Coronavirus, as an Air Pollutant

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The more recent cases of COVID-19 seem to be coming from homes and family contacts, rather than from strangers. And there, with the "opening up" of social mobility, is an increasing interest in the spread and dispersion of airborne COVID-19 particles. There are lessons to be learned from atmospheric science, especially when it pertains to the dispersion of small particles.

Indoor infectivity has been discussed in The Atlantic [2], Wired [3], and now the Wall Street Journal [4]. Rather than merely joining the chorus, we consider the dispersion of all types of airborne particles (including COVID-19), some of which have been regarded as significant health risks.

Many types of sources emit air pollutant particles; they have been grouped by particle size and regulated by the US EPA under the rubric “PM$_{2.5}$” or “fine” particles. These particles are submicroscopic and much smaller than a human hair. Their properties have been studied and discussed for decades, including those from indoor sources. What can this model tell us about COVID-19’s ability to travel, disperse, and pose the risk of lethal respiratory infections?

How are we exposed to airborne pollution? A simple model of dispersion and exposure

Air pollutants come from a variety of human-made and natural sources, some stationary such as
smokestacks and others mobile such as vehicular traffic. Most of those stationary sources now have effective pollution mitigation strategies, including cleaner fuels and particle filters. Exposure to these pollutants is inferred from location, such as where you live. Air pollution from tall smokestacks tends to be dispersed over broad, even multistate, areas, but we are directly exposed to emissions from ground-level sources, including traffic and home heating. As a result, living in an area of high traffic and population density results in more exposure to air pollution; however, individual air pollution exposures to from such aggregate measures will vary. Exposures also change over time with weather conditions.

Sources

Under the Clean Air Act, major outdoor air pollution sources are monitored and controlled to meet national standards for the outdoor (ambient) air we breathe. Vehicle emissions are controlled by design with devices installed at the factory and occasional regulatory inspections. Residential (domestic) air pollution emissions from home heating, barbeques, and gas-powered lawn, leaf, or snow blowers are virtually uncontrolled. Mathematical models are then used to assess the relative contributions of these types of sources to ambient air quality. Vehicular and domestic emissions are pooled as “area” sources without the identification of individuals. Ambient air pollutant particles, which vary significantly in terms of sources, size, and composition, have been grouped only by volume so that that cement dust may be bundled with much smaller and potentially carcinogenic soot particles.

Indoor air pollution sources have been identified but are not covered by the Clean Air Act. Studies have shown that, on average, about 50% of outdoor air pollution penetrates indoors depending on building design, use of air conditioning, etc. The most critical indoor sources are cigarette smoke, cooking, cleaning products, pets.

According to our COVID-19 analogy at the local scale, individuals who have had more contact with infected individuals will have had greater exposures and risks of infection. Large assemblies that include infected individuals comprise viral area sources.

Dispersion

Just as a colored dye disperses when poured into the sea, air pollution disperses after emission to the atmosphere, depending on the current (winds), wave action (turbulence), and buoyancy (relative temperature). Sand will settle to the bottom as do the larger and heavier airborne air pollution particles. Pollutants emitted within a closed room will disperse less rapidly as will dye in a quiescent bathtub. Here the diminishing dye color contrast resulting from dilution is analogous to reductions in pollutant concentrations and the accompanying inhaled doses. These principles apply to viral and pollution particles alike.

There are important distinctions between outdoor and indoor environments. Outdoors, particles stream downwind after emission, air pollutants from tailpipes or smokestacks and viral particles from the nose or mouth. Concentrations diminish as the plumes expand under the influence of wind variations. The scales for stack vs. oral emissions are vastly different, from km to cm, but the dispersion processes are fundamentally the same. However, the sources and receptors for air pollution tend to remain fixed, but viral sources, from infected individuals, are inherently mobile,
the worst cases being lengthy conversations in groups at close range.

Interactions between individuals are also important indoors; atmospheric conditions, less so. The major difference is that the indoor environment is basically a closed system with respect to virus transmission. Exhaled viral particles will remain until they are deposited and adsorbed onto surfaces or clothing. This is also the case with smoke and the smallest combustion particles. Air concentrations will drop as fresh air is introduced but the total particle counts will remain. Increased turbulence within a room, for example through ventilation, will provide some relief by increasing rates of particle deposition onto surfaces where they may contribute to tactile virus transmission.

These concepts illustrate the importance of the face mask to provide a filter between source and receptor. Instead of a downwind plume, exhaled breath will be filtered as it squeezes through the pores of the mask material or escape peripherally with its viral particles. In any event, the now diminished viral load remains in close proximity to its source, perhaps as a sort of invisible halo. Such halos will be dissipated in response to the wind outdoors, but indoors they still contribute to the overall viral burden. Social isolation is one response to such indoor conditions.

**Health effect processes**

Air pollution sources and patterns of dispersion are well known. While there are several biologically plausible ways these pollutants can affect longevity, none have been validated experimentally. Instead, statistically derived dose-exposure relationships are assumed to be causal. By contrast, viral particles including influenza and COVID-19 have long-standing, accepted causal pathways, but their patterns of transmission have not been demonstrated. While air pollution sources are diverse, indoors and out, viral particles originate from previously infected individuals.

Direct transmission of COVID-19 by contact with surfaces, packages or counters, seems to play a smaller role, despite earlier concerns. Airborne respiratory transmission now appears to be the predominant means of transmission. While individual and population susceptibility and infectivity for COVID-19 are still being researched, it seems clear that a minimum viral load is required that will likely vary among individuals.

**Conclusions**

Viral particles may resemble the tiniest air pollution particles released from combustion processes that have received far less research attention. Their atmospheric behavior is the key to understanding COVID-19 exposures and their health risks. It would be both impractical and immoral to conduct dispersion and exposure experiments on viral particles; viral air samples have been collected in hospital settings but not in residential or outdoor environments. The atmospheric science community should therefore assign a high priority to developing an experimental model for viral particles to improve our understanding of viral aerosol exposures.

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