Brief Report

The Air and Viruses We Breathe: Assessing the Effect the PM$_{2.5}$ Air Pollutant Has on the Burden of COVID-19

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Abstract: Evidence suggests an association between air pollutant exposure and worse outcomes for respiratory viral diseases, like COVID-19. However, does breathing polluted air over many years affect the susceptibility to SARS-CoV-2 infection or severity of COVID-19 disease, and how intense are these effects? As climate change intensifies, air pollutant levels may rise, which might further affect the burden of respiratory viral diseases. We assessed the effect of increasing exposure to PM$_{2.5}$ (particulate matter $\leq$ 2.5 microns in diameter) on SARS-CoV-2 susceptibility or COVID-19 severity and projected the impact on infections and hospitalisations over two years. Simulations in a hypothetical, representative population show that if exposure affects severity, then hospital admissions are projected to increase by 5–10% for a one-unit exposure increase. However, if exposure affects susceptibility, then infections would increase with the potential for onward transmission and hospital admissions could increase by over 60%. Implications of this study highlight the importance of considering this potential additional health and health system burden as part of strategic planning to mitigate and respond to changing air pollution levels. It is also important to better understand at which point PM$_{2.5}$ exposure affects SARS-CoV-2 infection through to COVID-19 disease progression, to enable improved protection and better support of those most vulnerable.

Keywords: SARS-CoV-2; COVID-19; air pollutant exposure; PM$_{2.5}$; climate change; modelling; respiratory viral disease

1. Introduction

Air pollution is the world's leading environmental cause of illness and premature death [1]. The effect of exposure to air pollutants has been associated with a broad range of health effects including respiratory and cardiovascular diseases, high blood pressure, lung cancer, type 2 diabetes, and neonatal disorders, together resulting in seven million premature deaths each year [2,3]. The cost of the damage to health related to air pollution is estimated at $8.1 trillion a year, amounting to 6.1% of global GDP [1]. Of the most common air pollutants, including PM$_{2.5}$, PM$_{10}$, ozone, nitrogen dioxide, carbon monoxide, and sulphur dioxide, PM$_{2.5}$ is associated with the greatest proportion of adverse health effects including premature death, particularly in those who have chronic cardiovascular or respiratory disease, and reduced lung function in children. PM$_{2.5}$ is fine inhalable particles with a diameter of 2.5 micrometres or less, which can enter the lungs and permeate the bloodstream. Long-term (months to years) exposure to PM$_{2.5}$ may stimulate the immune system, lead to increasing inflammatory response, and cause inflammatory disease, such as asthma, even in those who are young and healthy [4,5]. Chronic respiratory conditions,

like asthma, make sufferers more prone to infection by respiratory viral agents like SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2), the causative agent of COVID-19 (coronavirus disease 2019). This occurs as exposure to air pollutants may facilitate evasion of antiviral responses, enable viral entry into cells, promote viral replication, and enhance inflammation during viral infection [6]. This makes people more susceptible to oxidative damage, and can potentially worsen COVID-19 symptoms and could ultimately lead to death.

While results vary and are even contradictory, it has been estimated that particulate air pollution contributed to approximately 15% [95% CI 7–33%] [7] of the over 6.89 million COVID-19 deaths worldwide [8]. Moreover, as climate change worsens, it is expected that particulate matter concentrations could more than double in certain settings [9]. If this happens, higher pollution levels could have a greater impact on the burden of COVID-19. However, it remains unclear whether chronic exposure to polluted air directly affects severity of the COVID-19 disease or affects susceptibility to SARS-CoV-2 infection. There is also uncertainty around the degree of these associations.

Studies have shown that areas with higher levels of air pollutants also had higher spread of SARS-CoV-2. For instance, in Italy, a country that ranked seventh globally for COVID burden at the end of March 2021, an individual-level study was conducted in the northern city of Varese from the end of February 2020 to mid-March 2021 [10]. In this study, exposure to low $PM_{2.5}$ levels (annual average of 12.5 μg/m^3) was reported to have been associated with a 5.1% [95% CI 2.7–7.5%] increase in COVID-19 cases. From mid-February to mid-April of 2020 an ecological study was carried out in another Italian city, Lombardy, the most populated and industrialized region of the country and the country's epicentre for the first COVID-19 wave from March to April 2020. In this second study, a 10 μg/m^3 average annual increase in $PM_{2.5}$ was associated with a 58% increase in the incidence of COVID-19 [11]. Similarly, in another ecological study of sites across England, a small annual increase of one μg/m^3 over one year or five years of exposure was associated with a 12.7% [95% CI 8.3–17.3%] increase in SARS-CoV-2 cases and declared a major contributor to increased incidence [12].

