ORIGINAL ARTICLE

Long-Term Air Pollution Exposure and COVID-19 Mortality
A Patient-Level Analysis from New York City

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Abstract

Rationale: Risk factors for coronavirus disease (COVID-19) mortality may include environmental exposures such as air pollution.

Objectives: To determine whether, among adults hospitalized with PCR-confirmed severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), long-term air pollution exposure is associated with the risk of mortality, ICU admission, or intubation.

Methods: We performed a retrospective analysis of SARS-CoV-2 PCR–positive patients admitted to seven New York City hospitals from March 8, 2020, to August 30, 2020. The primary outcome was mortality; secondary outcomes were ICU admission and intubation. We estimated the annual average fine particulate matter (particulate matter $\leqslant$2.5 μm in aerodynamic diameter [$PM_{2.5}$]), nitrogen dioxide (NO_2), and black carbon (BC) concentrations at patients' residential address. We employed double robust Poisson regression to analyze associations between the annual average $PM_{2.5}$, NO_2, and BC exposure level and COVID-19 outcomes, adjusting for age, sex, race or ethnicity, hospital, insurance, and the time from the onset of the pandemic.

Results: Among the 6,542 patients, 41% were female and the median age was 65 (interquartile range, 53–77) years. Over 50% self-identified as a person of color (n = 1,687 [26%] Hispanic patients; n = 1,659 [25%] Black patients). Air pollution exposure levels were generally low. Overall, 31% (n = 2,044) of the cohort died, 19% (n = 1,237) were admitted to the ICU, and 16% (n = 1,051) were intubated. In multivariable models, a higher level of long-term exposure to $PM_{2.5}$ was associated with an increased risk of mortality (risk ratio, 1.11 [95% confidence interval, 1.02–1.21] per 1-μg/m^3 increase in $PM_{2.5}$) and ICU admission (risk ratio, 1.13 [95% confidence interval, 1.00–1.28] per 1-μg/m^3 increase in $PM_{2.5}$). In multivariable models, neither NO_2 nor BC exposure was associated with COVID-19 mortality, ICU admission, or intubation.

Conclusions: Among patients hospitalized with COVID-19, a higher long-term $PM_{2.5}$ exposure level was associated with an increased risk of mortality and ICU admission.

Keywords: air pollution exposure; COVID-19 mortality; COVID-19 ICU admission; race- or ethnicity-specific effects

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At a Glance Commentary

Scientific Knowledge on the Subject: Risk factors for coronavirus disease (COVID-19) mortality may extend to environmental exposures, including air pollution. Ecologic studies suggest that a higher long-term air pollution exposure is associated with a higher risk of COVID-19 mortality. However, ecologic analyses are limited by the lack of individual-level data, potentially leading to residual confounding.

What This Study Adds to the Field: These individual-level data suggest that among patients hospitalized with COVID-19, a higher long-term fine particulate matter exposure level is associated with an increased risk of mortality and ICU admission; exploratory analyses suggest that these estimated effects may differ by race or ethnicity. Given that air pollution is a modifiable risk factor, environmental regulations to reduce air pollution exposure may be a critical public health measure to improve infectious disease mortality in this and future pandemics.

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic has resulted in extensive morbidity and mortality (1). Individual-level risk factors for severe illness and death include male sex, older age, and the presence of comorbid diseases such as diabetes and obesity. The effects of systemic racism on coronavirus disease (COVID-19) outcomes have been extensively documented, with disproportionate infections and deaths occurring among Black, Latinx, and Indigenous populations (2). Risk factors for severe illness may extend to environmental exposures, including ambient air pollution, such that persons with a higher long-term air pollution exposure level may experience higher mortality after infection with SARS-CoV-2 (3). Recently published ecologic studies suggest that a higher long-term air pollution exposure level is associated with a higher COVID-19 mortality rate (4, 5). However, these ecologic analyses are limited by the lack of individual-level data, potentially leading to residual confounding (5, 6). Understanding the role of air pollution exposure in modifying COVID-19 disease mortality and severity is critical to informing public health policy. Furthermore, analyses of air pollution impacts on COVID-19 outcomes should include socioeconomically diverse and vulnerable populations.

Multiple biological pathways support a plausible link by which long-term air pollution exposure may increase the risk of death or severe disease once a person is infected with SARS-CoV-2. For example, long-term fine particulate matter (particulate matter $\leqslant$2.5 μm in aerodynamic diameter [$PM_{2.5}$]), black carbon (BC), and nitrogen dioxide (NO_2) exposures impair lung health, increasing the risk of asthma and chronic obstructive lung disease development and respiratory disease hospitalizations (7–10). Improvements in air pollution, conversely, reduce the risk of asthma and improve lung function (11, 12). Air pollution exposure, including $PM_{2.5}$ and NO_2 exposure, has been shown to increase the risk of acute respiratory distress syndrome, a hypoxemic respiratory failure syndrome seen in patients with severe COVID-19 disease (13, 14). Air pollution exposure may alter the pulmonary immune response to viral infections (15). For example, mice exposed to particulate matter have a blunted early immune response to respiratory syncytial virus infection, which is followed by a subsequent increase in inflammation and overall infection severity (16). Human respiratory epithelial cells exposed to diesel exhaust and influenza demonstrate a Toll-like receptor 3 pathway–dependent increase in inflammation (17). Long-term air pollution exposure is also associated with an increased risk of cardiovascular disease and metabolic syndrome, which are known risk factors for COVID-19 mortality (18–20). Given these links, we hypothesize that once a person is infected, a higher long-term air pollution exposure level may increase the risk of more severe COVID-19 disease and death as compared with a lower exposure level.

We conducted a retrospective analysis to examine how long-term exposure to air pollution, as indexed by the December 2018 to December 2019 annual average concentrations of $PM_{2.5}$ and NO_2 and the December 2017 to December 2018 annual average concentration of BC, is associated with COVID-19 outcomes among a cohort of patients hospitalized with COVID-19. Specifically, we leveraged individual-level electronic health record data from inpatient COVID-19 encounters within seven New York City (NYC) hospitals and linked these clinical data with air pollution levels at residential addresses and 1-km radial buffer socioeconomic indicators. Our primary outcome was mortality; secondary outcomes included ICU admission and the need for intubation and mechanical ventilation.

Methods

Study Sample

We extracted electronic health data for individuals with COVID-19 requiring hospitalization from seven NYC Hospitals: the NYC Health and Hospitals (H&H) Elmhurst and Queens hospitals and the Mount Sinai Hospital System hospitals, including Mount Sinai Brooklyn, Mount Sinai Queens, the Mount Sinai Hospital, Mount Sinai Morningside, and Mount Sinai West. Included patients were admitted between March 8, 2020, and August 30, 2020; were 18 years of age or older; had received a positive SARS-CoV-2 PCR test result within 7 days of hospitalization; and resided within NYC with a valid residential address.

Ethics

The study protocol was approved by the institutional review board at the Icahn School of Medicine at Mount Sinai and the research committees of NYC H&H Elmhurst and Queens hospitals.

Electronic Health Record Data: Study Inclusion, Covariates, and Outcomes

Electronic health record data were collected from the first recorded COVID-19–related admission (i.e., March 8, 2020) through August 30, 2020, therefore capturing the NYC spring and summer COVID-19 surge. The Mount Sinai Hospitals are private hospitals located in Manhattan (Mount Sinai Hospital [1,141 beds], Mount Sinai Morningside [495 beds], and Mount Sinai

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This article has an online supplement, which is accessible from this issue's table of contents at www.atsjournals.org.

