmental stressors (Barouki et al. 2021). Outdoor airborne transmission probability depends on a few parameters, still quite uncertain: viability and lifetime, virus-loaded aerosol concentrations, and the minimum dose required to spread the disease (Marques & Domingo 2020). Therefore, particulate matter pollution should be lessened in regions where positive cases of COVID-19 are unpredictably high (Paital et al. 2020, Newby et al. 2015). The morbidity and mortality may be understood by a systematic framework shown in Fig. 2.

Viral Transmission and Symptoms

COVID-19 transmission appears to spread human–to–human when a person is in the stage of showing symptoms or incubation stage. Transmission occurs via touching non-living objects, and infected surfaces (skin-to-skin) (He et al. 2020). COVID-19 viruses may survive on sterile sponges, aluminum, and surgical gloves for a long period and later can transmit via touch (Qu et al. 2020). SARS- CoV- 2 has also been noticed in tears, which is similar to SARS- CoV (He et al. 2020). Human coronaviruses can be contagious on non-living surfaces for 9 days at room temperature. Larger aerosol droplets may fall on objects such as desks and can be active for 3- 5 days. Fecal transmission may also be possible because COVID -19 has been detected in stool samples (Qu et al. 2020). The mean incubation time is about 5 days, from 1 to 14 days (He et al. 2020). Coronavirus can stay last on copper for 1 hour period, on cardboard for 3 hours, and can stay for up to 5.6 hours on stainless steel surfaces, and 6.8 hours on plastic, resulting in fomite transmission (Marques & Domingo 2020). People standing near an infected patient within less than 2 meters of the area can also get that infection by sneezing or being coughed by an infected person. An elevated temperature greater than 100.4°F along with other symptoms like runny nose, body aches, sore throat, etc. may occur during the infection (Fang et al. 2020). Generally, the air pollution-to-person transmission mechanism plays a major role instead of human-to-human transmission (Coccia 2021, Schiffrin et al. 2020).

Biomarkers

Red blood cells, Haemoglobin, Coagulation factors, Platelet count, Fibrinogen, C-reactive protein, Protein oxidation and Interleukin-6 measured in blood plasma and found that PM exposure was interconnected with increased C-reactive protein (CRP), plasma viscosity, and blood coagulation. Blood parameters change with plaque misbalance, which leads to coronary disease in patients (Ruckerl et al. 2006).

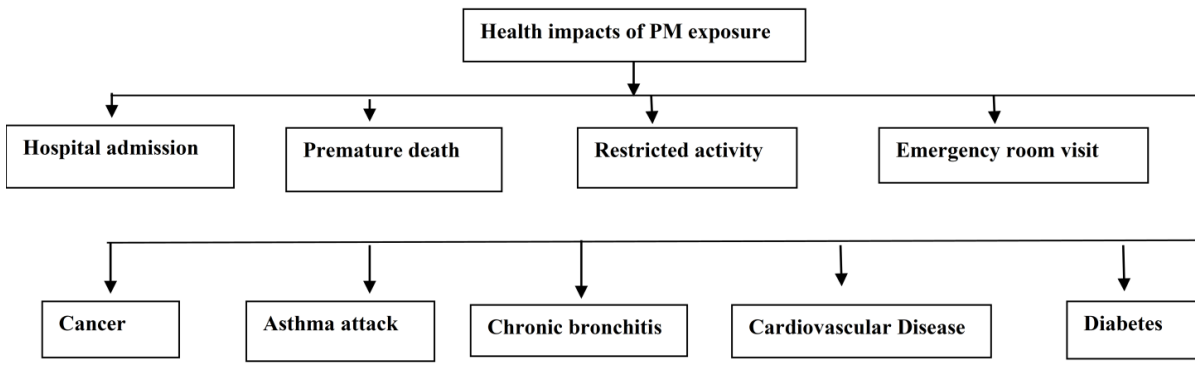

Fig. 2. Systematic framework for morbidity and mortality due to PM exposure.

Fibrinogen, a glycoprotein complex is produced in the liver. It is converted enzymatically by thrombin to fibrin and then to a fibrin-based blood clot. The amount of fibrinogen is responsible for clot formation (Bonzini et al. 2010). The Particulate effect increases with less diameter because of easier vascular penetration, high pulmonary deposition efficiency. These effects could develop within 2 hours after PM exposure and become powerful within the first 12 or 24 hours (Chen et al. 2015). High exposure to PM may increase the amount of C- reactive protein (CRP), coagulation of blood, plasma viscosity, fibrinogen, and platelet count. However, biomarkers such as inflammation marker (interleukin 6), haemostasis (platelet counts and coagulation factors) are used in clarifying the mechanism of action. Production of IL-6 stimulates by exposure to particulate matter causes the formation of thrombosis (Ruckerl et al. 2006, Zhu et al. 2020).

