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Title: Long-Term Fine Particulate Matter Concentrations and SARS-CoV-2 Prevalence:

Differential Relationships by Socioeconomic Status Among Pregnant Individuals in New York City

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Abbreviations: Coronavirus disease 19 (COVID-19); Electronic health record (EHR); fine particulate matter (PM_{2.5}); New York City Community Air Survey (NYCCAS); Odds ratio (OR); Polymerase-chain-reaction (PCR); Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2); Socioeconomic status (SES)

ABSTRACT

We aimed to determine if long-term fine particulate matter (PM_{2.5}) concentrations are associated with increased risk of testing positive for COVID-19 among pregnant individuals who were universally screened at delivery and if socioeconomic status (SES) modifies this relationship. We used obstetric data from Columbia University Irving Medical Center in New York City from March–December 2020, which included Medicaid use (low-SES surrogate) and coronavirus disease 2019 (COVID-19) test results. We linked 300m resolution estimated 2018-2019 PM_{2.5} concentrations and census tract-level population density, household size and income, and

mobility estimates. Analyses included 3318 individuals; 5% tested positive for COVID-19 at delivery, 8% tested positive during pregnancy, 48% used Medicaid, and average long-term PM_{2.5} concentrations were 7.4 µg/m³ (SD = 0.8). In adjusted multilevel logistic regression models, we saw no association between PM_{2.5} and ever-testing positive for COVID-19; however, odds were elevated among those using Medicaid (odds ratio = 1.6, 95% CI 1.0, 2.5 per 1-µg/m³ increase). Further, while only 22% of those testing positive showed symptoms, 69% of symptomatic individuals used Medicaid. SES, including unmeasured occupational exposures or increased susceptibility to the virus due to co-social and environmental exposures, may explain the increased odds of testing positive for COVID-19 confined to vulnerable pregnant individuals using Medicaid.

Elevated levels of long-term ambient air pollution may increase vulnerability to infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) by reducing immune system function and contributing to comorbidities such as diabetes and atherosclerosis (1-3). Most prior studies assessing this relation focused on mortality (4-8). A 2021 review summarized 10 studies from 7 countries and reported a consistent positive association between long-term fine particulate matter (PM_{2.5}) concentrations and coronavirus disease 2019 (COVID-19) incidence. These and several additional studies used ecologic study designs and relied on limited and selective COVID-19 testing (4, 5, 9-14), which might bias results (3). The testing paradigm in the U.S. has resulted in a low ratio of identified cases to all infections in the population and precluded accurate estimation of the denominator of infected people to accurately calculate infection fatality rates and prevalence (15).

A prior study evaluated the relation between air pollution and individual-level COVID-19 infection. This Spanish study leveraged enrolled participants (mostly aged 40-65 years), linked address-level long-term air pollution exposures, and completed COVID-19 serology testing (16). Despite many strengths, the study had a 62% participation rate. Universal COVID-19 screening could reduce bias. Kogevinas et al. found no relationship between four air pollutants (PM_{2.5}, black carbon, nitrogen dioxide, and ozone) and positive COVID-19 serology but did observe an association between PM_{2.5} and COVID-19 disease (i.e., hospitalizations, self-reported symptoms, and positive tests), which was stronger among individuals with lower educational attainment.

Health and environmental exposures tend to follow a social gradient (17). Individuals exposed to higher ambient air pollution levels are also poorer and sicker at baseline and thus more susceptible to COVID-19 infection. This renders previous analyses vulnerable to the ecologic fallacy, where observations made at the area-level may not apply to the individual-level (18), and suggests that lower socioeconomic status (SES) individuals may be at increased COVID-19 risk in part due to environmental exposures (19, 20). In New York City, lower SES has been associated with higher COVID-19 test positivity rate at the ZIP code level (21, 22).