Studies have also shown a relationship between air pollutant exposure and COVID-19 hospitalisations. For example, a study using antibody detection showed a 17% [95% CI 3–32%] increased risk of developing COVID-19 from June and November 2020 in a cohort of 9605 adults from Catalonia, Spain who were exposed to $PM_{2.5}$ pollutants from 2018 to 2019, even if they did not find significant association with SARS-CoV-2 infections [13]. A time-series modelling analysis further supported these findings, showing that exposure was estimated to account for an approximate 24% increase in COVID-19 hospitalisations in Kuwait [14].

Air pollutant exposure has also been associated with an increase in deaths. An examination of ecological studies spanning 3089 counties covering 98% of the US population reported an average annual increase of 1 μg/m^3 in $PM_{2.5}$ from 2000 to 2016 with an 11% [95% CI 6–17%] increase in the national COVID-19 mortality rate as of mid-June 2020 [15]. Another ecological modelling study from Wuhan City in China, the city where SARS-CoV-2 first emerged, revealed that those exposed to higher levels of $PM_{2.5}$ were more susceptible to COVID-19 mortality as of April 2020 [16]. This is not a new phenomenon: reports of the association between air pollution and coronavirus mortality date back to the 2003 SARS outbreak. An ecological study conducted from April to May 2003 in five regions in China with elevated Air Pollution Index (API) indicated that there was a linear relationship between API and SARS fatality [17]. The higher the API, the higher the mortality rate.

Here we assess, through simulations, the effect incremental increases in historical particulate matter, $PM_{2.5}$, exposure on SARS-CoV-2 susceptibility or COVID-19 severity will have on projected infections and hospitalisations.

2. Materials & Methods

2.1. Scenario Design

We explore the impact a 1 to 5 μg/m^3 increase in average annual PM$_{2.5}$ exposure has on the susceptibility to SARS-CoV-2 infection (assuming a 13% [8–17%] increase in susceptibility per exposure unit increase [12]) or on COVID-19 severity (assuming a 11% [95% CI 6–17%] increase in severity per exposure unit increase [15]). A simple schematic of the scenario design is shown in Figure 1. SARS-CoV-2 infections and COVID-19 hospitalisations (as well as ICU admissions and deaths, with results shown in the Supplementary Materials Figures S11a–S13, S16, and S17) were projected over a two-year period and compared with a no increase in exposure counterfactual (with baseline metrics shown in Figure S10 of the Supplementary Materials). Log$_{10}$ and linear increase in effect relationships between these associations were modelled as illustrated in Figure S9 of the Supplementary Materials. Simulations were conducted for a global setting capturing age and comorbidity risk, the effect of emerging variants and seasonality, and vaccination and treatment interventions.
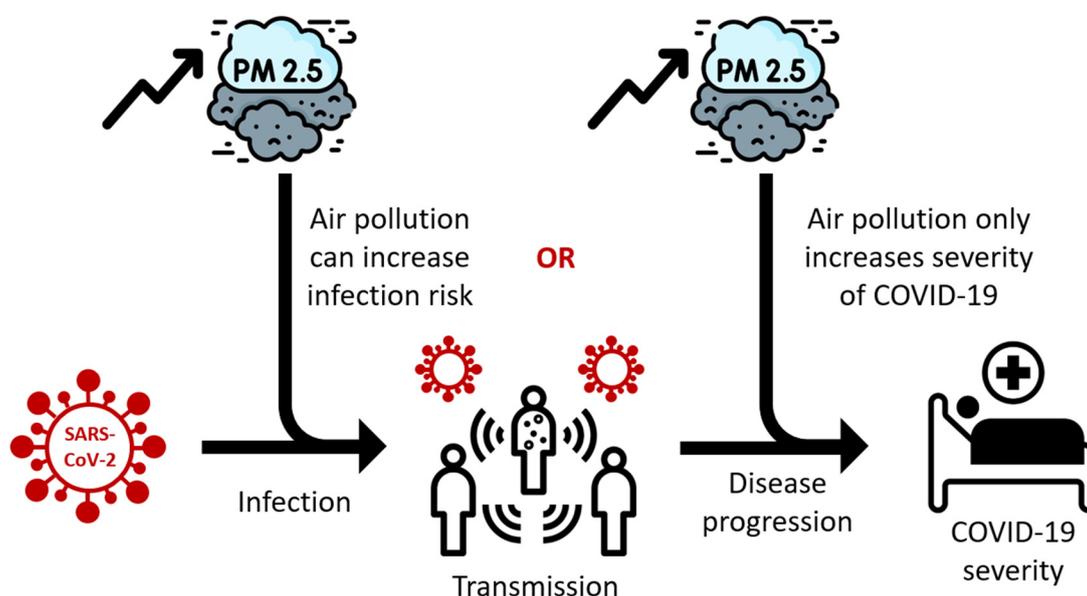