The Disarray of Fibrinolytic and Coagulation System in Covid-19 Patients

One of the important discoveries replicated over most preliminary COVID-19 studies includes disarray of the fibrinolytic and coagulation system. Hospitalized patients affected from severe and moderate COVID-19 infection are prominently showing elevated D-dimer, prolonged prothrombin time (PT), and activated partial thromboplastin time (APTT) (Han et al. 2020). It is found through pulmonary inflammation stimulating macrophages activation and release of Interleukin 6 (IL-6). Inhalation of PM enters the human lung and deposited on alveoli, leads to an inflammatory reaction (Fig. 3) that causes an elevation in circulating inflammatory biomarkers like C-reactive protein and fibrinogen but favours formation of thrombus which in turn triggers acute events. Fibrinogen in high amounts acts as a marker for acute inflammation, while they show activation of

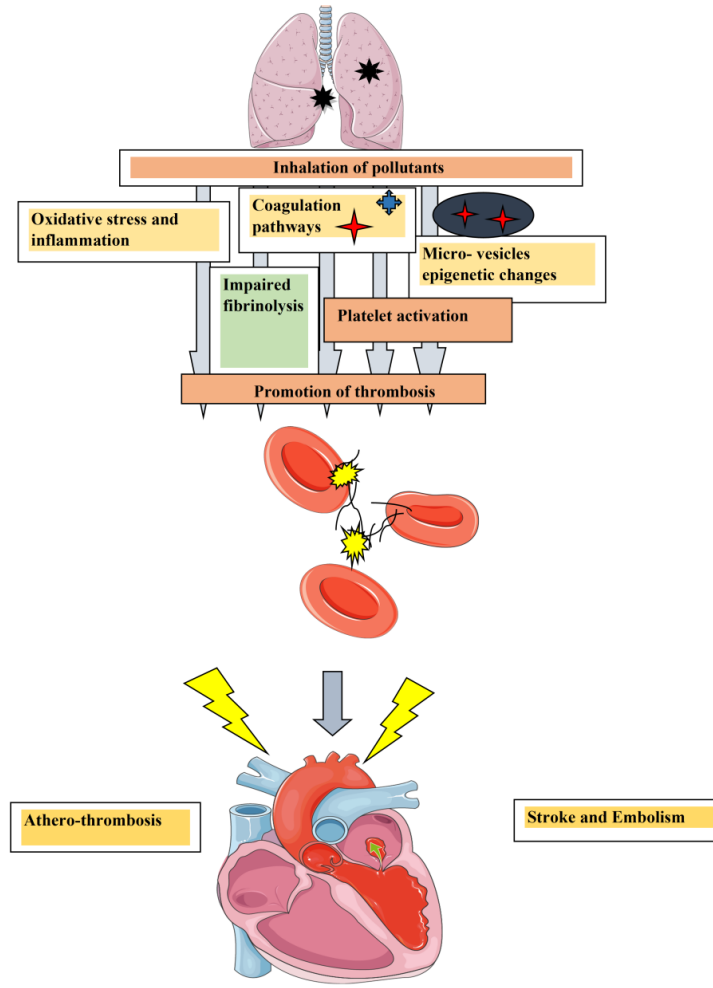

Fig. 3. Inhalation mechanisms and thrombosis formation.

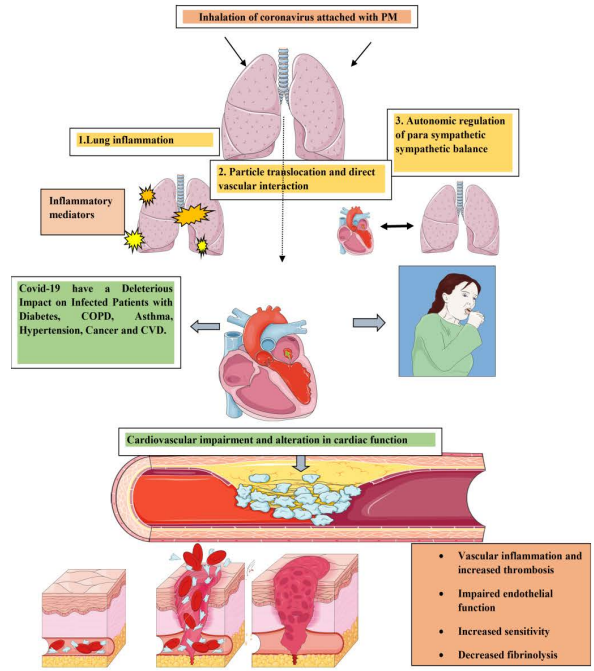

Fig. 4. Mechanism for cardiovascular impairment due to PM attached coronavirus inhalation.

clotting cascade in moderate amounts (Bonzini et al. 2010).

It is evidenced that air pollution exposure promotes coagulation and damages fibrinolysis, leads to an imbalance in haemostatic factors, and then can cause risk of thrombotic in individuals (Fig. 4).

COVID-19: Deleterious Impact on Infected Patients

Prominent changes in blood coagulation of patients with COVID-19 infection: The haemostasis function differences between SARS-CoV-2 patients and healthy controls composed the evaluation of nine parameters, as prior indicated. In comparison with healthy controls, values of antithrombin (AT) were accounted lower in COVID-19 cases. Thrombin time was found to be lower than that in healthy controls. The Prothrombin time (PT) was accounted low in COVID-19 patients as compared with healthy controls although the FDP and D-dimer values were found to be higher in COVID-19 patients than healthy controls. Remarkably, in SARS-CoV-2 cases, FIB values were also found to be higher as compared with the healthy control group (Han et al. 2020). The coagulation function of SARS-CoV-2 infected patients is undoubtedly unbalanced as compared with healthy individuals but controlling FDP and D-dimer values might be useful for the early recognition of serious cases (Han et al. 2020).