Because pregnant individuals with SARS-CoV-2 infection experience greater respiratory morbidity and risk of mortality than non-pregnant individuals (23, 24), as well as elevated risk of adverse pregnancy outcomes compared to their COVID-19 negative counterparts (23, 25-27), it is of particular interest to identify modifiable risk factors for COVID-19 in this vulnerable sub-population. Ours is the first study to investigate the relationship between PM_{2.5} exposure and testing positive for COVID-19 during pregnancy. We used comprehensive screening data from pregnant New York City residents, all of whom underwent universal SARS-CoV-2

nasopharyngeal quantitative polymerase-chain-reaction (PCR) testing at delivery to overcome the challenges of previous research designs. We stratified by Medicaid status to assess effect modification by SES.

METHODS

Our study aimed to answer the question: do higher long-term levels of air pollution increase risk of testing positive for COVID-19? To answer this question, it is necessary to control for non-causal pathways linking air pollution to COVID-19. In particular, an apparent association could occur if low-SES individuals are exposed to higher air pollution levels and are less able to isolate, leading to increased COVID-19 exposure. Previous studies have not attempted to control for the mobility pathway (which is tightly tied to SES); we used available census tract-level mobility data but acknowledge that individual-level mobility, occupational, or other exposure data would further reduce bias due to this spurious pathway.

We conducted a retrospective cohort study of New York City residents who delivered at NewYork Presbyterian/Columbia University Irving Medical Center between March and December 2020. Electronic health record (EHR) data provided information on delivery date, maternal race/ethnicity (an indicator of individual-level SES), age, Medicaid use (a low-SES surrogate) (28), and residential address, which we used to link air pollution and census tract-level exposures.

COVID-19 outcomes

On March 13, 2020, Columbia initiated universal SARS-CoV-2 testing for all pregnant individuals admitted to labor and delivery units (29). EHR data provided any additional SARS-

CoV-2 PCR results during pregnancy. We created two outcomes: positive COVID-19 PCR at delivery and ever-positive COVID-19 PCR during pregnancy (including delivery).

Air pollution exposure

Analyses used average 2018-2019 annual PM_{2.5} concentrations from the New York City Community Air Survey (NYCCAS). These prediction models leverage measured concentrations from one of the densest monitoring networks worldwide (60–150 monitors depending on year) that are placed for two-week periods around New York City and fuse these with geographic variables of pollutant-specific emission sources to produce predictions at a 300m² grid resolution (**Figure 1**) (30). Specifically, annual PM_{2.5} concentrations were modeled in a land-use regression model that included the following predictors: traffic density, ship traffic, commercial cooking, industrial structures, construction and demolition sites, and boilers burning residual oil. The model had excellent predictive accuracy, with a cross-validated R² of 0.83 (30). We linked the average of 2018 and 2019 annual average PM_{2.5} concentrations from the 300m² grid to the latitude/longitude coordinate of patients' residential address at the time of delivery. The 2018–2019 average concentration can be representative of longer-term PM_{2.5} exposure.

Spatial covariates

We geocoded residential addresses to the census tract and linked data from the 2015-2019 American Community Survey on median household income and average household size (31). These factors could potentially confound the relationship between long-term air pollution levels and risk of testing positive for COVID-19. Further, because mobility could increase risk of exposure to SARS-CoV-2, we linked data from SafeGraph (Denver, Colorado) (21), a company

that aggregates anonymized location data from numerous applications to provide insights about physical places, via the Placekey Community. To enhance privacy, SafeGraph excludes census block group information if fewer than five devices visited an establishment in a month from a given census block group. We used SafeGraph data provided at the weekly census tract-level, and all census tracts where study participants resided had non-missing data. We estimated the percent change in mobility in each person's census tract in the 1-week lagged month (e.g., if delivery date = December 31, 2020, we averaged mobility from November 29 to December 24, 2020) prior to their delivery date versus the six months preceding the pandemic (i.e., September 2019–February 2020). Prior studies have used these mobility data to model COVID-19 transmission dynamics (32, 33).

The original sample consisted of 3887 individuals, and we excluded those whose address did not geocode (n=41 [1%]); lived outside New York City and thus lacked air quality estimates (n=449 [12%]); or were missing Medicaid (n=46 [1%]), census tract-level income (n=15 [<1%]), or household size (n=18 [<1%]) data.

