

# Environmental Racism and the COVID-19 Pandemic

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## ABSTRACT

Exposure to air pollutants like ground-level ozone and particulate matter is closely associated with rates of COVID-19 mortality and transmission. Potential biological mechanisms connecting pollution with COVID-19 severity include overexpression of the ACE2 receptor, pollutants' role as a carrier or transport vector for COVID-19, destruction of the cilia, and compromised immune response of the lungs. Thus, individuals with more long-term exposure to air pollution are much more susceptible to severe symptoms from COVID-19. Social disparities dramatically affect which populations are regularly exposed to air pollutants and therefore incur disproportionate rates of respiratory diseases. Environmental racism and racial segregation have been shown to be significantly and positively associated with exposure to air pollution from sources like car engines and power plants. This results in individuals of low socioeconomic status, people of color, and those living in low-income areas being often exposed to increased levels of air pollution, making them more likely to suffer greater tragedies from COVID-19 and respiratory diseases. The results of this study provide insight into the public policy work necessary to ameliorate health inequities resulting from environmental racism and to prevent worse outcomes from future pandemics.

## Introduction

In December 2019, a new coronavirus—the severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2), which causes the Coronavirus Disease 2019 (COVID-19)—has claimed more than 743,000 American lives as of October 29, 2021 (The New York Times 2021). SARS-CoV-2 transmission in humans occurs when viral particles in the respiratory droplets of an infected individual reach the airways and enter the cells through receptors, such as Acetyl Cholinesterase Receptor (ACE2R). Inside the cell, the viral genome takes over the cell's replication machinery and produces exponential numbers of viral particles, resulting in the destruction of lung epithelial cells, leading to pneumonia and acute respiratory distress. This can result in lung complications such as pneumonia and, in the most severe cases, acute respiratory distress syndrome (Galiatsatos 2020).

When considering respiratory health, individuals of low socioeconomic status, people of color, and those living in low-income areas are often exposed to increased levels of air pollution. Because constant exposure to airborne pollution worsens respiratory disease, individuals in these populations may be particularly vulnerable to severe illnesses such as COVID-19.

This review analyzes environmental conditions as a function of COVID-19 mortality and transmission. First, two widespread air pollutants, ozone and particulate matter, their effect on lung function, and potential biological mechanisms connecting COVID-19 severity and pollution will be discussed. Because health disparities are driven by underlying socioeconomic inequalities, the intersection between systemic inequity, long-term exposure to air pollution, and COVID-19 will then be addressed.

In order to better understand the relationship between air pollution and lung diseases such as COVID-19, the different types of air pollutants and their effect on human lungs will be described in detail.