West [514 beds]), Brooklyn (Mount Sinai Brooklyn [212 beds]), and Queens (Mount Sinai Queens [235 beds]). The NYC H&H Elmhurst (545 beds) and Queens (253 beds) hospitals are public hospitals in Queens and are situated within highly diverse communities, including large Hispanic, Black, Asian, and immigrant communities with high poverty rates and significant healthcare disparities (21).

For each patient encounter, we extracted the date of admission, hospital, sex, age, self-reported race or ethnicity, residential address, insurance vendor, and date of SARS-CoV-2 PCR testing. SARS-CoV-2 PCR testing data included the hospital system and NYC Department of Health testing data. We classified self-reported race or ethnicity as White, Black, Hispanic, Asian, or other and classified insurance status as private or Medicare insurance, Medicaid insurance, or other or no insurance. We calculated the onset time as the difference in days from the date of the first COVID-19–related hospital admission (i.e., March 8, 2020) and the date of the first hospital admission for a given patient. For patients with multiple encounters, data collected at the first encounter that met inclusion criteria were used as covariates in analyses.

We assessed patient outcomes, including mortality, ICU admission, and intubation, across all hospital encounters and through August 30, 2020. Electronic health record mortality data also included health record–linked postdischarge deaths, such as death after transfer to an inpatient hospice facility. We classified patients assigned to the ICU at any point of any hospitalization as having an ICU admission, and we classified patients with an endotracheal tube, nonsurgical airway intubation, or surgical intubation during any encounter as being intubated with mechanical ventilation.

Spatial Data

We estimated particulate matter ($PM_{2.5}$ in $\mu g/m^3$), BC (in $\mu g/m^3$), and NO_2 (in parts per billion) concentrations at the patient-level residential street address provided at the time of admission. Briefly, we leveraged the NYC Community Air Survey (NYCCAS) annual average data that integrate air pollution samples collected every 2 weeks from 93 monitoring sites located throughout the five NYC boroughs with land-use regression models to estimate exposure levels (22, 23). The NYCCAS monitoring sites are at street level, with 80% of sites being chosen at random and 20% being strategically placed to ensure representation of all community districts and major roadways. As the response period of long-term air pollution exposure on COVID-19 outcomes is unknown, we examined associations between the most recently available, 1-year average exposure level for each pollutant and these outcomes. Analyses thus use the annual average concentration as predicted from the NYCCAS model and extracted for the 100-m grid of each residential address; for $PM_{2.5}$ and NO_2, the average from December 2018 to December 2019, the year that ended 3 months before the start of March 2020 NYC spring surge, was used, and for BC, the average from December 2017 to December 2018 was used, as the December 2018 to December 2019 estimates are not yet available. The accuracy of the final adjusted NYCCAS modeling approach was assessed in Clougherty and colleagues' 2013 study (24), which reported R^2 values for the predicted versus measured $PM_{2.5}$ ($R^2 = 0.86$), BC ($R^2 = 0.60$), and NO_2 ($R^2 = 0.65$) concentrations.

We created 1-km radial buffers around the geographic centroid of each 2010 census block in NYC. We used census data to estimate the following for each buffer: the percentage of persons living below the poverty level, the number of housing units, and the percentage of persons aged ⩾25 years with at least a high school education (25). We used patient residential addresses to identify their census block of residence and then used the block of residence to link the patient to the corresponding 1-km radial buffer.

Statistical Analyses

We calculated descriptive statistics for patient characteristics (medians and interquartile ranges [IQRs] for continuous variables and frequencies and proportions for categorical variables). We hypothesized that patient socioeconomic status was reflected by self-reported race or ethnicity, hospital of presentation, and insurance status. To confirm this, we examined the relationships between these variables and the 1-km radial buffer indicators.

Overall data completeness was high; therefore, we made no imputations and excluded patients with missing covariates ($n = 236$, 3.6%) from multivariable analyses. To account for potential selection bias, we generated propensity scores to include as weights in analyses, which were calculated by using air pollution measures specific to each analysis of patient age, sex, race or ethnicity, hospital of presentation, onset time, and insurance (26).

Given that our COVID-19 outcomes of interest were common, we employed bivariate and multivariable Poisson regression models with robust error variance to examine associations between each air pollutant and the risk of COVID-19 mortality, ICU admission, and intubation, which were considered separately (27, 28), by using the R package *sandwich* (R Foundation for Statistical Computing) (29, 30). Multivariable models adjusted for patient age, sex, race or ethnicity, onset time, hospital of presentation, and insurance. Sensitivity models additionally adjusted for 1-km neighborhood buffer variables, including the percentage of persons living below poverty, number of housing units, and percentage of persons aged ⩾25 years with at least a high school education.

We explored interactions between air pollution and self-identified race or ethnicity. First, we introduced an air pollution × race or ethnicity (referent group: White individuals) interaction term in the main regression model. Then, we examined associations between air pollution and COVID-19 outcomes in models stratified by race or ethnicity. As we observed that persons of color were younger than persons who self-identified as White, we further explored interactions among air pollution, self-identified race or ethnicity, and age dichotomized around the study median age of 65 years.

To examine whether the identified associations persisted at lower air pollution exposure levels, we repeated analyses while excluding patients with air pollution exposure levels above *a priori* thresholds, specifically the U.S. Environmental Protection Agency (EPA) annual standard (i.e., 12 $\mu g/m^3$), the previous World Health Organization (WHO) annual guidance targets (i.e., 10 $\mu g/m^3$), and the study's third quartile of exposure distribution (31, 32). Given the study's $PM_{2.5}$ IQR of 6.4–7.1 $\mu g/m^3$, we are unable to examine the updated WHO annual $PM_{2.5}$ standard of 5 $\mu g/m^3$.

Analyses were performed by using R version 3.5.2 (33).

Table 1. Patient Characteristics ($N = 6,542$)

Female, *n* (%)	2,690 (41.1)
Age, yr, median (IQR)	65 (53–77)
Race or ethnicity, *n* (%)	
White	1,132 (17.3)
Black	1,659 (25.4)
Hispanic	1,687 (25.8)
Asian	442 (6.8)
Other	1,512 (23.1)
Missing	110 (1.7)
Insurance, *n* (%)	
Private or Medicare	4,388 (67.1)
Medicaid or underinsured	1,880 (28.7)
Other	142 (2.2)
Missing	132 (2.0)
Hospital of presentation, *n* (%)	
Elmhurst Hospital, Health and Hospital	1,115 (17.0)
Queens Hospital, Health and Hospital	1,024 (15.7)
Mount Sinai Brooklyn Hospital	837 (12.8)
Mount Sinai Hospital	1,405 (21.5)
Mount Sinai Queens Hospital	869 (13.3)
Mount Sinai Morningside Hospital	818 (12.5)
Mount Sinai West Hospital	471 (7.2)
Missing	3 (0.05)
Onset time, d, median (IQR)*	26 (19–35)
Air pollution, annual exposure, median (IQR)†	
$PM_{2.5}$, $\mu g/m^3$	6.86 (6.39–7.13)
BC, $\mu g/m^3$	0.76 (0.67–0.86)
NO_2, ppb	18.24 (16.44–19.71)
One-kilometer radial buffer indicators, median (IQR)‡	
Proportion of persons below poverty line	0.18 (0.14–0.25)
Proportion of persons aged >25 yr with at least a high school education	0.8 (0.74–0.86)
Housing units	23,156 (13,923–35,520)
Outcomes, *n* (%)	
Deceased	2,044 (31.2)
ICU admission	1,237 (18.9)
Intubation and mechanical ventilation	1,051 (16.1)

Definition of abbreviations: BC = black carbon; IQR = interquartile range; NO_2 = nitrogen dioxide; $PM_{2.5}$ = particulate matter ⩽2.5 μm in aerodynamic diameter.
*Days between the first encounter in the cohort and the patient's date of admission.
†Air pollution exposure at each residential address was estimated on a 100-m grid by integrating air pollution samples with land-use regression models. Fine particulate matter ($PM_{2.5}$) and NO_2 concentrations are December 2018 to December 2019 annual averages, and the BC concentration is the December 2017 to December 2018 annual average.
‡One-kilometer neighborhood buffers were generated from the 2012–2016 American Community Survey census data set and were assigned to each patient by using their residential address.