Statistical analyses

We conducted adjusted multilevel logistic regression with random intercepts for census tracts to estimate the association between long-term exposure to PM_{2.5} and odds of testing positive for COVID-19 at delivery or ever during pregnancy. We used penalized splines to test for a departure from linearity in the PM_{2.5}-COVID-19 association and the generalized cross-validation (GCV) criterion to select optimal fit for the penalized spline. A linear term for PM_{2.5} was the best fit. Models controlled for age (natural cubic spline, 3 degrees of freedom), race/ethnicity (Hispanic, non-Hispanic White, non-Hispanic Black, other/missing), delivery month (natural

cubic spline, 4 degrees of freedom), and census tract-level population density (continuous), median household income (continuous), average household size (continuous), and pre-delivery mobility (continuous). To assess differential associations by individual-level SES, we stratified our main models by use of Medicaid as insurance and Cochran's Q test for equivalent treatment effects across Medicaid status (34). In an alternative assessment of effect modification, we added a product term between Medicaid status and long-term PM_{2.5} concentration to the main models. We expected this approach to have higher power (under correct specification) than the stratified analysis, but to be less robust because it assumes the same confounding structure in both Medicaid and non-Medicaid strata. Statistical significance for effect modification was assessed at $\alpha = 0.05$. We evaluated model residuals for spatial autocorrelation visually and using Moran's I (35). All analyses were conducted using R version 3.6.3.

RESULTS

Our analytic sample included 3318 individuals who lived in 702 New York City census tracts (**Web Figure 1**). Five percent tested positive for COVID-19 at delivery, 8% tested positive for COVID-19 at some point, i.e., ever, during pregnancy, and 48% used Medicaid. Average long-term PM_{2.5} concentrations were similar in Medicaid and non-Medicaid users (**Table 1**), with an overall average of 7.4 $\mu\text{g}/\text{m}^3$ (SD=0.8). While overall changes in mobility were pronounced (-54% in the 1-5 weeks prior to delivery compared to pre-pandemic levels), we saw limited difference by Medicaid use status. However, testing positive for COVID-19 at delivery or ever during pregnancy was more common among those who used Medicaid vs. not (7.2% vs. 2.9% and 10.8% vs. 5.1%, respectively). Of the 261 individuals who ever tested positive, the largest racial/ethnic group was Hispanic (70%) followed by non-Hispanic Black and White (both 10%).

Among the 58 (22%) symptomatic individuals who tested positive, 69% used Medicaid. Those who tested positive versus negative for COVID-19 at delivery had similar levels of long-term PM_{2.5} exposure (~7.5 µg/m³), census tract-level average household size (~2.7 individuals), and population density (~35,000/km²) but lower census tract-level median household income (\$46,000 versus \$66,000) (**Web Figure 2**). Parts of Manhattan and Brooklyn had the highest long-term levels of PM_{2.5}; however, lower household income did not track with these high PM_{2.5} concentrations (**Figure 1**).

Overall, in adjusted models, we did not observe an association between PM_{2.5} and COVID-19 at delivery or ever during pregnancy (**Figure 2, Web Table 1**); however, in the analysis stratified Medicaid status odds were elevated among those using Medicaid as insurance (odds ratio [OR] = 1.6, 95% CI: 0.9, 2.7 at delivery and OR = 1.6, 95% CI: 1.0, 2.5 ever positive, per 1-µg/m³ increase in PM_{2.5}). Among those not using Medicaid as insurance, no association was observed with testing positive for COVID-19 at delivery (OR = 0.6, 95% CI: 0.3, 1.2) or ever during pregnancy (OR = 0.8, 95% CI: 0.5, 1.3). The product-term between PM_{2.5} concentration and Medicaid as insurance on odds of testing positive for COVID-19 at delivery or ever during pregnancy was significant (*p*-value = 0.02 and 0.004, respectively). The Cochran's Q test did not reach statistical significance in its test for heterogeneity of effect by Medicaid use status (*p*-value = 0.07 and 0.30, respectively). We observed no residual spatial autocorrelation either upon visual inspection or using Moran's I (**Web Figure 3**).

