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Links between air pollution and COVID-19 in England☆

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ABSTRACT

In December 2019, a novel disease, coronavirus disease 19 (COVID-19), emerged in Wuhan, People's Republic of China. COVID-19 is caused by a novel coronavirus (SARS-CoV-2) presumed to have jumped species from another mammal to humans. This virus has caused a rapidly spreading global pandemic. To date, over 300,000 cases of COVID-19 have been reported in England and over 40,000 patients have died. While progress has been achieved in managing this disease, the factors in addition to age that affect the severity and mortality of COVID-19 have not been clearly identified. Recent studies of COVID-19 in several countries identified links between air pollution and death rates. Here, we explored potential links between major fossil fuel-related air pollutants and SARS-CoV-2 mortality in England. We compared current SARS-CoV-2 cases and deaths from public databases to both regional and subregional air pollution data monitored at multiple sites across England. After controlling for population density, age and median income, we show positive relationships between air pollutant concentrations, particularly nitrogen oxides, and COVID-19 mortality and infectivity. Using detailed UK Biobank data, we further show that $PM_{2.5}$ was a major contributor to COVID-19 cases in England, as an increase of 1 m^3 in the long-term average of $PM_{2.5}$ was associated with a 12% increase in COVID-19 cases. The relationship between air pollution and COVID-19 withstands variations in the temporal scale of assessments (single-year vs 5-year average) and remains significant after adjusting for socioeconomic, demographic and health-related variables. We conclude that a small increase in air pollution leads to a large increase in the COVID-19 infectivity and mortality rate in England. This study provides a framework to guide both health and emissions policies in countries affected by this pandemic.

1. Introduction

In December 2019, a high number of pneumonia cases with an unknown aetiology were detected in Wuhan, China. A molecular analysis of samples from affected patients revealed that their symptoms were caused by an infection with a novel coronavirus, later named severe acute respiratory syndrome (SARS) coronavirus (CoV) 2 (SARS-CoV-2), the pathogenic agent of coronavirus disease 19 (COVID-19) (Zhu et al., 2020a). Within five months, this disease had affected more than 210 countries and became a global pandemic, causing devastating consequences to public health (Wang et al., 2020a). Coronaviruses are a genus of enveloped, non-segmented, positive-sense RNA viruses belonging to the family Coronaviridae and classified within the Nidovirales order (Yi et al., 2020). Historically, illnesses caused by coronaviruses have ranged in severity, with some, including human coronaviruses-229E and -OC43, causing common cold symptoms, but SARS-CoV and Middle East respiratory syndrome coronavirus have initiated outbreaks of life-threatening pneumonia (Yi et al., 2020). While the initial symptoms of COVID-19 include fever with or without respiratory syndrome, a crescendo of pulmonary abnormalities may subsequently develop in patients (Huang et al., 2020). According to recent studies, most patients present with only a mild illness, but approximately 25% of hospital-admitted patients require intensive care because of viral pneumonia with respiratory complications (Wang et al., 2020a).

While extensive research into the pathogenesis of COVID-19 suggests that the severe disease likely stems from an excessive inflammatory response (Cao, 2020), the exact predisposing factors contributing to increased clinical severity and death in patients remain unclear. Individuals over the age of 60 years or with

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SARS-CoV-2 cases and deaths are associated with regional variations in air pollution across England.

Nitrogen oxides are the main contributors to increased numbers of COVID-19 deaths and cases in the early phase of the pandemic.

We next sought to increase both the resolution and accuracy of our analysis. We gathered data on COVID-related cases and deaths from all the local authorities in England and expanded the number of the pollutant species (n = 5). We also retrieved the longitude and latitude for each local authority. The levels of ozone, nitrogen oxide, nitrogen dioxide and PM with aerodynamic diameters of 2.5 and 10 μm ($PM_{2.5}$ and PM_{10}, respectively) are reported as averages of the 10 values measured nearest the centre of each local authority in England. Local authority-level population density, mean annual earnings and median age were included as potential confounding variables (Fig. 1). We calculated the estimated regression coefficients of each variable and their respective mortality and infectivity rate ratios (Fig. 3 and Supplementary Tables 4 and 5) relative to the different air pollutants mentioned. In our single-year model (2018), higher nitrogen dioxide levels predict an increase in COVID-19 deaths and cases in the early phase of the pandemic (Fig. 3). Moreover, the levels of nitrogen dioxide have a infectivity rate ratio of 1.033 [95% confidence interval (CI): 1.043–1.022] and mortality rate ratio of 1.031 [95% CI: 1.040–1.021], indicating that a 1 $\mu g/m^3$ increase in nitrogen dioxide concentration in 2018 was associated with 3.3% more cases and 3.1% more deaths in England. Similar to nitrogen dioxide, the levels of nitrogen oxides show mortality and infectivity rate ratios of approximately 1.01 (Fig. 3). The incidence rate ratios of cases and deaths for ozone levels are less than 1, indicating that higher ozone levels lead to lower numbers of deaths and cases. $PM_{2.5}$ and PM_{10} are negatively associated with the number of cases, and they are not significant predictors of the number of COVID-19-related deaths based on 2018 air pollution data. To determine the effect of spatial-temporal variations in air pollution exposure in England, we further increased the temporal scale of our analysis to include temporally averaged air pollution data for the years 2014–2018 (Fig. 3). Our results show that the estimated effect of air pollution on COVID-19 mortality and infectivity remains roughly constant over the multi-year modelling period (Fig. 3). Levels of nitrogen oxides and nitrogen dioxide remain significantly associated with an increase in COVID-19 infectivity [OR: 1.012 95% CI: 1.016–1.008 and OR: 1.020 95% CI: 1.027–1.013, respectively] and mortality [OR: 1.015 95% CI: 1.019–1.011 and OR: 1.025 95% CI: 1.032–1.019, respectively] approximately one month after England was placed on lockdown (Fig. 3). Similarly, we found that an increase in long-term average of ozone is negatively associated with both COVID-19 mortality [OR: 0.832 95% CI: 0.864–0.801] and infectivity [OR: 0.774 95% CI: 0.806–0.743]. In the case of $PM_{2.5}$ and PM_{10}, we found a negative and statistically significant association between the long-term average of these air pollutants and COVID-19 cases [OR: 0.962 95% CI: 0.981–0.944 and OR: 0.968 95% CI: 0.981–0.955, respectively] but not for deaths.