Results

The demographic characteristics of patients ($N = 6,542$) included in the cohort are presented in Table 1. The largest proportion of patients was from the Mount Sinai Hospital ($n = 1,405$) and Elmhurst NYC H&H Hospital ($n = 1,115$). Patients were 41% female ($n = 2,690$), and the median age was 65 years (IQR, 53–77 yr). Twenty-six percent ($n = 1,687$), 25% ($n = 1,659$), 7% (442), and 23% ($n = 1,512$) of patients self-identified as Hispanic, Black, Asian, or other, respectively. Sixty-seven percent ($n = 4,388$) of the cohort were insured privately or through Medicare; 29% ($n = 1,880$) were insured through Medicaid or emergency Medicaid. Across all patients, the median (IQR) annual levels of exposure to $PM_{2.5}$, BC, and NO_2 were 6.9 (6.4–7.1) $\mu g/m^3$, 0.76 (0.67–0.86) $\mu g/m^3$, and 18.2 (16.4–19.7) ppb, respectively. Exposure levels were highly correlated (Pearson correlation: $PM_{2.5}$ and NO_2 $r = 0.86$; $PM_{2.5}$ and BC $r = 0.88$; NO_2 and BC $r = 0.90$; $P < 0.01$). Figure 1 shows that cases were distributed among all NYC boroughs, with the exception of low coverage in Staten Island, and also shows that air pollution exposure levels were variable across patients included in the study.

Overall, 31% ($n = 2,044$) of the cohort died, 19% ($n = 1,237$) were admitted to the ICU, and 16% ($n = 1,051$) were intubated. In an examination of patient demographics served by each hospital, we noted that the hospitals that serve Queens, specifically Mount Sinai Queens and the Elmhurst and Queens NYC H&H hospitals, had the largest proportion of patients insured through Medicaid (chi-square $P < 0.01$; *see* Figure E1A in the online supplement). Elmhurst and Queens NYC H&H hospitals disproportionately served populations that self-identified as being of color (chi-square $P < 0.01$; Figure E1B). Mortality rates differed by hospital, with the lowest mortality rates being demonstrated in Manhattan-based hospitals (chi-square $P < 0.01$).

Mortality rates differed by race or ethnicity and insurance (Figure 2). Persons self-identifying as White and Asian had the highest mortality rates (White: $n = 409$ [36%]; Asian: $n = 153$ [35%]; other: $n = 506$ [34%]; Black: $n = 483$ [29%]; Hispanic: $n = 454$ [27%]; chi-square $P < 0.01$). Persons self-identifying as White had the highest median age (White: 73.5 [IQR, 62–84] yr; Black: 66 [IQR, 56–76] yr; Hispanic: 61 [IQR, 48–73] yr; Asian: 64 [IQR, 54–75] yr; other: 62 [IQR, 51–74] yr; data not shown, Kruskal-Wallis $P < 0.01$). When age was stratified around the cohort median of 65 years, we found that mortality among those >65 years was highest in persons self-identifying as Asian ($n = 107$, 53%) and lowest in persons self-identifying as Black ($n = 332$, 39%). Among those ⩽65 years, mortality was highest in persons self-identifying as other ($n = 197$, 23%) and lowest in persons self-identifying as White ($n = 56$, 16%) (Table E1).

The 1-km radial buffer indicators of poverty, high school education, and housing density varied by hospital, self-identified race or ethnicity, and insurance (Figure 3), suggesting that these variables reflected both individual and 1-km radial buffer socioeconomic composition. Air pollution exposure levels were higher for 1-km radial buffers with more housing units, higher education levels, and higher median incomes (Figure 4).

Associations between Air Pollution Exposure and COVID-19 Mortality

In unadjusted models that examined associations between air pollution exposure levels, considered separately, and COVID-19 mortality, higher levels of exposure to $PM_{2.5}$,

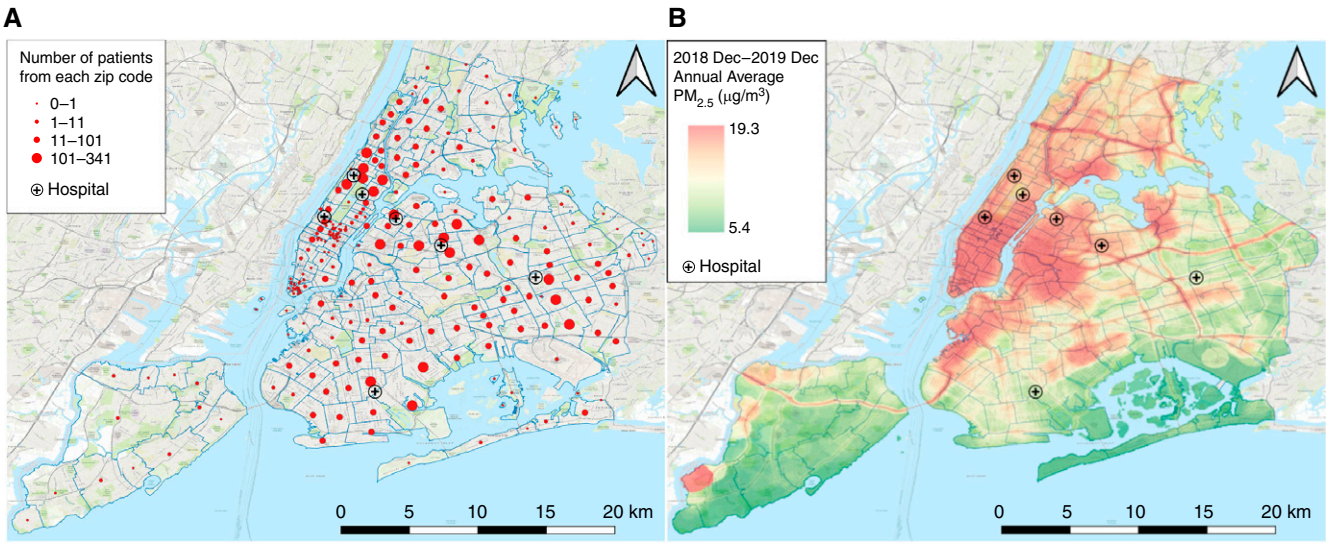

Figure 1. Distribution of patients and fine particulate matter ($PM_{2.5}$) exposure across New York City (NYC). These figures demonstrate (*A*) sampling of patients across NYC boroughs as well as (*B*) the variability of air pollution exposure. $PM_{2.5}$ = particulate matter ≤2.5 μm in aerodynamic diameter.

BC, and NO_2 were associated with a reduced risk of mortality ($PM_{2.5}$: risk ratio [RR], 0.94 [95% confidence interval (CI), 0.89–0.98] per 1-μg/m^3 increase; BC: RR, 0.57 [95% CI, 0.44–0.72] per 1-μg/m^3 increase; NO_2: RR, 0.97 [95% CI, 0.96–0.98] per 1-ppb increase) (Table 2). However, after propensity score weighting and adjustment for individual-level factors, including age, sex, race or ethnicity, hospital, time since pandemic onset, and insurance, a 1-μg/m^3-higher $PM_{2.5}$ exposure was associated with an 11% increased risk of mortality (RR, 1.11 [95% CI, 1.02–1.21] per 1-μg/m^3 increase) (Table 2; complete model output in Table E2). Neither BC nor NO_2 exposure levels were associated with an increased risk of mortality (BC: RR, 1.19 [95% CI, 0.75–1.88] per 1-μg/m^3 increase; NO_2: RR, 1.00 [95% CI, 0.98–1.03] per 1-ppb increase). The complete mortality model output for BC and NO_2 exposure is provided in Tables E3 and E4.