Diabetes and COVID-19: Diabetic patients are prone to get infections because of the potential of impaired phagocytic cells, however, some other factors elevate the COVID-19 risk in diabetic susceptible individuals (Ejaz et al. 2020). The spike (S) protein of SARS-CoV-2 connects to the ACE-2 receptors and is turned on by huge furin levels. Consequently, an unbalanced immune response with furin expression and increased ACE-2 receptors may result in lower insulin levels and an increase in lung inflammation rate (Ejaz et al. 2020). Diabetes-induced hyperglycemia resulting in several issues, such as chronic inflammation and weaken immune system through oxidative stress and decreased functioning of macrophages, antibodies, and chemokines. It also causes diabetes related problems like endothelial dysfunction and coagulation (Bradley et al. 2022).

COPD and COVID-19: The COVID-19 disease resulted in hypoxemia development in 15–20% of the cases, which needed the support of a ventilator in critical situations (Qu et al. 2020). However, primary studies did not report COPD with increased COVID-19 cases, the ACE-2 receptors expression is elevated in this disease, which leads to the development of serious symptoms in COVID-19 cases, such as weak immunity, structural damage to lungs, and mucous production (Newman et al. 2020).

Asthma and COVID-19: Air quality has an impact on

symptoms of asthma and on activating asthma attacks as explained in figure 2. Still, COVID-19 can be a serious disease for already damaged lungs from chronic asthma, especially in asthma–chronic obstructive pulmonary disease (COPD) overlaps syndrome (ACOS) patients (Yin et al. 2018). Although asthma is not directly correlated with COVID-19 disease, people with respiratory diseases and other complications are more prone to being infected during asthma (Ejaz et al. 2020). $PM_{2.5}$ can lead to asthma attacks and risk of chronic obstructive pulmonary disease (Nishida & Yatera 2022).

Hypertension and COVID-19: COVID-19 infection is correlated with uncontrolled blood pressure along with an increased case fatality rate (CFR) (Fang et al. 2020, Schiffrin et al. 2020). Pneumonia with increased blood pressure was noted in hypertensive patients with the covid-19 disease while blood pressure controlling should be important among COVID-19 cases to reduce the burden of disease (Ejaz et al. 2020).

CVD and COVID-19: CVD had a strong correlation with MERS (30%) and SARS (8%) (Nishiga et al. 2020). However, the increased prevalence of COVID-19 cases was mostly observed among patients with serious signs and symptoms of CVD (Zhou et al. 2020). Among pre-existing CVD cases having a high risk of COVID-19 disease can be due to the presence of ACE-2 receptors on cardiac muscle cells, implying the cardiovascular system in the SARS-CoV-2 infection (Ejaz et al.2020).

Cancer and COVID-19: According to a Chinese cohort, cancer patients are more prone to COVID-19 infection. Cancer patients are especially vulnerable towards their immunocompromised state (Han et al. 2022). Cancer patients found to be the highly susceptible group in this COVID-19 outbreak as studied cancer shows poor outcomes and worsen conditions such as dry cough (81%), fever (82.1%), and dyspnoea (50.0%), along with increased C-reactive protein (82.1%), lymphopaenia (82.1%), hypoproteinaemia (89.3%) and anaemia (75.0%) (Yu et al. 2020).

Impact of COVID-19 on children: Additionally, studies found that children younger than 10 years old seem to get contaminated from COVID as adults, but they don't show any serious symptoms. A logical theory for why most of the children show milder symptoms is that they have fewer ACE2 receptor proteins that act as an entry receptor for the SARS-CoV-2 virus in cells (Mallapaty 2020). Another study concluded that the association of higher mortality due to COVID-19 to PM2.5 in the region of northern Italy imparts a sharp piece of evidence that individual living in a region over high $PM_{2.5}$ level are more prone to promoting respiratory diseases, mostly in children (Senthilumaran et al. 2020).

Mortality due to air pollution and COVID-19: Ambient PM exposure and household air may be dangerously polluted, resulting in seven million avoidable deaths every year. Moreover, there is more evidence for $PM_{2.5}$ which shows negative effects on the cardiovascular system of heart failure, diabetes, hypertension, and cardiac arrhythmias through mechanisms of thrombosis (Fig. 4), and inflammation (Isaifan 2020).

CONCLUSION

Despite the strong hypothesis and evidence, people seem to be locked in the old perspectives that only direct links can cause the spread of viral infection. It will be too late when this pandemic is over, and backward data will reveal the importance of airborne-based transmission. Furthermore, the lessons we learned now will make us prepare better for the next viral pandemic, then we will strongly consider the transmission of this virus through air. Therefore, reduction of air pollution mainly $PM_{2.5}$ emission is essential to minimize the COVID-19 infection.

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