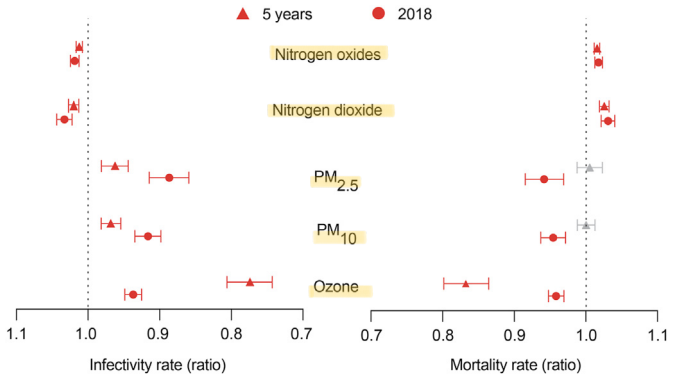

Fig. 3. Cases and deaths in local authorities.

Levels of PM pollutants and nitrogen oxides are associated with an increase in SARS-CoV2 infections in UK Biobank participants living in England.

We next used information from the UK Biobank to further assess whether people exposed to increased pollution levels are more likely to contract SARS-CoV-2 at the individual scale. This resource contains data from more than half a million UK volunteers recorded across multiple years. We obtained COVID-19 data reported by the UK Biobank up to and including April 26, 2020. This dataset contained COVID-19 test results for 1464 participants, of whom 664 were diagnosed as positive for COVID-19. The location of each subject included in the analysis is shown in Fig. 4A. Compared to the local authority case model, the UK Biobank analysis provides a higher resolution air pollution estimate (less than 2 km away from their self-reported address) and includes potentially asymptomatic cases.

In our model, we accounted for a list of confounding variables (Supplementary Table 1), which we selected based on a previous study (Williamson et al., 2020). Our analysis identified $PM_{2.5}$ and PM_{10} as significant predictors of increased SARS-CoV-2 infectivity based on our single-year exposure model (Fig. 4B). The odds ratios are 1.127 [CI: 1.173–1.083] and 1.078 [CI: 1.109–1.048] for $PM_{2.5}$ and PM_{10}, respectively (Fig. 4B). When the long-term averages of $PM_{2.5}$ and PM_{10} levels were considered, the estimated coefficients remain positive and statistically significant, with a similar magnitude to those identified based on 2018 air pollution data alone (Fig. 4B). That is, we found that a single unit increase in $PM_{2.5}$ levels was associated with a statistically significant 12% increase in COVID-19 cases, regardless of the primary exposure measure (i.e., single year or multiyear exposure). For PM_{10}, a one-unit increase was associated with approximately 8% more COVID-19 cases in the UK biobank. Interestingly, these results are inconsistent with data obtained from the subregional models, where PM was not found to predict the number of cases, which may be related to the lack of individual-level data. Nonetheless, both our subregional and individual-level models suggest that the levels of nitrogen oxides and dioxide were positively associated with COVID-19 infectivity, with an odds ratio of approximately 1.03 for both the single-year and multi-year model (Fig. 4B). Based on our results, we predict that an increase of only 1m3 in the long-term average of nitrogen dioxide levels increased COVID-19 cases by 4.5% [95% CI: 5.99%–3.05%] while a similar increase in nitrogen oxides was associated with approximately 2% more cases [95% CI: 2.92%–1.35%]. Conversely, ozone levels are not significant predictors of infectivity at the individual level, although they were significantly associated with deaths and cases at the subregional level (Figs. 3 and 4B). In addition to air pollution, we observed an association between current smokers and a lower likelihood of COVID-19 positivity than previous and non-smokers. However, according to our model, population density and predisposing health factors, such as age, sex, diabetes and a previous history of cancer and lung problems, are not predictors of the probability of being infected (Supplementary Table 6).

4. Discussion

Here, we identified associations between air pollution and COVID-19 deaths and cases in England, expanding on previous evidence linking high mortality rates in Europe with increased toxic exposure to air pollutants (Conticini et al., 2020; Ogen, 2020). Air pollution exposure and health impact estimates have been