Associations between Air Pollution Exposure and COVID-19 ICU Admission, Intubation, and Mechanical Ventilation

In unadjusted models that examined associations between air pollution exposure and COVID-19 ICU admission, higher exposure to $PM_{2.5}$, BC, and NO_2 was associated with an increased risk of ICU admission ($PM_{2.5}$: RR, 1.07 [95% CI, 1.02–1.13] per 1-μg/m^3 increase; BC: RR, 1.49 [95% CI, 1.11–2.01] per 1-μg/m^3 increase; NO_2: RR, 1.02 [95% CI, 1.01–1.04] per 1-ppb increase). In multivariable models with propensity score weighting and adjustment for individual-level factors, including age, sex, race or ethnicity, hospital, time since pandemic onset, and insurance, a 1-μg/m^3-higher $PM_{2.5}$ exposure level was associated with a 13% increased risk of ICU admission (RR, 1.13 [95% CI, 1.00–1.28] per 1-μg/m^3 increase) (Tables E5–E7).

In unadjusted models, higher air pollution exposure levels were associated with a decreased risk of intubation and mechanical ventilation ($PM_{2.5}$: RR, 0.92 [95% CI, 0.86–0.99] per 1-μg/m^3 increase; BC: RR, 0.61 [95% CI, 0.43–0.87] per 1-μg/m^3 increase; NO_2: RR, 0.97 [95% CI, 0.95–0.99] per 1-ppb increase). After propensity score weighting and adjustments, $PM_{2.5}$, BC, and NO_2 exposure were not associated with the risk of intubation and mechanical ventilation ($PM_{2.5}$: RR, 1.05 [95% CI, 0.91–1.20] per 1-μg/m^3 increase; BC: RR, 0.91 [95% CI, 0.43–1.92] per 1-μg/m^3 increase; NO_2: RR, 1.00 [95% CI, 0.96–1.04] per 1-ppb increase; Tables E8–E10).

Associations between Air Pollution and COVID-19 Outcomes Modified by Race or Ethnicity

Annual average $PM_{2.5}$ exposure differed by self-identified race or ethnicity, although IQRs overlapped (Black: median, 6.64 μg/m^3 [IQR, 6.18–7.01 μg/m^3]; Hispanic: median, 6.98 μg/m^3 [IQR, 6.77–7.24 μg/m^3]; Asian: median, 6.97 μg/m^3 [IQR, 6.43–7.23 μg/m^3]; other: median, 6.82 μg/m^3 [IQR, 6.43–7.07 μg/m^3]; White: median, 6.91 μg/m^3 [IQR, 6.22–7.41 μg/m^3]; Kruskal-Wallis $P < 0.01$). In stratified adjusted models, higher exposure to $PM_{2.5}$ was associated with an increased risk of mortality, ICU admission, and intubation among patients self-identifying as Hispanic (mortality: RR, 1.22 [95% CI, 1.02–1.46] per 1-μg/m^3 increase in $PM_{2.5}$; *P* for interaction [*P*-interaction] = 0.20; ICU admission: RR, 1.26 [95% CI, 1.02–1.56] per 1-μg/m^3 increase in $PM_{2.5}$; *P*-interaction = 0.42; intubation: RR, 1.37 [95% CI, 1.08–1.73] per 1-μg/m^3 increase in $PM_{2.5}$; *P*-interaction = 0.22) (Table 3), and there was an increased risk of mortality among patients self-identifying as other (RR, 1.20 [95% 1.02–1.41] per 1-μg/m^3 increase; *P*-interaction = 0.04). Associations between $PM_{2.5}$ exposure levels and COVID-19 outcomes were not statistically significant in stratified analyses among any other race or ethnicity group. All *P*-interaction terms were >0.10, except for in the mortality models for Black race (*P*-interaction = 0.08) and other race or ethnicity (*P*-interaction = 0.04).

Given the observation that patients of color were younger than patients who self-identified as White, we further stratified the sample around the study age median of 65 years. Among younger Hispanic patients, long-term $PM_{2.5}$ exposure was positively associated with the risk of mortality, ICU admission, and intubation (mortality: RR, 1.44 [95% CI, 1.07–1.93] per 1-μg/m^3

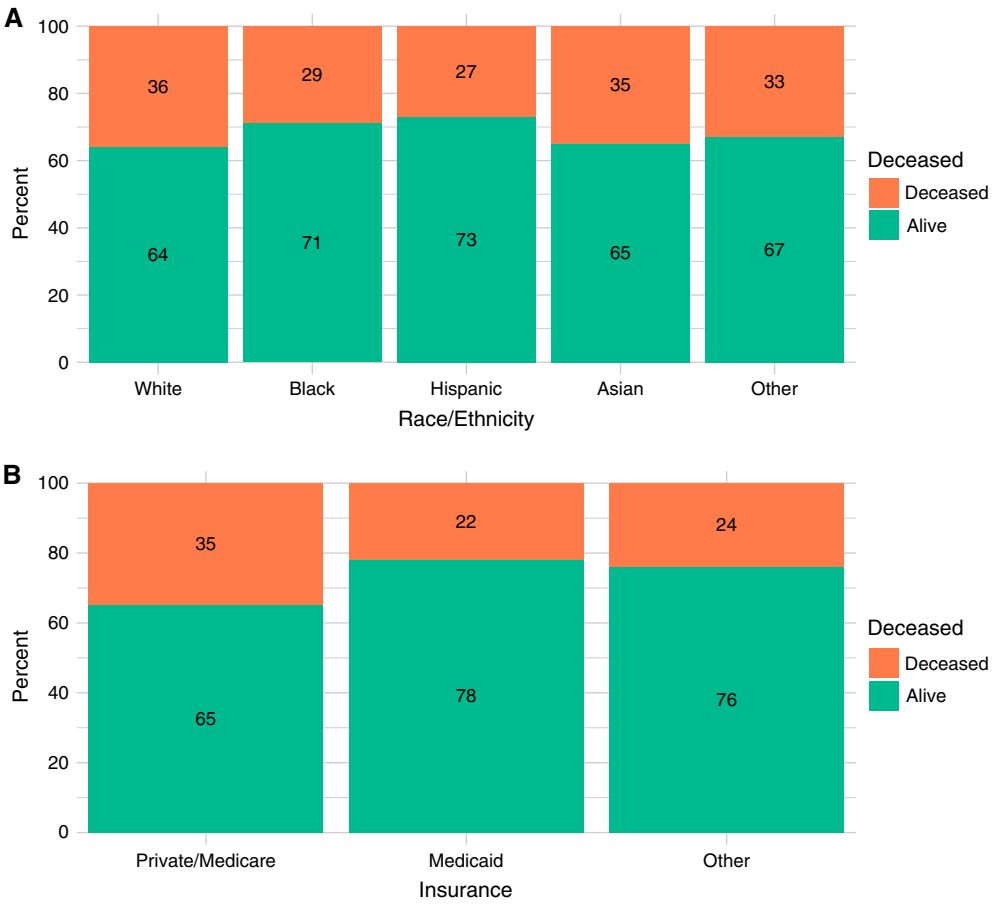

Figure 2. Inpatient coronavirus disease (COVID-19) mortality by (*A*) self-identified race or ethnicity and (*B*) insurance status. (*A*) Persons self-identified as White and Asian had mortality rates of 36% ($n = 409$) and 35% ($n = 153$), respectively. The median age of persons self-identifying as White was significantly higher than that of persons identifying as other races or ethnicities (White: 73.5 [interquartile range (IQR), 62–84] yr; Black: 66 [IQR, 56, 76] yr; Hispanic: 61 [IQR, 48–73] yr; Asian: 64 [IQR, 54–75] yr; other: 62 [IQR, 51–74] yr; Kruskal-Wallis $P < 0.01$). (*B*) Thirty-five percent ($n = 1,553$) of persons with private or Medicare insurance died. The age distribution of this group was higher (median age of those with private or Medicare insurance who died, 76 [IQR, 68–84] yr) than that of the other groups combined (median age of those without private or Medicare insurance who died, 60 [IQR, 51.5–67] yr).