Figure 1. A schematic of how the potential impact of increasing exposure to the air pollutant PM$_{2.5}$ (particulate matter with a diameter of 2.5 micrometres or less) was modelled to either increase the risk of SARS-CoV-2 infection, with the potential for onwards transmission and disease progression or to only increase the severity of COVID-19 disease.

2.2. Model Framework

Simulations were conducted using a previously published dynamic stochastic, individual-based model of SARS-CoV-2 infection and COVID-19 disease, OpenCOVID [18]. Simplified schematic of the OpenCOVID model structure is shown in Figure S5 of the Supplementary Materials. The model simulates viral transmission between infectious and susceptible individuals that come in contact through an age-structured, small-world network (Figure S2 of the Supplementary Materials). This study was conducted in a global, archetypal setting with the probability of transmission in each exposure influenced by the infectiousness of the infected individual, the profile of emerging variants, the immunity of the susceptible individual (acquired through previous infection and/or vaccination) and a background seasonality pattern (see the Seasonality section below). Once infected, a latency period is followed by a pre-symptomatic stage, after which an individual can be asymptomatic, or experience mild or severe disease influenced by age-dependent comorbidity risk. Severe cases can lead to hospitalisation, intensive care unit admission, and ultimately death. See Figure S7 and Table S1 of the Supplementary Materials for further details on prognosis probabilities by age and Figure S6 for default durations for infection, disease, and hospi-

talisations used in the model. Recovery after infection leads to development of immunity. This immunity is assumed to wane over time as illustrated in Figure S3, with the risk of new infection depending on the probability of exposure and properties of existing and potential novel SARS-CoV-2 variants (i.e., infectiousness and immune evading profile). Further model details are provided in [18–20].

2.3. Model Parameterisation and Initialisation

We simulate a 730-day (2-year) period just ahead of mid-summer in year one to just ahead of mid-summer in year two. We apply a global demographic distribution [21] to a simulated population of 1,000,000 people and assume 98% of the population had previously been infected with SARS-CoV-2 at least once in the three years prior to the simulation period [22].

Using an average number of daily contacts, the model was fit to an initial effective reproduction number, R_e, of 0.9 at the start of the simulation period. This represents a global trend of case numbers for a dominant Omicron variant. This inherently captures the effect of any non-pharmaceutical interventions that were in place at that time, such as masking. To reflect the element of chance that naturally occurs in model transmission dynamics 800 random stochastic simulations were performed for each scenario with 95% prediction intervals presented.

2.4. Variants

Omicron (BQ.1) was assumed to be the dominant transmission variant at the start of the simulation period for this study. Variant properties were modelled as follows, (1) infectivity (a transmission multiplication factor per exposure of 14.4 (calculated as 3.2 versus the previously dominant Delta variant [23], multiplied by 1.5 for Delta versus wild-type [24]) (viral load profile is shown in Figure S1), (2) disease severity (a multiplication factor per infection of 1.54 (calculated as 0.75 versus Delta [25] multiplied by 2.1 for Delta (mean for hospitalisations [26] and deaths [27] versus wild-type), and (3) immune evasion capacity (assumed to be zero versus wild-type). In the model, once infected, variant severity influences the chance to manifest severe symptoms, rather than experiencing mild or no symptoms. One hundred percent immune evasion means fully evading any previously naturally acquired or vaccine-induced immunity, making an individual fully susceptible to infection with a new variant. The immune evasion property is assumed to only relate to the individual's previously acquired immunity (their susceptibility), thus only affecting the rate of new infections and not influencing severity once infected. A novel variant was assumed to emerge at day 90 with 10 people infected with this new variant imported into the simulation space with an assumed infectivity of 1.1 compared with the dominant variant, severity of 1.0, and an immuno-escape capacity of 0.2. On day 274, 10 people infected with a new emerging variant of concern were imported, with the same profile as the previously emerging variant applied. Full model details on the viral transmission and viral variant properties are described in Sections 1.3 and 1.4, respectively, of the Supplementary Materials.