DISCUSSION

In New York City during 2020, we found an association between exposure to higher levels of long-term PM_{2.5} and odds of testing positive for COVID-19 during pregnancy or at delivery only

among those using Medicaid as insurance, a surrogate for low individual-level SES. While only 22% of those that tested positive showed symptoms, 69% of symptomatic individuals used Medicaid, further highlighting this group for increased attention and intervention due to adverse outcomes related to COVID-19.

Several factors motivated our study of COVID-19 among pregnant individuals in New York City. First, pregnant persons with COVID-19 are more likely to experience significant respiratory morbidity and more likely to die than non-pregnant persons with COVID-19 (23, 24). Second, pregnant persons with COVID-19 are more likely to experience adverse pregnancy outcomes compared to pregnant persons without COVID-19, including preterm delivery, preeclampsia, and possibly stillbirth (23, 25-27). Third, exposure to PM_{2.5} during pregnancy has been shown to increase risk of preterm birth as well as delivery of a low-birth-weight neonate (36). Thus, investigation of modifiable risk factors for COVID-19 infection in this population is of particular importance.

Our findings fit within a broader literature linking air pollution to respiratory infection. Large-scale epidemiologic studies reveal consistent associations between higher long-term PM_{2.5} concentrations and increased risk of acute respiratory infection in the general population (37, 38); risk of infection may increase during pregnancy due to cardiovascular and immune system changes, for example, attenuation of cell-mediated immunity by T1-helper cells as the system moves towards T2-dominance to protect the fetus (39). Pregnant women exposed versus unexposed to unconventional natural gas development were more likely to receive an antibiotic during pregnancy, a proxy for infection (40). Biologically, PM_{2.5} exposure increases C-reactive protein levels in pregnant women, indicating an inflammatory response (41), which may in turn

alter maternal immune system function (42). Further, long-term ambient air pollution exposure has also been linked to altered DNA methylation of sites with functions related to immune function (43). Animal studies of COVID-19 have also indicated particulate matter increases expression of molecules required for SARS-CoV-2 to enter host cells (44).

Many studies have evaluated the association between long-term concentrations of PM_{2.5} and COVID-19 morbidity (45). Nearly all used ecologic designs, which increases the likelihood of confounding and exposure misclassification. Just two studies, to our knowledge, have used address-level patient geocoordinates (16, 46). Kogevinas et al.'s cohort study in Catalonia, Spain found no association between 100m resolution long-term PM_{2.5} concentrations and SARS-CoV-2 infection but did identify an association with COVID-19 disease (defined based on hospital admission, positive test, or ≥ 4 COVID-19 symptoms after contact with a diagnosed case) (16). Bowe et al. used US Department of Veterans Affairs health records for ~170,000 COVID-19 positive veterans and found 2018 PM_{2.5} concentrations associated with increased risk of hospitalization for COVID-19 with effects below the national PM_{2.5} standard of 12 $\mu\text{g}/\text{m}^3$ (46). Our study used residential address at time of delivery in 2020 to assign 2018-2019 average 300m resolution PM_{2.5} exposures in NYC. As in Kogevinas and Bowe, the use of residential addresses eliminated cross-level bias (a result of ecologic designs) (3, 47) and reduced exposure misclassification. We know, however, that person-to-person contact spreads COVID-19 and while we accounted for clustering at the census tract level and adjusted models for population density, mobility, and markers of SES, we could not account for the source of infection. Thus, unmeasured factors—e.g., large gatherings, occupational exposure—that correlate with air pollution and testing positive for COVID-19 could potentially explain our results, although we partially account for such factors by controlling for area-level socioeconomic status and

mobility. One option is to consider outbreaks (those in healthcare institutions, acute and long-term care facilities, and homeless shelters) and sporadic cases of COVID-19 separately, as in Stieb et al. 2021 (11).