increase in $PM_{2.5}$; *P*-interaction = 0.09; ICU admission: RR, 1.43 [95% CI, 1.12–1.83] per 1-μg/m^3 increase in $PM_{2.5}$; *P*-interaction = 0.48; intubation: RR, 1.54 [95% CI, 1.16–2.04] per 1-μg/m^3 increase in $PM_{2.5}$; *P*-interaction = 0.36). Furthermore, among younger patients self-identifying as other, a higher level of exposure to $PM_{2.5}$ was associated with an increased risk of ICU admission (RR, 1.43 [95% CI, 1.07–1.90] per 1-μg/m^3 increase in $PM_{2.5}$; *P*-interaction = 0.10). All *P*-interaction terms were >0.10 except for in the mortality model for Hispanic ethnicity (*P*-interaction = 0.09).

Examination of Air Pollution Exposure Thresholds

In total, 6,528 (99.8%) hospitalized patients had annual $PM_{2.5}$ exposure levels below the U.S. EPA annual national ambient air quality standard of 12 μg/m^3, 6,433 (98.3%) hospitalized patients had annual exposure levels below the previous WHO annual guideline of 10 μg/m^3, and 4,899 (74.9%) hospitalized patients had annual $PM_{2.5}$ exposure levels below the study's third quartile of 7.13 μg/m^3 (Figure E2). Similarly, 2,041 (99.9%) deaths occurred in persons with annual $PM_{2.5}$ exposure levels below the U.S. EPA annual national ambient air quality standard of 12 μg/m^3, 2,017 (98.7%) deaths occurred in persons with annual exposure levels below the previous WHO annual guideline of 10 μg/m^3, and 1,521 (74.4%) deaths occurred in persons with annual exposure levels below the study's third quartile of 7.13 μg/m^3.

The risk of mortality was similar across stratifications of $PM_{2.5}$ exposure. In adjusted analyses, we observed positive associations between $PM_{2.5}$ concentrations at ⩽12 μg/m^3 ($N = 6,292$ patients included in the analyses), ⩽10 μg/m^3 ($N = 6,197$ patients included in the analyses), and ⩽7.13 μg/m^3 ($N = 4,702$ patients included in analyses) considered separately and the risk of mortality (⩽12 μg/m^3: RR, 1.12 [95% CI, 1.02–1.22] per 1-μg/m^3 increase; ⩽10 μg/m^3: RR, 1.15 [95% CI, 1.04–1.27] per 1-μg/m^3 increase; ⩽7.13 μg/m^3: RR, 1.17 [95% CI, 1.01–1.36] per 1-μg/m^3 increase; Table 4). We observed positive associations between $PM_{2.5}$ concentrations at ⩽12 μg/m^3 or ⩽10 μg/m^3 considered separately and the risk of ICU admission (⩽12 μg/m^3: RR, 1.16 [95% CI, 1.02–1.31] per 1-μg/m^3 increase; ⩽10 μg/m^3: RR, 1.20 [95% CI, 1.04–1.39] per 1-μg/m^3 increase). $PM_{2.5}$ at ⩽7.13 μg/m^3 was positively but not significantly associated with ICU admission (RR, 1.03 [95% CI, 0.83–1.28] per 1-μg/m^3 increase).

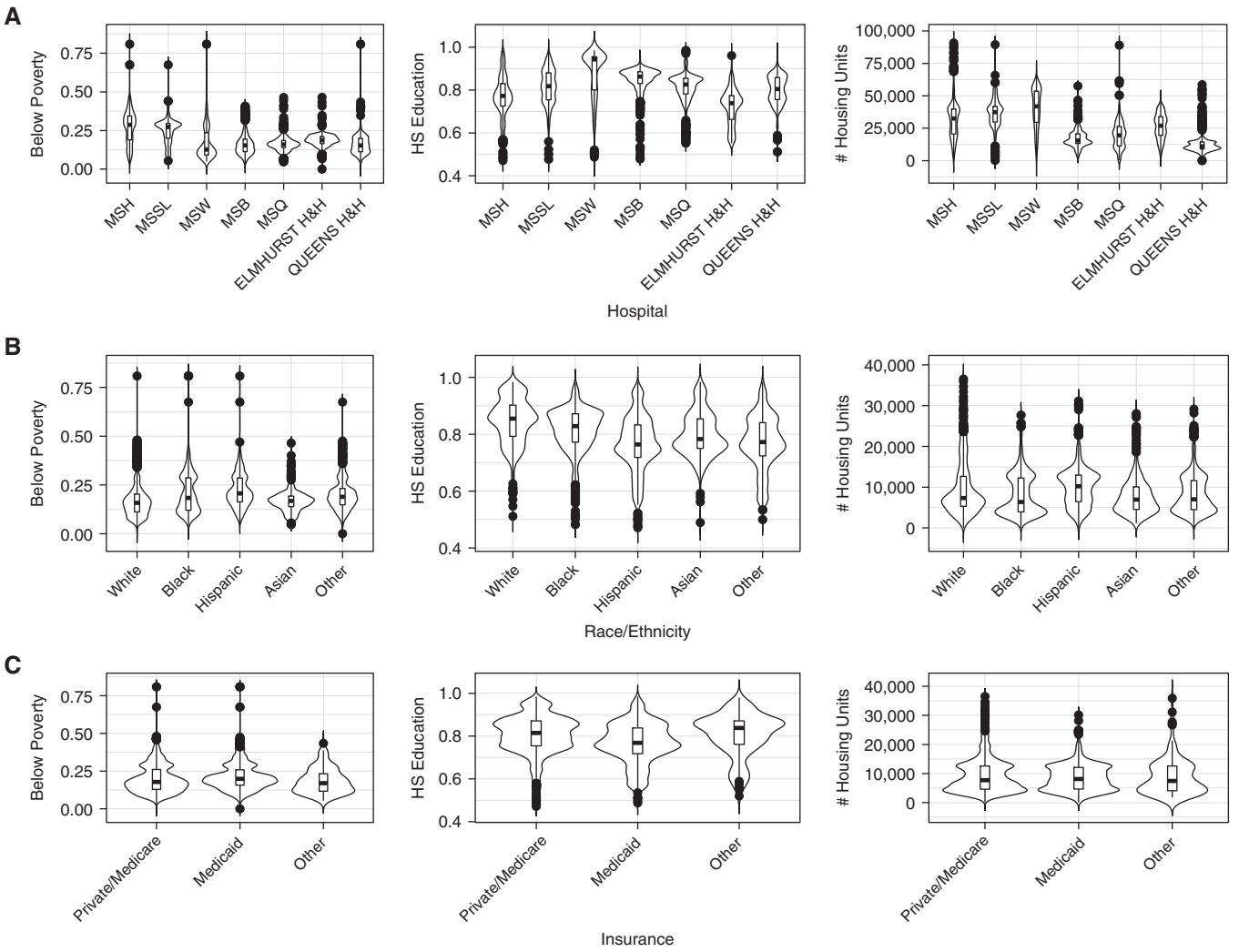

Figure 3. Violin plots, in which curved lines represent kernel probability density, with box-plot insets of the relationship between the hospital, race or ethnicity, and insurance and the 1-km radial buffer indications, including the percentage of persons living below poverty, the percentage of persons aged ⩾25 years with at least a high school education, and the housing unit density. (*A*) Patients presenting to different hospitals resided in areas with differing levels of poverty, high school education attainment, and housing density (all Kruskal-Wallis $P < 0.01$). (*B*) Patients' self-identified race or ethnicity differed by the 1-km radial buffer indications of poverty, high school education, and housing density (all Kruskal-Wallis $P < 0.01$). (*C*) The insurance status differed by poverty and high school education levels (both Kruskal-Wallis $P < 0.01$) but did not differ by housing density levels ($P = 0.20$). ELMHURST H&H = Elmhurst NYC H&H Hospital; H&H = Health and Hospitals; HS = high school; MSB = Mount Sinai Brooklyn; MSH = Mount Sinai Hospital; MSQ = Mount Sinai Queens; MSSL = Mount Sinai Morningside; MSW = Mount Sinai West; NYC = New York City; QUEENS H&H = Queens NYC H&H Hospital.