2.5. Seasonality

Simulations represent an archetypal or global setting approximately representative of Northern hemisphere seasonality. Seasonality is assumed to follow a scaled cosine function. It oscillates between minimum seasonal forcing during the peak summer period and maximum forcing during peak winter months as illustrated in Figure S4 of the Supplementary Materials. As people tend to remain indoors during cooler winter months, the probability of transmission per contact is increased. Seasonal scaling was assumed to be 0.30 [0.26–0.34] based on a previous study in Switzerland which simultaneously fit this seasonal scaling factor along with several other parameters to align the model to context-specific epidemiological data [18].

2.6. Non-Pharmaceutical Interventions

Non-pharmaceutical interventions (NPIs) such as physical distancing, mask usage, and lockdowns were not explicitly modelled in this study. However, since it is assumed that during the simulation period a proportion of the population would have worn masks in some situations, such as on public transport in certain settings, this protective effect was indirectly captured using the number of effective network contacts to calibrate the model to the effective reproduction number at the start of the simulation period.

2.7. Vaccinations

In this analysis, we simulate the impact of mRNA COVID-19 vaccination. Vaccines have a two-fold effect; first, they provide protection against new infection through development of immunity. Second, once infected, vaccines reduce the probability of developing severe symptoms, leading to reduced hospitalisations, intensive care unit admissions, and potentially death.

Prior to the start of the simulation period, primary vaccination (doses one and two) was given at 85% coverage in the high-priority group (those 65 years and older or with comorbidities) and 60% in the low-priority group (those 5 to 64 years of age). Individuals who received primary vaccination were considered eligible to receive booster doses during the simulation. Booster doses were given at 80% coverage in the high-priority group every 182 days starting at day 183 and at 50% coverage in the low-priority group every 365 days starting at day 244. Coverage levels are based on historical global COVID vaccination trends.

Following administration of each booster dose, vaccine-induced immunity is assumed to immediately peak at 85% [28] before exponentially waning to 15% with a half-life of 105 days (based on longer-term waning for dose two from Andrews and colleagues [29] (Figure S3 of the Supplementary Materials). The infection-blocking component of the vaccine was assumed to represent 77% [30] of the overall 85% vaccine efficacy, with the remaining 5% attributed to preventing infections from progressing to severe disease. Vaccine protection is modelled in the context of naturally acquired immunity following SARS-CoV-2 infection, which is assumed to peak at 95% [31] before waning exponentially to 20% in 600 days [32].

SARS-CoV-2 infections will result in a certain proportion of COVID-19 hospital admissions based on assumed prognosis probabilities (Table S1 of the Supplementary Figure S3). The protective effect of immunity from previous infection or vaccination are modelled to only prevent 5% of hospitalisations, but will be protective against ICU admissions and COVID-19 deaths as illustrated in Figure S8 of the Supplementary Materials showing the impact of vaccine doses by variant per infections experienced by disease state (severe, critical) or death.

2.8. Treatment

Individuals are only eligible for treatment if they have been diagnosed with SARS-CoV-2 infection. There may be a delay between infection and diagnosis and between diagnosis and treatment initiation. Here we assume a total treatment delay of 5 days. Treatment can be targeted at any disease stage, those with mild symptoms not in hospital, those with severe symptoms in hospital, and those in critical condition admitted to ICU. Treatment coverage can be differentiated by priority group. Based on the reported global drug supply for a commonly used COVID-19 treatment drug regimen, we assume treatment coverage of 50% for the high-priority group [33], those aged 65 years of age or older or those living with comorbidities who are at high-risk of developing COVID-19 disease. We assume a treatment efficacy to reduce the risk of hospitalisation or death of 89% based on a commonly used COVID-19 drug regimen with efficacy reported in high-risk, ambulatory adults [34]. Treatment efficacy informs the proportion who will be successfully treated in the model. Those successfully treated return to the recovered stage and are once more susceptible to infection. We assume the immunity of a person who has been successfully treated is the

same as that for a person who naturally recovered. This may not be fully representative but was modelled this way for simplicity. Pre-exposure prophylaxis (PrEP) with monoclonal antibodies to prevent SARS-CoV-2 infection was not considered in this study; however, PrEP can be modelled in OpenCOVID.

