Title: Identifying chronic traffic-related air pollution's causal effects on COVID-19 outcomes: beware variations in defensive behavior

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Abstract: The environmental justice literature has documented the differential ability of community members to adopt defensive behaviors that mitigate threats from environmental hazards. We consider the implications of this fact while exploring the causal relationship between traffic-related air pollutants (TRAP) and the intensity of COVID-19 disease in New York City. Because defensive behaviors are not equally available to all city residents, we use an instrumental variable approach to develop measures of ambient air pollution uncorrelated with demographic characteristics that might confound causal inference. We find that increases in the average chronic concentration of three TRAPs do not have meaningful impacts on COVID-19 death or hospitalization counts in census tracts in New York City. Our findings contrast with the large correlations reported in observational studies, which likely generate biased estimates due to correlation between demographics and ambient air quality and due to systematic measurement error in the rate-based metrics that are commonly used to track public health outbreaks. Our use of cellphone location data from Safegraph showcases how residents from wealthier, whiter parts of the city were able to avoid the risks of COVID-19 by fleeing the city, resulting in systematic measurement error in rate-based outcome variables that confounds results presented in previous studies. Our results highlight the importance of causal analysis in guiding policy and the need to account for defensive behaviors when measuring the impacts of environmental and public health hazards.

Key words: causal identification, COVID-19, defensive behavior, instrumental variables, measurement error, traffic-related air pollution

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I. Introduction

Policymakers and researchers seek to understand the drivers of differential impacts of environmental and public health hazards to provide targeted aid to the most vulnerable populations. In the context of COVID-19 disease, this research is motivated by concerns about new virus variants and potential outbreaks that could threaten public health. COVID-19's impact on respiratory function and previous work on SARS and air-quality conditions (1) has led researchers to explore the relationship between air quality conditions and COVID-19 disease severity. The causal effect of air quality on COVID-19 disease severity is challenging to identify because air pollution is not randomly distributed across geographies, and existing research has shown that adoption of defensive behavior to limit exposure to air pollutants is associated with demographic characteristics that might also affect both biological and social response to COVID-19, including education (2), age, and insurance status (3).

Direct mechanisms to explain how air pollution might influence COVID-19 disease severity include damage to the cilia (4) or overexpression of ACE-2 receptors (5). Potential indirect mechanisms include aggravation of pre-existing cardiovascular and respiratory conditions (4, 6). A survey of existing observational research suggests a correlation between COVID-19 mortality and both acute and chronic ambient concentrations of several compounds, including nitrogen dioxide and particulate matter (7). While acute exposures are sufficient to trigger these events, outcomes become more severe, often fatal, with chronic exposure.

Existing studies have found positive correlations between worse chronic air quality and the severity of COVID-19 disease at the county level in the United States (8) and globally (9). However, these studies are unable to estimate causal impacts---air quality and COVID-19 outcomes might both be highly correlated with unobservable community and demographic characteristics, thus causal identification remains a key challenge. Our work offers unique insights into the challenges of identifying causal links between ambient air quality and health outcomes through 1) the use of hyperlocal air-quality data; 2) census-tract level data on COVID-19 outcomes; 3) econometric techniques to identify the variation in air quality that is independent of unobservable community characteristics; and 4) cellphone mobility data to highlight concerns about systematic measurement error that biases results in rate-based models that depend on administrative data for population estimates.

We use an instrumental variable (IV) approach and detailed air-quality data from New York City (NYC), the one-time global epicenter of the pandemic (10), to overcome this challenge of identifying the causal relationship between chronic air-quality conditions and the intensity of COVID-19 disease. This method compares COVID-19 outcomes in tracts within the same neighborhood and same distance of a highway that differ in the fraction of time spent downwind of the highway. Such tracts should be comparable on both observable and unobservable characteristics, while differing in their ambient air quality, so that we can isolate the effect of pollution on COVID-19 separately from other demographic characteristics. In the first stage of our estimation, we explain the variation in chronic traffic-related air-pollutant (TRAP) concentrations, here defined as a 10-year average preceding the pandemic, that results from being downwind of a highway. We then estimate the effect of this exogenous portion of pollution on COVID-19 outcomes, as measured by mortality and hospitalizations between February 29 and August 30, 2020. This period is prior to vaccine availability, which could confound the relationship, as individuals at high-risk were often prioritized for vaccines. Furthermore, our dense monitoring network and tract-level COVID-19 data disaggregated by age and race represent improvements relative to existing studies that have relied on county-level data.