Discussion

The COVID-19 pandemic has highlighted the critical impact of environmental, social, and structural factors, rooted in poverty and racism, on health disparities. Leveraging hospital admission data from the spring and summer 2020 COVID-19 surge in NYC and including private and public hospitals with patients from wide-ranging socioeconomic backgrounds, these individual-level analyses suggest that vulnerability factors extend to long-term air pollution exposure. Specifically, these individual-level data suggest that among patients hospitalized with COVID-19, patients with higher long-term air pollution exposure levels are at an increased risk of death and ICU admission, as compared with those with lower long-term air pollution exposure levels.

These data suggest that among hospitalized patients a 1-$\mu g/m^3$-higher long-term $PM_{2.5}$ exposure level is associated with an 11% increase in the risk of death (RR, 1.11 [95% CI, 1.02–1.21]) and a 13% increase in the risk of ICU admission (RR, 1.13 [95% CI, 1.00–1.28]). These results are consistent with recent ecologic analyses, which found that a 1-$\mu g/m^3$-higher county-level $PM_{2.5}$ exposure level is associated with an 11% increase (95% CI, 6–17%) in the county-level mortality rate (5). Similarly, an analysis of COVID-19 mortality across 3,143 U.S. counties found that a 1-$\mu g/m^3$-higher $PM_{2.5}$ exposure level was associated with an 8% increase in the COVID-19 mortality rate (34). In Italy, an ecologic analysis found that a 1-$\mu g/m^3$-higher long-term $PM_{2.5}$ exposure level was associated with a 9% increase in the

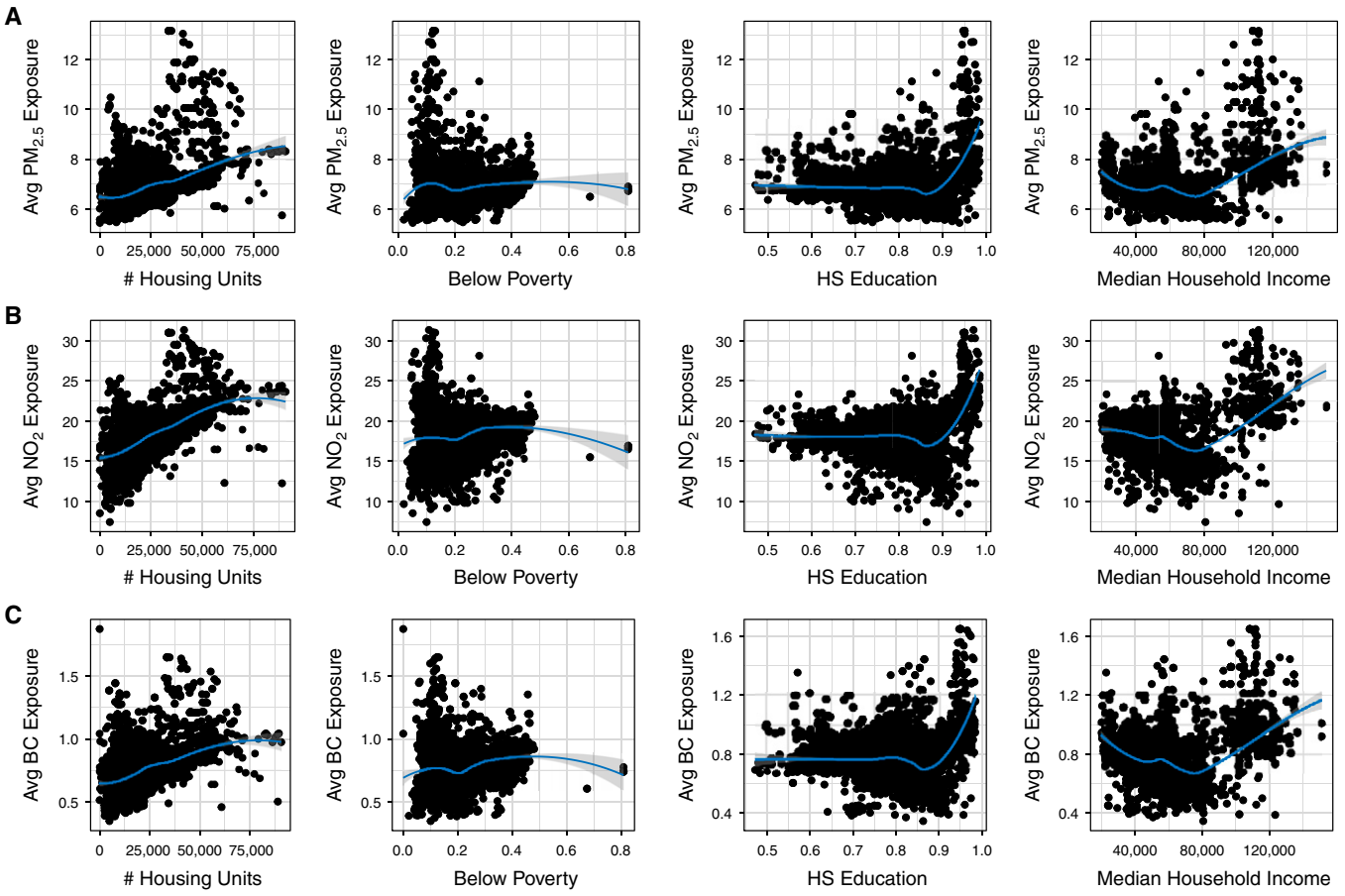

Figure 4. Relationship between the 1-km radial buffer socioeconomic composition indicators and (*A*) fine particulate matter ($PM_{2.5}$), (*B*) NO_2, and (*C*) BC exposure levels. These plots represent the relationship between 1-km radial buffer variables (*x*-axis) and air pollution exposure levels (*y*-axis); locally estimated scatterplot smoothing curves are shown in blue. These data suggest that air pollution exposure levels increase with the number of housing units, do not appear to be related to the proportion of the census block living below the poverty line, increase at a higher proportion of the census block having an age over 25 years and an HS education, and increase at a higher median income.
Avg = average; BC = black carbon; HS = high school; NO_2 = nitrogen dioxide; $PM_{2.5}$ = particulate matter ≤2.5 μm in aerodynamic diameter.

COVID-19 mortality rate (35). More recently, an analysis leveraging the Department of Veterans Affairs COVID-19 Data Resource found that a 1.9-μg/m^3 increase in the long-term $PM_{2.5}$ exposure level was associated with a 10% increased risk of hospitalization (36).