Prior air pollution studies have relied on area-level COVID-19 incidence data (45). As discussed, this can lead to ecologic bias but is also based on selective COVID-19 testing. In 2020, only 1 in 4.6 infections were reported in the U.S. (33). This under-reporting may have biased prior studies. For example, increased screening capacity, messaging about testing, or awareness of infection could have accounted for the observed association between elevated long-term air pollution levels and increased risk of COVID-19 in urban areas (47). Because all women in our cohort received a COVID-19 swab at delivery, we circumvent these issues for the COVID-19 at delivery analysis. In New York City, community-level low SES appeared to cluster with low testing rates (48, 49), so basing analyses on available incidence data could have biased results.

We observed an association between long-term $PM_{2.5}$ concentrations and testing positive for COVID-19 only among individuals who used Medicaid as insurance. This differential association was statistically significant as measured by a product term but not using Cochran's Q test, potentially due to limited statistical power in the stratified models. In Catalonia, Spain, Kogevinas et al. 2021 reported an overall association between long-term $PM_{2.5}$ and COVID-19 disease that was stronger among those with less than a university education. Decades of research suggest SES can modify the relationship between air pollution and adverse health outcomes in several ways. Individuals of lower SES may experience harmful co-exposures, energy and food insecurity, psychosocial stress, and preexisting conditions making them more susceptible to the health effects of air pollution (50). Additionally, higher SES may have enhanced ability to self-

isolate (51), masking any biological effect of air pollution on COVID-19 susceptibility. If long-term levels of air pollution do increase the risk of testing positive for COVID-19, longstanding issues of environmental injustice (52, 53) may help partially explain the disproportionate burden of COVID-19 among low SES Americans (54).

Our study had several limitations. Patients in our study underwent nasopharyngeal quantitative PCR testing, which while considered the gold standard is not perfect. Among asymptomatic individuals, simulations suggest this type of testing has a 77% sensitivity (55). Additionally, we did not have information on the timing of COVID-19 tests conducted prior to delivery; we estimated changes in census tract-level mobility for all participants based on timing of delivery. We linked air pollution data based on address at the time of delivery and cannot confirm mothers lived there in 2018-2019 (years of air pollution measures). Furthermore, although the exposure prediction model performed well, some exposure measurement error is expected. There is no reason, however, to believe that the error would be correlated with testing positive for COVID during pregnancy; any non-differential exposure measurement error, thus, would most likely bias our results towards the null (56, 57). Our study focused on the relationship between long-term PM_{2.5} concentrations and COVID-19, but it is possible that short-term exposures also influence risk of testing positive for COVID-19. Future studies should investigate this pathway and consider the short-term reductions in PM_{2.5} levels that occurred during the initial lockdown (58, 59). We lacked data on several potentially important risk factors including occupation, educational attainment, social networks, and comorbidities. However, we did adjust for both individual- and tract-level SES, which may drive most of these factors. Census tract mobility data do not fully characterize individual ability to isolate or risk of exposure to COVID-19. Our study period spans the first three waves of the pandemic, but findings may not translate into later

time periods, including the delta and omicron surges. A strength of our study, however, was that we confined the analysis to dates prior to widespread vaccination, which would have complicated analyses. Further, the study population included only individuals that resided in New York City and delivered a live-born infant at New York Presbyterian/Columbia University Irving Medical Center between March and December 2020. During the first wave, New York City saw a crude fatality rate of 9.2% (60), triple that of the worldwide fatality rate (61). Therefore, our results may not generalize to other time periods or other populations, especially if pregnant Medicaid users in New York City are not exchangeable with those in other locations.

The COVID-19 pandemic has disproportionately affected low-income Americans who face fragmented healthcare, a higher prevalence of pre-existing conditions, and less ability to social distance due to occupational exposures (51). Our results suggest that lower-SES pregnant women may also experience a heightened risk of COVID-19 related to long-term air pollution exposures and point to the need for additional research.