We find that exogenous variation in the chronic ambient concentrations of our focal pollutants does not cause significant changes in COVID-19 outcomes in census tracts near highways throughout NYC, with precisely estimated null effects for those pollutants most closely associated with traffic (NO and NO_2).

These results are in contrast to the large correlations reported in a number of observational studies that are confounded by correlations between ambient pollutant concentrations and demographic characteristics that also affect health outcomes. We can only replicate these findings of a positive effect when we use a sample in which our instrument fails the exclusion restriction.

II. Methods

Air pollution is correlated with many socioeconomic variables that may influence COVID-19 outcomes. Even a rich set of control variables may result in biased estimates of the effect of air quality, if some of the covariates have multi-directional causality with air quality or if there are interaction effects between variables. To overcome this issue, we use an IV approach that relies on variation in pollution resulting from wind direction relative to nearby highways to identify the causal effects of chronic ambient air pollution on COVID-19 disease intensity. A key assumption underlying this method is that wind direction is exogenous, or uncorrelated, with individual or community characteristics that are correlated with COVID-19 outcomes. In other words, wind direction only effects COVID-19 outcomes through its effect on air quality.

We use the two-stage least squares (2SLS) estimation method. In the first stage, we construct the instrumental variable $\widehat{AQ}$, the exogenous component of air quality (AQ), by modelling AQ as a linear combination of wind-related variables and other exogenous observables. $\widehat{AQ}$ can be considered the "exogenous portion" of air quality. In the second stage, we regress COVID-19 outcomes on $\widehat{AQ}$ and the same set of exogenous observables.

First-stage Regression

We first predict $PM_{2.5}$, NO_2 and NO as linear functions of the percent of time downwind of a highway at each distance:

$$AQ_i = \eta + \beta Downwind_i + \mu HighwayDistance_i + PUMA_i + Station_i + \epsilon_i \quad \text{(Eq. 1)}$$

$Downwind_i$ is the percent of time that census tract I is downwind of any highway segment that is located within 0.5 kilometers (km) across a 10-year period (2009-2018). The model also controls for distance to the closest highway, $HighwayDistance_i$, dummy variables for Public Use Microdata Areas (PUMAs), and dummy variables for the nearest weather station.

The key assumptions necessary for instrumental validity are (1) instruments are correlated with the pollutant concentrations (relevancy), and (2) instruments must not be related to COVID-19 disease intensity except through their relationships with pollutant concentrations (exclusion restriction). The relevancy assumption is met – the downwind variables are significantly associated with increased pollution concentration at distances less than 0.5 km (see table S2). Focusing on the Citywide models, we find that a tract within 0.5 km of a highway and downwind 100% of the time would have increased average ambient concentrations by 0.32 μg/m3 of $PM_{2.5}$, 0.70 ppb of NO_2 and 1.41 ppb of NO, relative to a tract that is downwind 0% of the time. We report the weak instrument Wald F-stats from the first stage and reject the null hypothesis that the instruments are irrelevant.

As for the exclusion restriction assumption, while exposure to poor air quality as a result of living near a highway is likely endogenous with factors related to COVID-19 disease intensity (e.g., income, healthcare access, etc.), we assume that, conditional on living near a highway and in the same neighborhood, our instruments only affect COVID-19 outcomes through their effect on pollution concentration. This assumption is reasonable because the pollutants of interest are generally not

detectable via sight or smell at concentrations in NYC, and differences of the magnitudes our coefficients report would clearly not be detectable.

To further ensure that our instrument for ambient air quality is unrelated to a set of observable characteristics, $\mathbf{X}_i$, that might determine health outcomes, we run placebo regressions of the following form, where x_i denotes each element in the observable characteristics set $\mathbf{X}_i$:

$$x_i = \eta + \beta Downwind_i + \mu HighwayDistance_i + PUMA_i + Station_i + \epsilon_i \quad \text{(Eq. 2)}$$

The results of these regressions are reported in table S3, which presents the p-values for the coefficients of our air quality instrument in regression models for each of the demographic characteristics included. For our sample of interest, the set of census tracts that lie between 0.05 and 0.5 km from the nearest highway, there are no statistically significant correlations in the Citywide sample and only a single marginally significant correlation (at the 10% level) between our instrument and tract population, with more people living in downwind census tracts than in upwind census tracts in the Outer Boroughs sample. This correlation would bias our estimate upward, making our result of a null effect even more robust. These results give us great confidence that our instrument allows us to identify the causal effect of ambient air quality on COVID-19 outcomes in our analysis.