Although our analyses suggest a positive association between BC exposure and COVID-19 mortality and ICU admission, these results did not reach statistical significance. Furthermore, NO_2 exposure did not appear to be associated with COVID-19 outcomes (mortality: RR, 1.00 [95% CI, 0.98–1.03]). These results therefore are in contrast to those of a previously published county-level report from 3,076 counties, which found that a 4.6-ppb increase in the NO_2 exposure level was associated with an 11% increase in the COVID-19 case-fatality rate and a 16% increase in the COVID-19 mortality rate (37). Of note, these authors did not find an association between $PM_{2.5}$ exposure and COVID-19 case-fatality or mortality rates.

The effect of air pollution independent of other socioeconomic factors in modifying the association with COVID-19 outcomes may be difficult to isolate. Air pollution exposure levels differ across socioeconomic factors. Lower-income communities may have poorer access to health care and healthy food options, may live in crowded housing, and may be more likely to be frontline, essential workers. Indeed, unadjusted analyses examining the associations between air pollution and the risk of mortality and intubation suggest an inverse relationship, with higher exposure levels being associated with a lower risk of mortality and intubation. To account for potential confounding, our analyses adjusted for race or ethnicity and individual-level indicators of socioeconomic status such as the insurance vendor and hospital of presentation, which we show are associated with the 1-km radial buffer indicators of socioeconomic composition, including poverty, education, and housing density. We also generated propensity scores to use as weights in adjusted analyses to account for potential selection bias of NYC residents who were able to leave NYC during the surge or who may have selected other hospital systems.

Our data suggest that in NYC, higher-income communities are more exposed to air pollutants. In NYC, the local sources of $PM_{2.5}$, BC, and NO_2 are largely combustion-related and from roadways, restaurants, and space heating. We note that in NYC, the wealthiest communities with the highest population density are in Manhattan, with densely packed streets, buildings, and

Table 2. Associations between Air Pollution Exposure Levels, Considered Separately, and COVID-19 Outcomes among Hospitalized Patients: Poisson Regression with Robust Error Variance

	Unadjusted Model (*N* = 6,542)			Adjusted Model* (*N* = 6,306)		
Outcome	**RR**	**95% CI**	***P* Value**	**RR**	**95% CI**	***P* Value**
Mortality						
$PM_{2.5}$	0.94	0.89–0.98	0.01	1.11	1.02–1.21	0.02
BC	0.57	0.44–0.72	<0.01	1.19	0.75–1.88	0.46
NO_2	0.97	0.96–0.98	<0.01	1.00	0.98–1.03	0.76
ICU admission						
$PM_{2.5}$	1.07	1.02–1.13	0.01	1.13	1.00–1.28	0.045
BC	1.49	1.11–2.01	0.01	1.07	0.55–2.07	0.83
NO_2	1.02	1.01–1.04	0.01	1.01	0.98–1.05	0.44
Intubation and mechanical ventilation						
$PM_{2.5}$	0.92	0.86–0.99	0.02	1.05	0.91–1.20	0.52
BC	0.61	0.43–0.87	0.01	0.91	0.43–1.92	0.80
NO_2	0.97	0.95–0.99	<0.01	1.00	0.96–1.04	0.98

Definition of abbreviations: BC = black carbon; CI = confidence interval; COVID-19 = coronavirus disease; NO_2 = nitrogen dioxide; $PM_{2.5}$ = particulate matter ≤2.5 μm in aerodynamic diameter; RR = risk ratio.
RRs are per 1-$\mu g/m^3$ increase in $PM_{2.5}$ or BC or per 1-ppb increase in NO_2.
*Adjusted models include propensity score weighting and were adjusted for age, sex, race or ethnicity, hospital of presentation, time since pandemic onset, and insurance.

restaurants likely contributing to higher annual average air pollution exposure levels. That NYC higher-income communities are more exposed has been replicated elsewhere (38) and is in contrast to prior work in different parts of the United States finding that poorer communities that have faced systemic racism and economic and political marginalization have higher air pollution exposure (39).

Our exploratory analyses suggest that as compared with patients self-identifying as White, patients self-identifying as Hispanic and other, and especially those under 65 years of age, were particularly susceptible to the effects of long-term $PM_{2.5}$ exposure on COVID-19 outcomes, including mortality. Importantly, the distributions of $PM_{2.5}$ exposure levels for White and Hispanic patients were similar (Hispanic: median, 6.98 $\mu g/m^3$ [IQR, 6.77–7.24 $\mu g/m^3$]; White:

Table 3. Associations between Long-Term Exposure to Particulate Matter ≤2.5 μm in Aerodynamic Diameter and COVID-19 Mortality by Race or Ethnicity

	Black		Hispanic		Asian		Other		White	
Outcome	**RR**	**95% CI**	**RR**	**95% CI**	**RR**	**95% CI**	**RR**	**95% CI**	**RR**	**95% CI**
Mortality										
All patients	1.08	0.87–1.33	1.22	1.02–1.46	0.92	0.66–1.27	1.20	1.02–1.41	0.99	0.83–1.18
Stratified by age										
>65 yr	1.04	0.81–1.32	1.09	0.87–1.36	0.98	0.68–1.41	1.16	0.96–1.40	0.97	0.80–1.19
≤65 yr	1.18	0.78–1.80	1.44	1.07–1.93	0.60	0.28–1.25	1.17	0.86–1.59	1.09	0.68–1.75
ICU admission										
All patients	1.10	0.81–1.50	1.26	1.02–1.56	0.83	0.47–1.48	1.14	0.90–1.45	1.01	0.76–1.35
Stratified by age										
>65 yr	1.27	0.85–1.92	1.02	0.71–1.48	1.22	0.50–2.94	0.85	0.57–1.27	1.00	0.67–1.47
≤65 yr	0.97	0.62–1.51	1.43	1.12–1.83	0.60	0.30–1.17	1.43	1.07–1.90	0.99	0.62–1.58
Intubation										
All patients	0.83	0.57–1.19	1.37	1.08–1.73	0.70	0.41–1.21	1.15	0.87–1.51	0.88	0.61–1.25
Stratified by age										
>65 yr	0.74	0.44–1.25	1.19	0.80–1.78	0.79	0.37–1.69	1.07	0.70–1.65	0.73	0.46–1.15
≤65 yr	0.91	0.54–1.53	1.54	1.16–2.04	0.56	0.27–1.17	1.21	0.85–1.73	1.01	0.54–1.89

Definition of abbreviations: CI = confidence interval; COVID-19 = coronavirus disease; *P*-int = *P* for interaction; $PM_{2.5}$ = particulate matter ≤2.5 μm in aerodynamic diameter; ref = reference; RR = risk ratio.
Poisson regression with robust error variance models was used. RRs are per each 1-$\mu g/m^3$ increase in fine particulate matter ($PM_{2.5}$). "All patients" models are stratified by race or ethnicity, include propensity score weighting, and were adjusted for age, sex, hospital of presentation, time since pandemic onset, and insurance. "Stratified by age" models are further stratified by age dichotomized around the study median of 65 years, include propensity score weighting, and were adjusted for sex, hospital of presentation, time since pandemic onset, and insurance. In unstratified models with interaction terms, all *P*-int terms were >0.10, except for the mortality *P*-int of $PM_{2.5}$ × race or ethnicity (ref: White) for all patients (Black *P*-int = 0.08; other *P*-int = 0.04) and the dichotomous *P*-int of $PM_{2.5}$ × race or ethnicity (ref: White) × age (Hispanic *P*-int = 0.09).