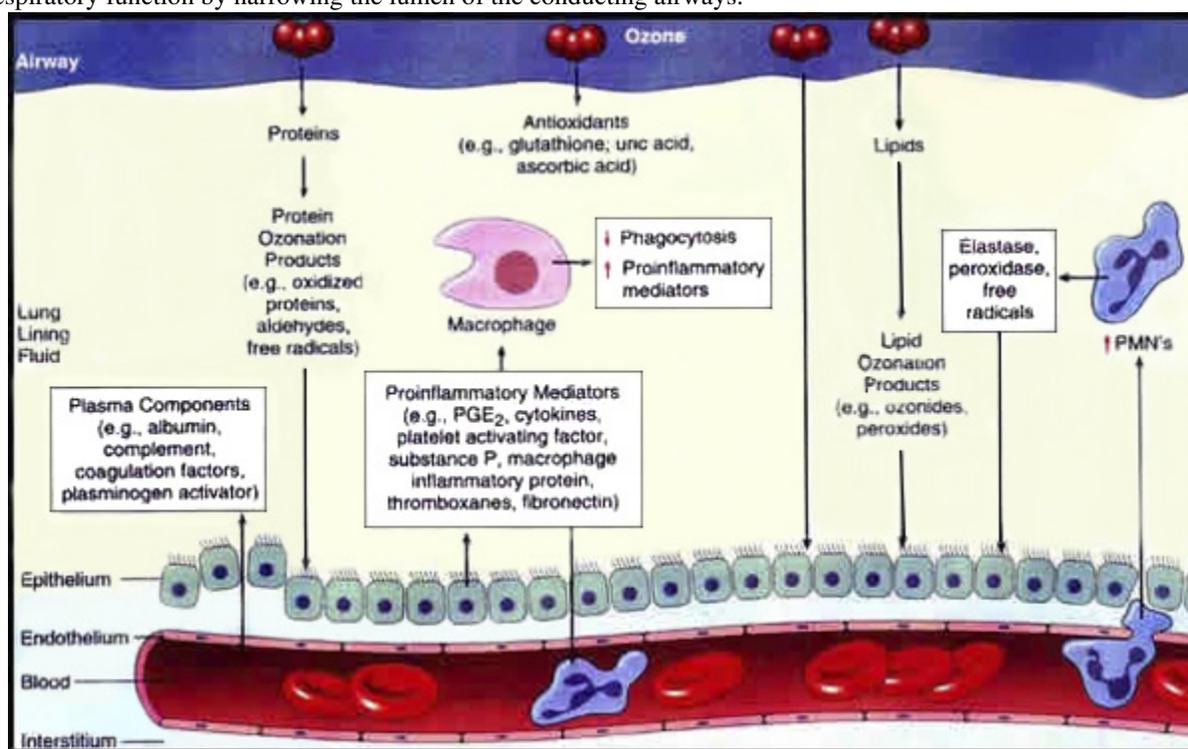
## Major Players in Air Pollution

Among the various types of outdoor air pollutants, ground-level ozone and particulate matter emerge as the most common and deadly, increasing threats to public health in communities around the country.

### Ground-Level Ozone

Ozone (O<sub>3</sub>) is a gas composed of three oxygen atoms, often referred to as “smog.” Ozone is frequently emitted from power plants, motor vehicles, and other sources of high-heat combustion. When found in the troposphere—the lowest region of the Earth’s atmosphere which contains the air we breathe—ozone causes serious health problems.

When inhaled, ozone reacts with the lung tissue and attacks it. Ozone’s limited solubility in water prevents the upper respiratory tract from dissolving ozone into the mucus lining, where pollutants are typically expelled through coughing. This allows ozone to reach the lower respiratory tract and dissolve in the thin layer of epithelial lining fluid (ELF). Impacted cells are injured and leak intracellular enzymes into the airway lumen. Upon injury, epithelial cells also release various pro-inflammatory mediators like cytokines and PGE<sub>2</sub>, which can attract immune cells into the lung and exacerbate lung inflammation (Leikauf *et al.* 2020). Ultimately, chronic inflammation in the lung reduces respiratory function by narrowing the lumen of the conducting airways.



**Figure 1.** The reaction of ozone in the respiratory tract (U.S. Environmental Protection Agency 2021).

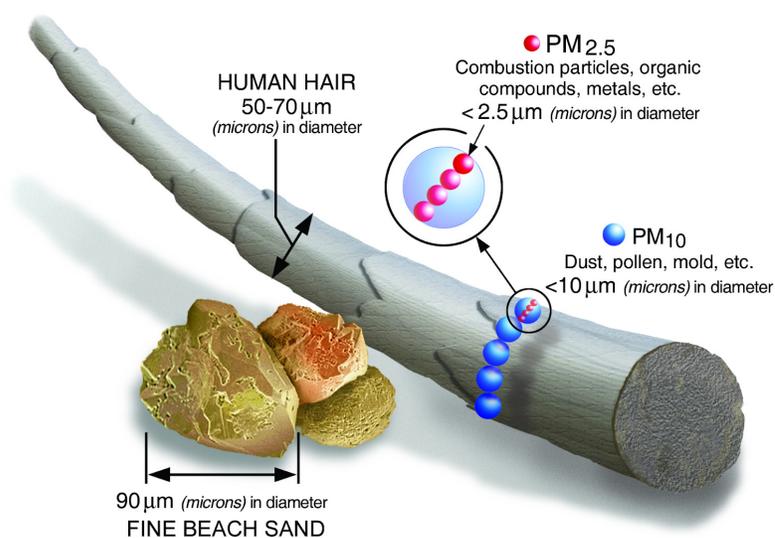
Exposure to ozone can result in shortness of breath, wheezing, coughing, asthma attacks, increased risk of respiratory infections, chronic lung diseases like obstructive pulmonary disease (COPD), cardiovascular disease, and premature death (American Lung Association 2020).



**Figure 2.** Effects of ozone on lung function: A healthy lung airway on the left compared to an inflamed lung airway on the right (U.S. Environmental Protection Agency 2021).