We then test the assumption that populations in our main sample that spend relatively more time downwind of highways are similar to those that spend less time downwind by conducting balance checks comparing variables constructed from ACS data representing tract level socioeconomic and demographic characteristics. A tract is considered "Downwind" if the amount of time the tract spends downwind of a highway is greater than the average amount of time tracts the same distance from a highway spends downwind and is considered "Upwind" otherwise. We show that no normalized differences between Upwind and Downwind tracts are greater than 0.25, supporting our assumption that conditional on living in a tract near a highway, there are not significant socioeconomic or demographic differences between tracts based on the amount of time they spend downwind of highways (table S4).

While there are many reasons for residential sorting to occur in NYC, the above results suggest that it is unlikely that individuals are sorting into tracts based on the amount of time the tract spends downwind of a highway. For this reason, we assume that our instruments are exogenous, and that the exclusion restriction is met.

Second-Stage Regression

Our second stage regressions estimate the causal impact of a change in ambient pollutant concentration on two different measures of the intensity of COVID-19 disease: deaths and hospitalizations. Before detailing the model specification, we first provide motivation for our use of count variables as dependent variables.

Many NYC residents fled the city in the spring of 2020 to avoid exposure to the COVID-19 disease, behavior that was reported to be concentrated in the city's wealthiest neighborhoods (11). We incorporate cell phone mobility data from Safegraph to examine variation in avoidance behavior. Safegraph collects location data from 45 million mobile devices and provides aggregated statistics at the census block group level on the amount of time that mobile devices are in the home, outside of the home, and engaged in work behavior, as well as distance travelled.

Figure 1 shows that while pollutant concentrations are highest in the wealthier, whiter parts of Manhattan (panels A-C), the estimated case rate, defined as the number of positive tests divided by the census tract population, is substantially lower for this part of the city when based on population numbers from

administrative data (panel F). This may be due to increased adoption of defensive behaviors in these areas, as the Safegraph data shows a significant decrease in the number of cell phone devices residing in these census tracts during the first wave of the pandemic, consistent with individuals in these neighborhoods leaving the city to avoid exposure to the disease (panel E).