3. Results

We used a model of SARS-CoV-2 transmission and COVID-19 disease progression that considers age, risk, emergence of new variants, seasonality, and the effect of COVID-19 vaccination and treatment to project the effect of increases in $PM_{2.5}$ exposure on either SARS-CoV-2 susceptibility or directly on COVID-19 disease severity. Assuming an 11% [95% CI 6–17%] increase per $PM_{2.5}$ exposure unit increase [15] over a two-year period, we found that if exposure affects severity, then as expected new infections would not be affected. COVID-19 hospital admissions, however, could increase by a small amount, 5% for a one-unit increase in exposure with a $\log_{10}$ association and 10% with a linear association compared with a no exposure increase baseline (Figures 2–4).
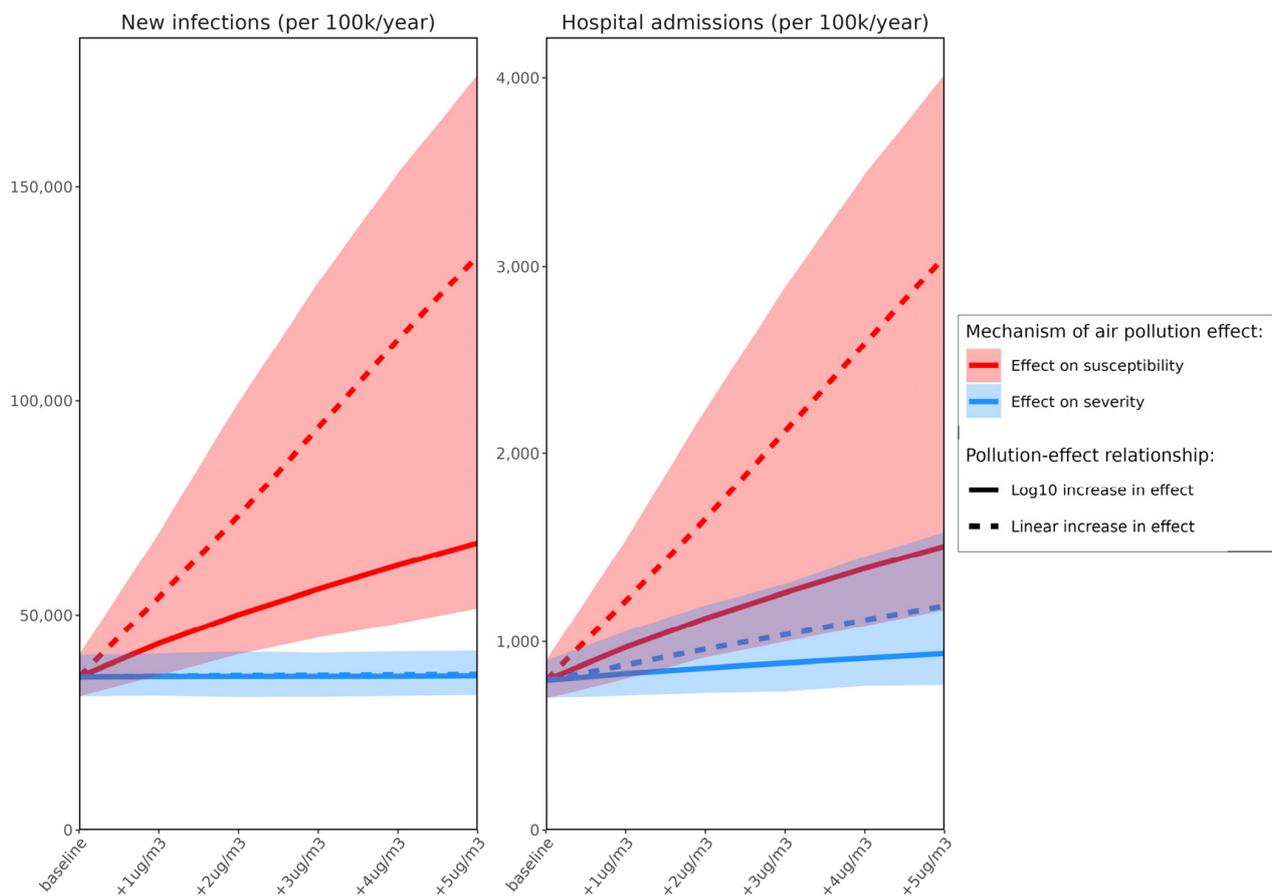
Figure 2. The projected effect of exposure to $PM_{2.5}$ (particulate matter ≤2.5 microns in diameter) (+1–5 μg/m^3) on new SARS-CoV-2 infections (**left panel**) and COVID-19 hospital admissions (**right panel**). This was simulated in 100,000 people per year with either a $\log_{10}$ (solid curves) or linear (dashed curves) increase in effect on susceptibility to SARS-CoV-2 infection (red curves) or COVID-19 severity (blue curves) with uncertainty and differences in pollutant–effect relationships shown in shaded areas surrounding the curves and between the curve pairs. See Figure S11b of the Supplementary Materials for the uncertainty attributed to the best estimate and upper and lower bounds assumed per unit increase in exposure: 13% [95% CI 8–17%] for susceptibility [12] and 11% [95% CI 6–17%] for severity [15].
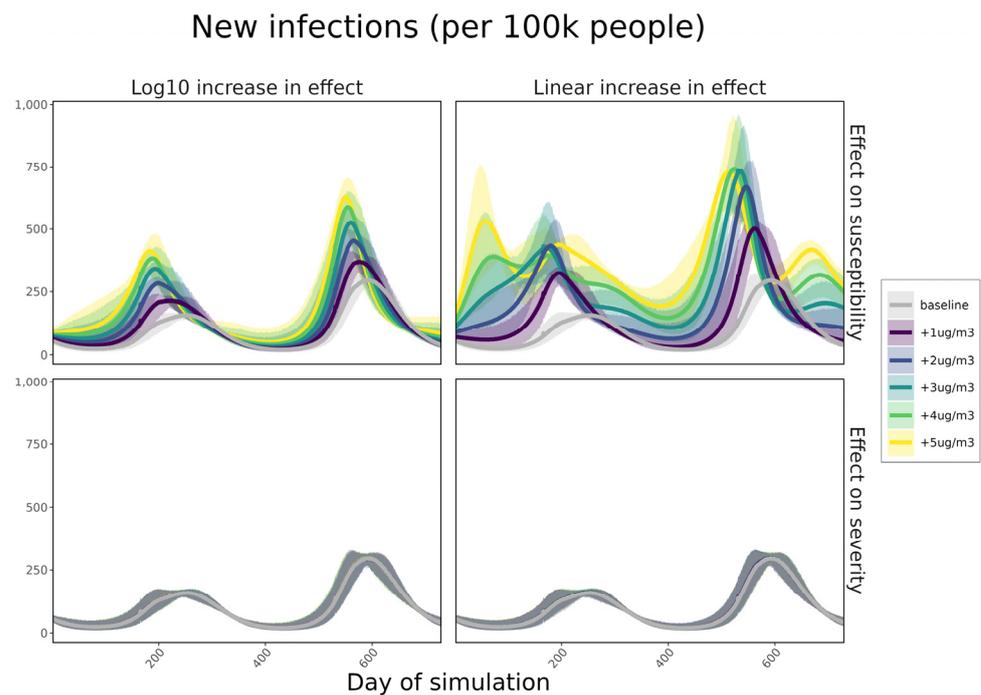
New infections (per 100k people)