### Particulate Matter

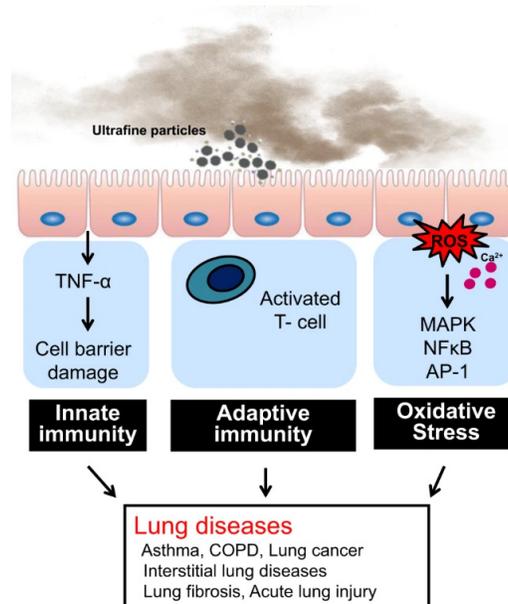
Particulate matter (PM), also known as particle pollution, refers to a mixture of solid particles and liquid droplets found in the air. Most particles form in the atmosphere as a result of complex reactions of chemicals such as sulfur dioxide and nitrogen oxides. Particles can also be produced from dust and debris in roads, construction sites, industrial facilities, wildfires, and automobiles. Consisting of hundreds of different chemicals, airborne particles are classified by size. Two particle sizes, particles of 10 microns (PM 10) and 2.5 microns (PM2.5), are common forms of pollutants that have known consequences on respiratory health (New Hampshire Environmental Public Health Tracking 2018).



**Figure 3.** Size comparisons for PM particles with a strand of human hair and fine beach sand (U.S. Environmental Protection Agency 2021).

According to both animal and controlled human exposure studies, sufficient amounts of particles deposited in the respiratory tract can induce inflammation. At the cellular level, inflammation may damage or kill cells and compromise the integrity of the alveolar-capillary barrier. Because oxygen exchange happens in the alveolar-capillary barrier, without it, O<sub>2</sub> is unable to diffuse readily into the bloodstream and waste products cannot leave it. Repeated exposure to particle pollution aggravates initial injury and promotes chronic inflammation (Berend 2016). Airway inflammation generally increases airway responsiveness to irritants such as cold air, allergens, and pollutants. This increased responsiveness in the respiratory tract causes tightening of the muscles surrounding the airways, which reduces airway size and causes less oxygen to be delivered to the lungs. Therefore, exposure to particulate matter can

increase the occurrence of symptomatic flare-ups in diseases such as asthma and COPD, diseases where airway size is already reduced. Prolonged exposure to particles has also been linked to premature death (California Air Resources Board 2021).



**Figure 4.** Inflammatory Pathways Activated in the Response to Particulate Matter in the Respiratory Tract (U.S. Environmental Protection Agency 2021).

## Potential Biological Mechanisms Connecting COVID-19 Severity and Pollution

Severe lung inflammation caused by ozone and particulate matter may increase the mortality rate and prevalence of COVID-19 in the most polluted areas. ACE2, the receptor SARS-CoV-2 binds to in order to enter the cell, generates anti-inflammatory peptides, which help to mitigate the effects of PM<sub>2.5</sub> exposure. Because of its beneficial role in inflammation response, ACE2 is overexpressed in individuals exposed to pollution. This increase in ACE2 receptors could potentially increase the probability of SARS-CoV-2 entering the cells via ACE2 receptor binding (Comunian *et al.* 2020). Additionally, when SARS-CoV-2 is bound to the ACE2 receptor, the enzyme would no longer be able to aid in anti-inflammatory functions, worsening airway damage.

Like other viruses such as avian influenza virus (H5N1) and measles, COVID-19 is transmitted by airborne droplets. Recent data suggests particulate matter may function as a carrier or as a transport vector for these viruses, allowing viruses to get deeper into the lungs when attached to particulate matter. An analysis of the spread of measles in China, studying PM<sub>2.5</sub> concentrations in 21 Chinese cities and the number of measles cases per day per city, showed that a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> per day was associated with a significant rise in the disease incidence (Chen *et al.* 2017). A similar positive correlation between particulate matter and virus spread is likely to happen in COVID-19 (Comunian *et al.* 2020; Travaglio *et al.* 2020). Therefore, because COVID-19 is spread through direct microbial pathogenic transmission through the air and can attach to pollutant particles, there would be increased rates of COVID-19 transmission in highly polluted areas.