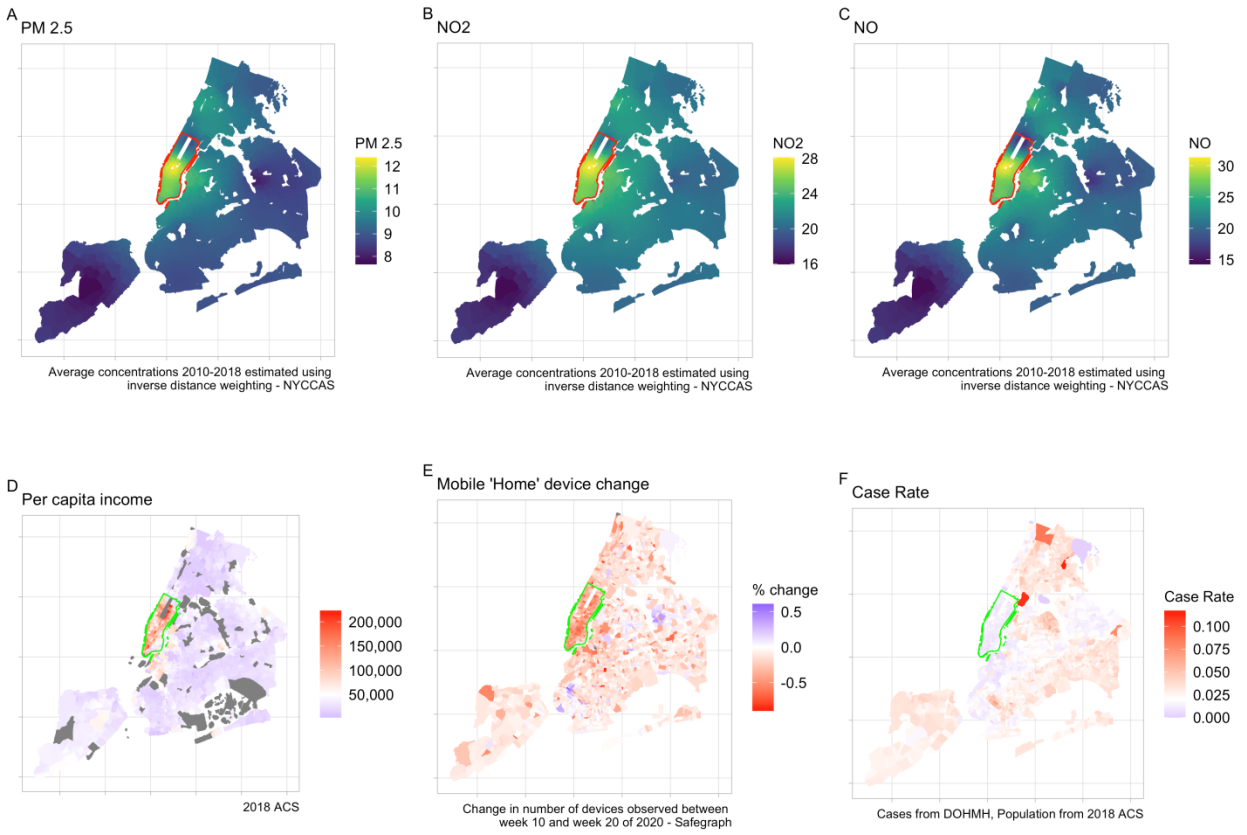

Figure 1. This figure depicts tract-level characteristics relating to chronic concentrations of $PM_{2.5}$ ($\mu g/m^3$) (a), NO_2 (ppb) (b), NO (ppb) (c); 2018 per-capita income (d); the change in mobile devices based in each tract between week 10 and 20 of 2020 (e); and the case rate (positive test results divided by population) as of 08/31/2020 (f).

We incorporate the Safegraph mobility data to adjust the rate-based outcome variables that have been used in previous studies. Figure 2 displays COVID-19 death and hospitalization rates, where these rates are calculated using different population estimates. Panels A and D of figure 2 show these rates using population figures from the 2018 American Community Survey (ACS) in the denominator, and panels B and E use this population adjusted for the number of devices that left the city between March 8 and May 11, 2020 (weeks 10 and 20 of the year), which aligns with the peak of the initial wave of COVID-19 in NYC. Blue points in the scatter plots (panels C and F) show the importance of measurement error in census tract population - addressing this measurement error with cellphone mobility data results in much higher death and hospitalization rates in Manhattan relative to the rest of the city. A rough attempt to account for these departures would effectively double death rates in this part of Manhattan, making them more comparable to those in the rest of the city.