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Table 1: Study population characteristics for pregnant individuals who delivered at Columbia University Irving Medical Center in New York City between March–December 2020, stratified by Medicaid use status

Characteristic	Insurance status (n = 3318)			
	Medicaid (n = 1608)		No Medicaid (n = 1710)	
Maternal characteristics	No.	%	No.	%
Positive COVID-19 test at delivery	116	7.2	49	2.9
Positive COVID-19 test ever during pregnancy	174	10.8	87	5.1
Symptomatic with COVID-19 ever during pregnancy	40	2.5	18	1.1
Age at time of delivery, y, mean (SD)	29.4	6.2	33.9	5.1
Race/ethnicity				
Hispanic	1135	70.6	453	26.5
Non-Hispanic White	54	3.4	526	30.8
Non-Hispanic Black	229	14.2	185	10.8
Other/missing	190	11.8	546	31.9
Fine particulate matter concentration, $\mu\text{g}/\text{m}^3$, mean (SD)	7.3 (0.5)	0.5	7.6	1.0

Census block group characteristics				
Average household size, count, mean (SD)	2.8 (0.6)	0.6	2.5	0.4
Population density, individuals/sq-km ^a	37,900 (18,200)		30,090 (18,200)	
Median household income, \$ ^a	43,421 (20,844)		84,439 (48,391)	
Percent change in mobility, % ^b	-55 (-62, -46)		-57 (-70, -47)	

Abbreviations: COVID-19, coronavirus disease 2019.

^a Values are expressed as mean (standard deviation)

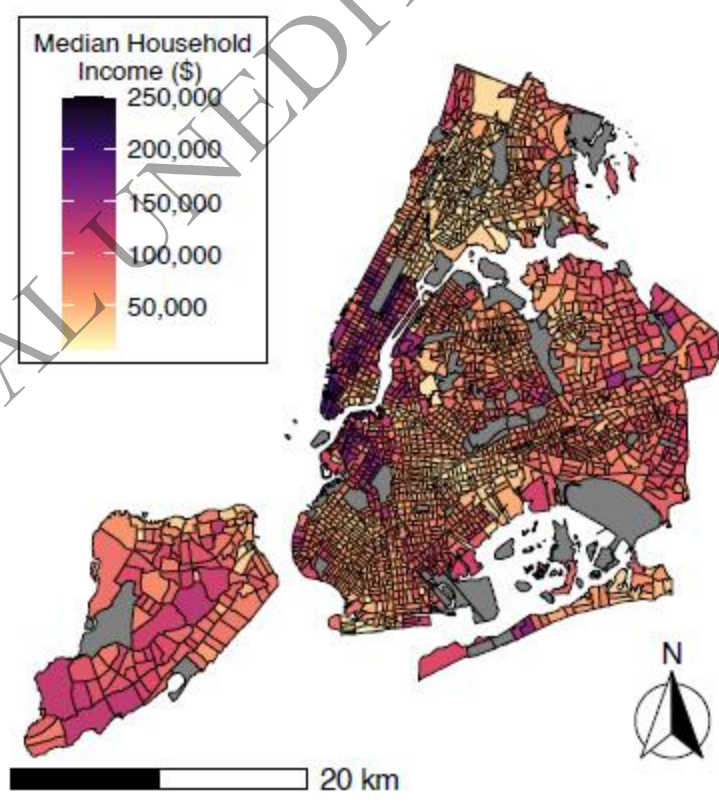
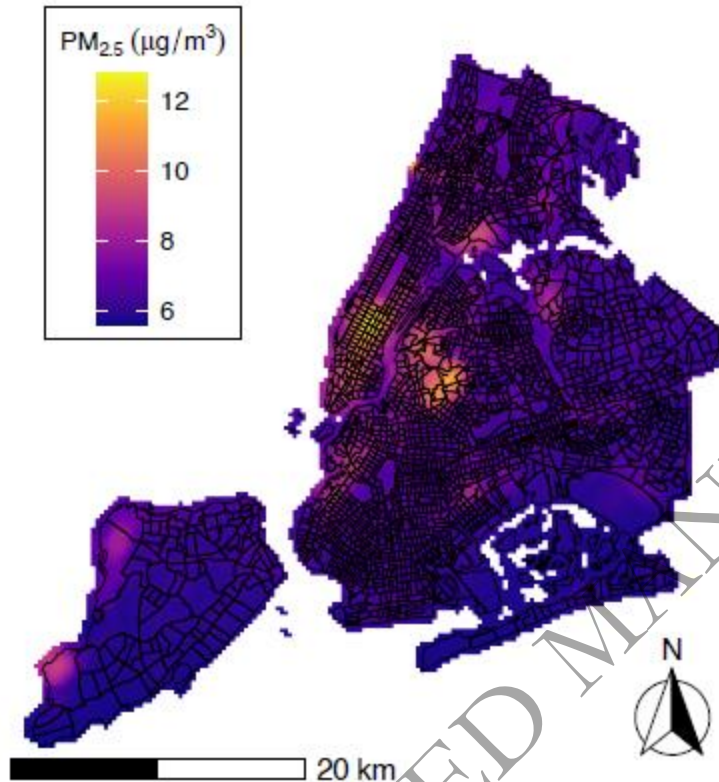
^b Values are expressed as median (interquartile range)