Once viral particles have gained access to the respiratory tract, the effect of pollution on the health of the respiratory cells themselves may also increase the severity of viral infection. Specifically, there are connections between the increased severity of COVID-19 cases and exposure to air pollution through the damage done to cilia, small projections that move dirt and pollutants up and out of the respiratory tract. Pollution and smoking can destroy cilia,

prompting germs, dust, and mucus to collect in the pockets of airways and get stuck. These effects of pollution lead to an increased prevalence of lung diseases such as asthma, COPD, and lung fibrosis (Prunicki 2020).

Air pollutants often cause changes in the transcriptional machinery of the cells by altering the epigenetic milieu of the cells, thereby compromising the immune response to viral onslaught. Studies have shown that exposure to polluting agents alters the immune response of the lung cells and induces an increase in oxidative and inflammatory stress (Comunian *et al.* 2020; Prunicki 2020). Long-term exposure to air pollutants leads to an increased prevalence of COVID-19 cases and increases the risk of severe COVID-19 symptoms by damaging respiratory organs and the immune system.

## The Effects of Environmental Racism on COVID-19

Social disparities dramatically affect which populations are regularly exposed to air pollutants and therefore incur disproportionate rates of respiratory disease. Environmental racism, defined as “systems that produce and perpetuate inequalities in exposure to environmental pollutants” (Washington 2020) is closely related to racial segregation. This means that people of color are often concentrated in neighborhoods that have frequently been disempowered, both politically and financially. For these reasons, neighborhoods with large non-white populations have historically seen lower property values, meaning that land in those areas is cheaper for industrial actors to acquire—leading to greater pollution (Perry *et al.* 2018). Similarly, the harms of emissions from cars and trucks have been concentrated in communities of color through the siting of freeways and shipping centers.

In the United States, black communities in particular face greater effects of environmental pollution. According to the “State of Black America,” the COVID-19 infection rate for black Americans is 62 per 10,000, in comparison to 23 per 10,000 for whites, making black communities one of the hardest hit by the pandemic (Soucheray 2020). When considered alongside the increased exposure to pollution faced by communities negatively impacted by social disparities, the relationship between COVID-19 outcomes and environmental racism comes to light. Racial segregation has been shown to be “significantly and positively associated” (Bravo *et al.* 2016) with exposure to air pollution from sources like car engines and power plants.

According to a statistical analysis using data collected by the U.S. Census Bureau in November 2020, high tropospheric ozone concentrations contribute to a greater COVID-19 mortality rate (Liu and Li 2020). Another study conducted by the U.S. Environmental Protection Agency to quantify nationwide disparities in the residential populations surrounding particulate matter-emitting facilities found that those in poverty had a 1.35 times increase in exposure burden to PM<sub>2.5</sub> than the overall population did. Non-whites had a 1.28 times higher exposure burden and blacks, specifically, had a 1.54 times higher burden of exposure than the overall population did (Mikati *et al.* 2018). Further studies also show black populations experience exposure to greater than average concentrations of PM<sub>2.5</sub> nationwide (Tessum *et al.* 2021).

Studies performed in 2020 have also uncovered a correlation between air pollution and COVID-19 outcomes: increases in exposure to hazardous pollutants are associated with a 9% increase in death among patients with COVID-19 in one study (Petroni *et al.* 2020), and exposure to both ozone and particulate matter was positively correlated with mortality in another (Liang *et al.* 2020). These data emphasize the association between exposure to ozone and particulate matter and mortality in COVID-19 infection. Taken together with the increased exposure of marginalized populations to these air pollutants, social disparities and systemic environmental racism play a considerable role in the tragic effect of COVID-19 on these populations.

## Conclusion

Policy choices have been a major driving factor of these dangerous gases towards communities of color and away from wealthier, whiter neighborhoods, caused by imbalances in political power. Air pollution exposure is a well-

documented risk factor for respiratory disease, particularly worse outcomes in COVID-19 infections. As described in this review, social disparities in the United States result in increased exposure to air pollution in these populations. While there are longstanding policy proposals that would have helped the health and well-being of Americans before the pandemic, there is little policy in place to weather the crisis in the United States.

Political action such as environmental justice proposals like the Environmental Justice for All Act (116th Congress 2020) would reduce overall emissions and help close racial, social, and health disparities if put into place. Furthermore, fully funding policies and proposals to integrate neighborhoods such as the Housing Choice Voucher Program (Center on Budget and Policy Priorities 2009) would allow individuals to move to areas of better air quality. Implementing these policies would boost public health overall in addition to alleviating several key risk factors for COVID-19.

Implementing policies aimed at improving air quality in areas where social disparities exist would ameliorate the effect of COVID-19 on communities of color and of low socioeconomic status, and work to lessen the health disparities during COVID-19 and into the future.

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