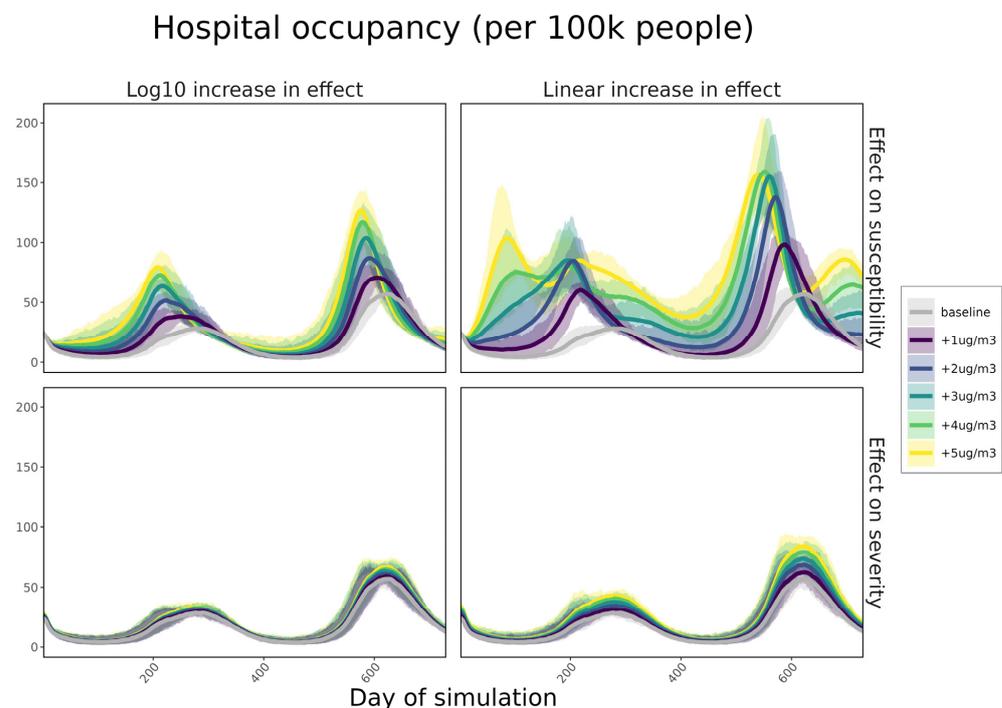**Figure 3.** Daily projected impact of exposure to PM$_{2.5}$ (particulate matter ≤2.5 microns in diameter) (+1–5 μg/m^3) on new SARS-CoV-2 infections per 100,000 people over a two-year period. Log$_{10}$ (**left panels**) and linear (**right panels**) increases in pollutant exposure effect on susceptibility to SARS-CoV-2 infection (**top row**) and COVID-19 severity (**bottom row**) are shown alongside a no exposure increase baseline (grey curves). Uncertainty is shown in shaded areas surrounding the curves.