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Figure 1: Distribution of (A) 2018-2019 annual average PM_{2.5} concentrations from the New York City Community Air Survey (NYCCAS) product and (B) Median household income at the census tract level from the 2015-2019 American Community Survey (ACS). Grey indicates missing income data from the ACS.

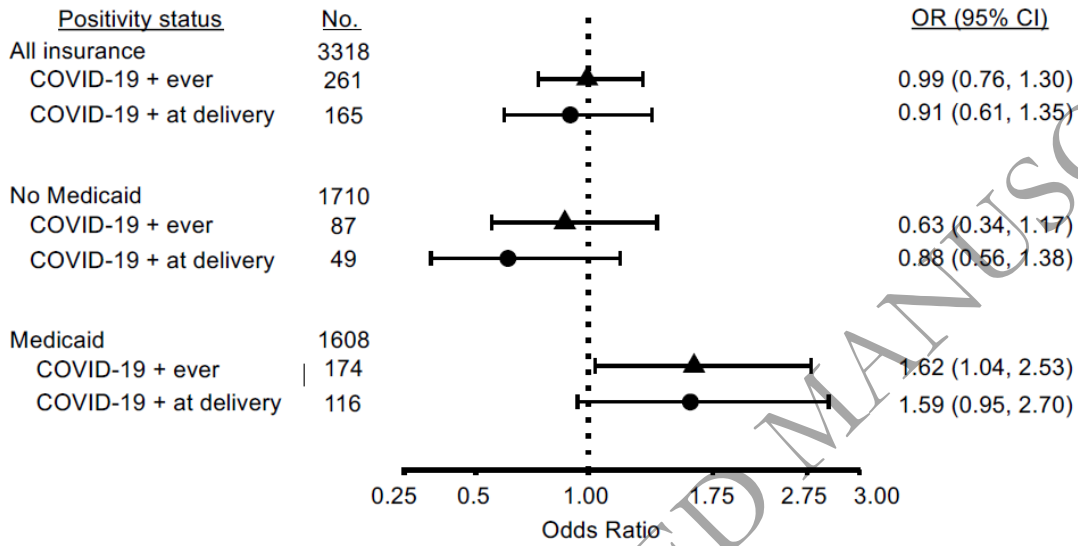
Figure 2: Estimated associations between long-term PM_{2.5} concentrations and odds of testing positive for COVID-19 at delivery or ever during pregnancy (A) overall (n=3318), and in models stratified by (B) those that used Medicaid (n=1608) and (C) those who did not use Medicaid (n=1710) and who delivered at Columbia University Irving Medical Center between March and December 2020. Figures show odds ratios and 95% CIs for a 1- $\mu\text{g}/\text{m}^3$ increase in 2018–2019 PM_{2.5} concentration. All estimates were adjusted for age, race/ethnicity, delivery month, and census tract-level population density, median household income, average household size, and pre-delivery mobility and included a random intercept for census tract. The overall model also controlled for Medicaid use. CI, confidence interval, OR, odds ratio, PM_{2.5}, fine particulate matter.

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