Table 4. Associations between Long-Term Fine Particulate Matter ($PM_{2.5}$) Exposure Levels below Different Thresholds and COVID-19 Mortality, ICU Admission, and Intubation

Outcome	RR	95% CI	*P* Value	*N*
Mortality				
⩽12 μg/m^3	1.12	1.02–1.22	0.02	6,292
⩽10 μg/m^3	1.15	1.04–1.27	0.01	6,197
⩽7.13 μg/m^3	1.17	1.01–1.36	0.03	4,702
ICU admission				
⩽12 μg/m^3	1.16	1.02–1.31	0.03	6,292
⩽10 μg/m^3	1.20	1.04–1.39	0.02	6,197
⩽7.13 μg/m^3	1.03	0.83–1.28	0.76	4,702
Intubation				
⩽12 μg/m^3	1.07	0.92–1.23	0.39	6,292
⩽10 μg/m^3	1.12	0.95–1.32	0.17	6,197
⩽7.13 μg/m^3	0.93	0.74–1.17	0.53	4,702

Definition of abbreviations: CI = confidence interval; COVID-19 = coronavirus disease; $PM_{2.5}$ = particulate matter ⩽2.5 μm in aerodynamic diameter; RR = risk ratio.
RRs are per each 1-μg/m^3 increase in $PM_{2.5}$. A Poisson regression with propensity score weighting that was adjusted for age, sex, race or ethnicity, hospital of presentation, time since pandemic onset, and insurance was used. Thresholds examined include the U.S. Environmental Protection Agency annual standard of 12 μg/m^3, the previous World Health Organization annual standard of 10 μg/m^3, and the study's third quartile of 7.13 μg/m^3.

median, 6.91 μg/m^3 [IQR, 6.22–7.41 μg/m^3]), suggesting that other factors may play a role in susceptibility to air pollution. Our descriptive results (Figure 3) suggest that communities of color more frequently live in areas with higher levels of poverty and lower educational attainment. Thus, although White and Hispanic patients were similarly exposed in our study, we hypothesize that education, income, and other unmeasured individual- and neighborhood-level factors may thus have protective effects against the demonstrated associations between air pollution and COVID-19 outcomes, including mortality. These findings warrant further investigation.

Although these data do not elucidate the underlying mechanisms by which long-term air pollution exposure places persons infected with SARS-CoV-2 at an increased risk of severe disease and mortality, a robust literature supports associations between long-term $PM_{2.5}$ exposure and poorer health. For example, long-term $PM_{2.5}$ exposure impairs lung function and increases the risk of chronic pulmonary disease, including chronic obstructive pulmonary disease (10). Persons with chronic pulmonary disease may also be at an increased risk of severe COVID-19 (40). Long-term $PM_{2.5}$ exposure may increase the severity of pulmonary infections, including viral infections such as influenza and severe acute respiratory syndrome (41–43). $PM_{2.5}$ exposure may even increase the risk of acute respiratory distress syndrome, a pattern of severe hypoxemic respiratory failure seen in end-stage COVID-19 (13). Long-term $PM_{2.5}$ exposure is also associated with an increased risk of metabolic syndrome and cardiovascular disease, which are both known risk factors for severe COVID-19 (44). Mechanisms central to the pathophysiology of SARS-CoV-2 infection and air pollution overlap and involve a cascade of events that include disrupted immune, neuroendocrine, and autonomic function as well as oxidative stress (45). Indeed, the pathogenesis of COVID-19 appears to be dominated by a hyperinflammatory and hypercoagulable phenotype (46). In summary, long-term air pollution exposure may result in poorer health with more comorbid disease at baseline and may directly and indirectly increase the risk of hypoxemic respiratory failure and death once a person is infected with SARS-CoV-2.

Air pollution exposure levels within the cohort are largely below U.S. EPA regulatory annual guidelines (median $PM_{2.5}$, 6.9 μg/m^3 [IQR, 6.4–7.1 μg/m^3]; n = 6,528; 99.8% below the U.S. EPA annual national ambient air quality standard of 12 μg/m^3) and suggest that the negative impacts of air pollution on COVID-19 mortality exist even at low exposure levels. Analyses excluding patients with average $PM_{2.5}$ exposure levels above U.S. EPA standards, previous WHO annual standards, and the third quartile of exposure within the cohort suggest that those at higher exposure levels do not drive the observed associations. Rather, these analyses suggest that associations persist and are consistent among those with lower $PM_{2.5}$ exposure levels. These data are thus in line with prior literature suggesting that there is no safe threshold of air pollution exposure and that it is important to consider the impact of pollution exposure at any level on health, specifically on mortality risk (47). Importantly, air pollution is a modifiable risk factor. These data suggest that state, national, and local air pollution regulation to lower long-term exposure levels should be considered a critical public health measure to reduce infectious disease mortality, particularly in the current, and in any future, pandemic (48). Public health policies to reduce air pollution exposure may be most effective if prioritized in communities disproportionately susceptible to the deleterious effects of air pollution. It is critical to note that these data, in conjunction with the aforementioned ecologic data, come at a time when the U.S. EPA has announced its intention to reconsider the particulate matter national ambient air quality standard (49).

The mortality rate of the overall cohort is high and may reflect the lack of therapeutics early in the pandemic and hospital system factors associated with operational loads, among other factors, which have been reported elsewhere (50–52). Strengths of our study include a large data set of complete, individual-level COVID-19 hospital admissions encompassing all PCR-positive admissions from the NYC spring 2020 surge. Our COVID-19 definition is robust, as it includes only patients with positive SARS-CoV-2 PCR results. We include patients from both private and public hospital systems and across different NYC boroughs, thereby capturing patients across a range of socioeconomic backgrounds. Our patient population is racially and ethnically diverse, with over 50% of the population self-identifying as Hispanic (26%), Black (25%), or Asian (7%) and only 17% self-identifying as White. By leveraging patient-level residential locations, we were able to merge these clinical data with robust measures of long-term air pollution exposure and 1-km radial buffer socioeconomic indicators. Our analyses therefore account for the individual-level socioeconomic composition that may confound the

association between long-term air pollution exposure and COVID-19 outcomes among hospitalized patients.

We also note limitations. Our study conditions on COVID-19 hospitalization and therefore does not examine how long-term air pollution exposure modifies the COVID-19 clinical course in all affected persons. There were times, especially early in the pandemic, in which PCR capacity was suboptimal and patients were hospitalized with clinically suspected COVID-19 that was not confirmed through PCR testing. These patients were excluded from the present analyses. However, given that this was an issue with testing capacity seen at all hospitals, we hypothesize that patients with non–PCR-confirmed COVID-19 were not differentially exposed to air pollution, as compared with those patients who underwent PCR testing. Although we assessed intubation and ICU admission, we also note that a number of patients with COVID-19 received high-level supplemental oxygen (e.g., high-flow nasal cannula, nonrebreathing masks, or both) in a non-ICU setting, and examining intubation and ICU admission alone therefore does not fully capture all patients who were critically ill with acute hypoxemic respiratory failure. We assigned air pollution exposure levels on the basis of the residential address at the time of admission, and it is plausible that misclassification bias occurred. Furthermore, this strategy makes it difficult to examine longer-term exposure, given the higher likelihood that patients may not have lived at a residential address over longer periods of time. Higher measurement error in the NYCCAS model BC and NO_2 predictions, as compared with the $PM_{2.5}$ predictions, may have reduced our ability to detect associations between BC and NO_2 concentrations and COVID-19 outcomes. Although we adjusted for multiple variables meant to capture socioeconomic indicators, we cannot rule out residual confounding. We also note that electronic health records have inherent limitations, including misrecorded and missing data. Finally, we note that we could not account for clustering by the source of an outbreak (e.g., congregate care settings, places of employment), as these data were not available and as the time period of the study was at a moment of widespread community-level transmission.

In summary, we find that among patients hospitalized with COVID-19, higher long-term $PM_{2.5}$ exposure levels, even at levels below current regulatory thresholds, are associated with an increased risk of COVID-19 mortality and ICU admission. These findings suggest that environmental regulations to further reduce air pollution levels are critically important as governments begin to consider policies to build back the economy. ■

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