Hospital occupancy (per 100k people)

Figure 4. Daily projected impact of exposure to PM$_{2.5}$ (particulate matter ≤2.5 microns in diameter) (+1–5 μg/m^3) on COVID-19 hospital occupancy per 100,000 people over a two-year period. Log$_{10}$ (**left panels**) and linear (**right panels**) increases in pollutant exposure effect of exposure on susceptibility to SARS-CoV-2 infection (**top row**) and COVID-19 severity (**bottom row**) are shown alongside a no exposure increase baseline (grey curves). Uncertainty is shown in shaded areas surrounding the curves.

However, if exposure to $PM_{2.5}$ affects susceptibility to SARS-CoV-2 infection (assuming a 13% [8–17%] increase per unit increase in exposure [12]), then infections could increase by 45% for a one-unit increase in exposure for a $\log_{10}$ association and by over 90% if the association is linear. Moreover, and importantly, with an increase in cases there would be a potential for onward transmission. Taken together, as $PM_{2.5}$ exposure can influence susceptibility to SARS-CoV-2 infection, and as each of these cases could transmit the virus to other people, the increase in hospitalisations will be much higher (45% for $\log_{10}$ and over 90% for a linear association, with a one-unit increase in $PM_{2.5}$) (Figure 2).

Daily projections of the potential impact of increasing $PM_{2.5}$ exposure levels on new SARS-CoV-2 infections, assuming either a $\log_{10}$ or linear effect of exposure on SARS-CoV-2 susceptibility or COVID-19 severity, are shown in Figure 3. We assume newly emerging variants with increasing infectiousness will emerge every six months and we capture the seasonality effect, therefore, if susceptibility is affected with a linear increase in effect, then waves of infections are predicted at approximately days 50, 200, 600, and 700. Peak timing and wave sizes over a two-year simulation are proportional to the historic level of $PM_{2.5}$ increase over baseline (no increase in exposure). For a $\log_{10}$ association, waves are only predicted at days 200 and 600 with less pronounced impact. As expected, when the effect is on severity, infections are not impacted.

Figure 4 shows similar timing for peaks and relative intensity levels for hospital occupancy when susceptibility is affected to those shown in Figure 3 (showing the impact on infections). Following modelled prognosis probabilities (shown in Table S1 of the Supplementary Materials), a proportion of people infected with SARS-CoV-2 and people they transmit the virus to will develop COVID-19 symptoms requiring hospitalisation. However, to a lesser extent, as expected, hospital occupancy is impacted when COVID-19 severity is effected by $PM_{2.5}$ exposure with wave peaks at approximately day 275 and day 600, noting that immunity acquired from previous infection and/or vaccination are assumed to prevent only 5% of hospital admissions. To note, acquired immunity is more protective against ICU occupancy and COVID-19 mortality, and the effect of $PM_{2.5}$ exposure decreases with each downstream outcome as illustrated in Figure S11a of the Supplementary Materials.

In the model, SARS-CoV-2 infections will result in a certain proportion of COVID-19 hospital admissions based on assumed prognosis probabilities and the protective effect of immunity from previous infection or vaccinations, which only prevent a small proportion of hospitalisations. Therefore, the relative numbers and trends of infections and hospitalisations are linked as shown in Figures 2–4 and in Figures S14 and S15 of the Supplementary Materials.

4. Discussion

Previous studies have reported an association between air pollutant exposure and the incidence of respiratory viral infections, including influenza [35], respiratory syncytial virus (RSV), rhinovirus, measles, and mumps [6]. While much of the existing evidence is based on ecological studies and subject to bias, this is also true for COVID-19, even considering the uncertainty around the mechanism and degree of association between pollutant exposure and COVID-19 burden. Here we show that, if we can assume that if exposure to $PM_{2.5}$ directly affect susceptibility to SARS-CoV-2 infection rather than COVID-19 disease severity, then the number of COVID cases attributed to this exposure will be much higher, since for each new infection there is a potential for onward transmission. Therefore, hospitalisations will also be much higher than if only severity were affected. A review carried out in 2020 suggests that chronic exposure to certain air pollutants, including $PM_{2.5}$, may lead to more severe forms of COVID-19, may delay and complicate recovery [36], and may lead to higher COVID-19 fatality rates, as shown here.

The aim of this study is to project the impact of increasing $PM_{2.5}$ exposure on either incidence or severity, not for a specific setting. We assume a given initial reproductive number and calibrate using the number of effective network contacts at the start of the

simulation period. We model new variants emerging every six months, booster doses administered every six months to high-risk groups and annually to lower risk groups at given coverage levels based on global trends, and immunity waning based on published evidence. We also assume a seasonality effect with broad weather patterns capturing some uncertainty. Key factors will influence these projections including uncertainty surrounding the frequency of variant emergence, infectiousness and transmissibility of new variants that emerge, contact behaviour, vaccination rates and efficacy of next-generation vaccines. In addition, exposure to $PM_{2.5}$ is modelled to have occurred in incremental increases from one to five $\mu g/m^3$. Again, we acknowledge the uncertainty surrounding the exact mechanism and degree to which exposure to air pollutants, like $PM_{2.5}$, are associated with COVID-19 burden. Villeneuve and Goldberg suggest that exposure–response curves between $PM_{2.5}$ and COVID-19 can also change substantially over time [37]. While their county-level study in the US suggested a positive linear relationship between exposure and response until the end of June 2021, they caution that "ecological analyses are prone to showing spurious relationships".

Practical implications of our study highlight that in order to inform setting-specific air pollutant-related public health policies to help alleviate or mitigate the potential effect of exposure on COVID-19, it will be important to project the impact of different air pollutants at various exposure levels has on the burden of COVID-19 in specific settings. Here we examine the potential impact of $PM_{2.5}$, one of several air pollutants as a marker for ambient air pollution, on the burden of COVID-19. One such study looked at air pollutant exposure from 2017 to 2019 in counties across 49 US states and used random coefficients and coordinates to map the association between exposure and COVID-19 incidence and mortality through to May 2022 [38]. While associations and attributable burden varied between regions and particulates examined, on average, $PM_{2.5}$ was found to be significantly positively associated with COVID-19 incidence across all 49 states.

While air pollutant exposure may be associated with COVID-19 transmission and severity, the extent of this effect will depend on several factors like the protective measures in place (e.g., masking, isolation, vaccination), the efficiency of the healthcare system to provide care and treatment, other socioeconomic factors (ability to access care, etc.) [36], and other environmental factors [39,40]. In addition, those most vulnerable to air pollutant exposure (e.g., those of lower socioeconomic status, the very young and very old, and those living with chronic comorbidities) will be most affected by the impact of poor air quality on COVID-19 burden.

The World Health Organization concluded that global transition from fossil fuels to renewable energy sources will not only slow climate change, but will avert air-pollution-exposure-related deaths [41], including from COVID-19. While action to transition to clean and low-carbon energy sources are ongoing, public health decision-makers must account for the immediate need in responding to respiratory health issues, including those from respiratory viral disease, like COVID-19.

5. Conclusions

Simulations demonstrate that if exposure to $PM_{2.5}$ affects not only COVID-19 severity but also makes people more susceptible to SARS-CoV-2 infection, there would be a potential for onward transmission, and the impact on hospital admissions could be over nine-fold higher. However, the mechanism and point at which exposure to $PM_{2.5}$ (the pollutant associated with the greatest proportion of adverse health effects) increases the risk of SARS-CoV-2 infection and/or COVID-19 disease severity, and the degree of these associations, are not fully understood. Therefore, additional investigation would be helpful in providing further insight into the potential role of pollutants in modifying the risk of novel viral infections, like COVID-19 [42]. A better understanding will enable improved protection and support of those most vulnerable to COVID-19 disease and to other viral respiratory disease. Our findings emphasize that multipronged regulatory actions to control air pollution should remain a critical public health priority, including the science-based adjustment of air

quality standards [42]. Last, as part of sustainable preparedness strategies for responding to viral respiratory disease outbreaks, like COVID-19, planners should consider the potential negative impact exposure to air pollutants might have.

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