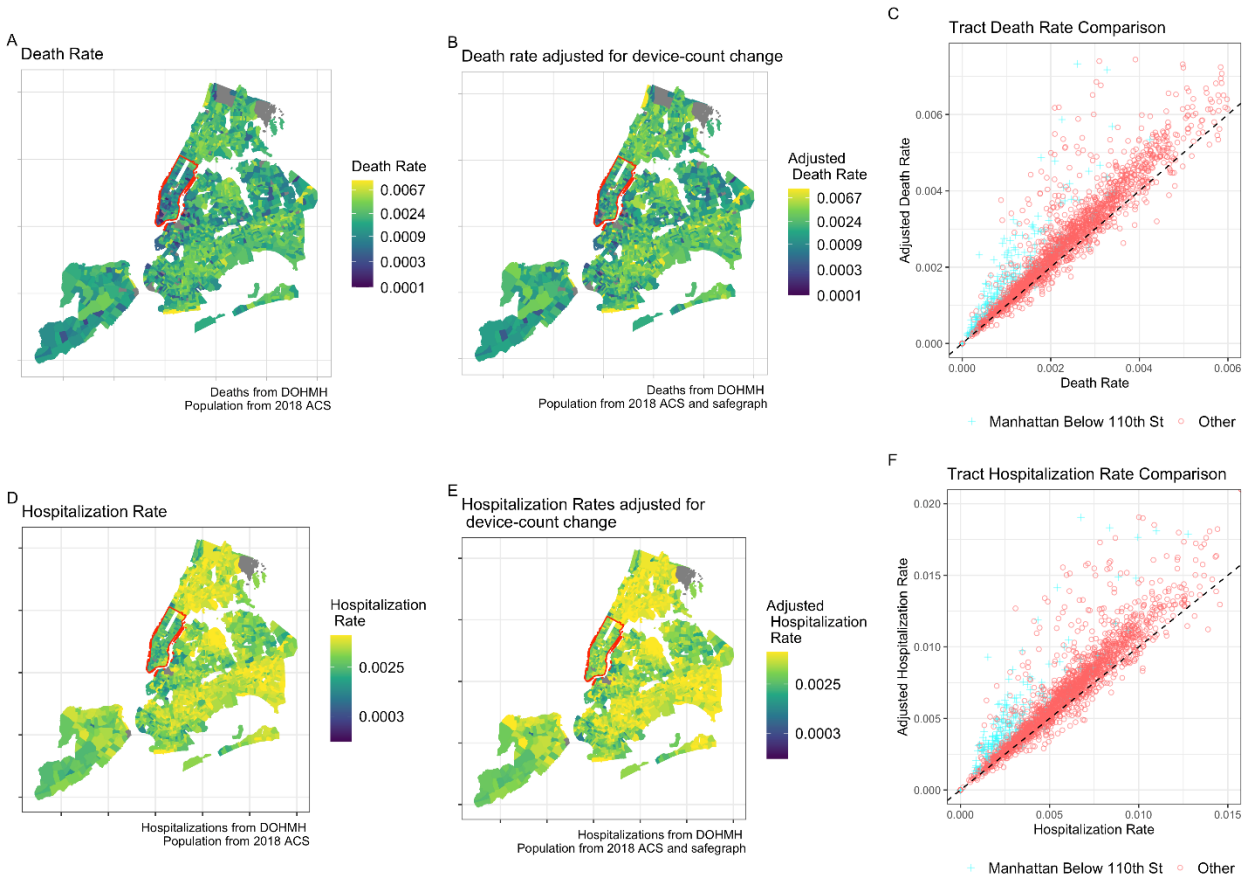

Figure 2. Measures of COVID-19 death and hospitalization rates based on different measures of census tract population. Coarse adjustment of census tract population measures from the 2018 American Community Survey based on observed changes in home cellular device counts from Safegraph leads to adjusted death and hospitalization rates in Manhattan below 110th Street that are more comparable to those in the rest of the city, emphasizing how the inequitable access to defensive behaviors (namely relocating during the initial phase of the COVID-19 pandemic) can explain much of the observed gap in COVID-19 outcomes across different demographic groups in New York City.

For these reasons, we use log-transformed counts of hospitalizations and deaths, which, along with our IV estimation approach, can compare tracts with similarly-sized populations without suffering from systematic measurement error in the dependent variable. Thus, our results can be interpreted as the change in deaths in an average tract, as our instruments are exogenous to tract population. Our identification strategy compares tracts within the same PUMA that lie within the same distance of the highway that spent different amounts of time downwind from 2009-2018. Our key specification is:

$$\log(Y_i) = \alpha_0 + \alpha_1 \widehat{AQ}_\iota + \alpha_2 HighwayDistance_i + PUMA_i + Station_i + \nu_i \quad \text{(Eq. 3)}$$

Y_i are counts of deaths and hospitalizations. We use log-transformed counts and add one to the count variables to adjust for tracts with zero deaths or hospitalizations. $\widehat{AQ}_\iota$ is the instrumented measure of ambient concentration for $PM_{2.5}$, NO_2, and NO predicted from equation (1). We include weather station and PUMA dummy variables in the second stage as well. The key parameter of interest is α_1. Please see

the supplement for more details on our data construction, methods, results and discussions on robustness checks.

Results

We find that being continuously downwind of a nearby highway increases the 10-year average of ambient concentrations of $PM_{2.5}$ by 0.32 $\mu g/m^3$, NO_2 by 0.70 ppb and NO by 1.41 ppb relative to a comparable tract that is never downwind (see table S1). The exogenous portion of pollutant concentrations at each tract are then used in our second-stage regressions to identify the causal impact of increased pollutant concentrations on our outcomes of COVID-19 disease.

Figure 3 presents the causal effects of chronic exposure to three pollutants on COVID-19 deaths (row 1) and hospitalizations (row 2). The figure panels illustrate results for two sets of observations: Citywide and Outer Boroughs, which includes tracts in the Bronx, Brooklyn, Queens, Staten Island, and those tracts above 110th street in Manhattan. For each set of observations, the figure presents the estimated coefficients and 95% confidence intervals for the effect of the focal pollutant on the logged count of our chosen measure of disease intensity. The point estimate of the causal impact of increased chronic pollutant concentration is near zero and statistically insignificant for each of our considered pollutants across our samples, whether we are considering COVID-19 deaths or hospitalizations. We see a much tighter confidence interval for NO_2 and NO, the pollutants that are more directly related to traffic. Our findings are also robust to employment of an IV Poisson model and the addition of demographic control variables. (See tables S5, S6, S12, S13)

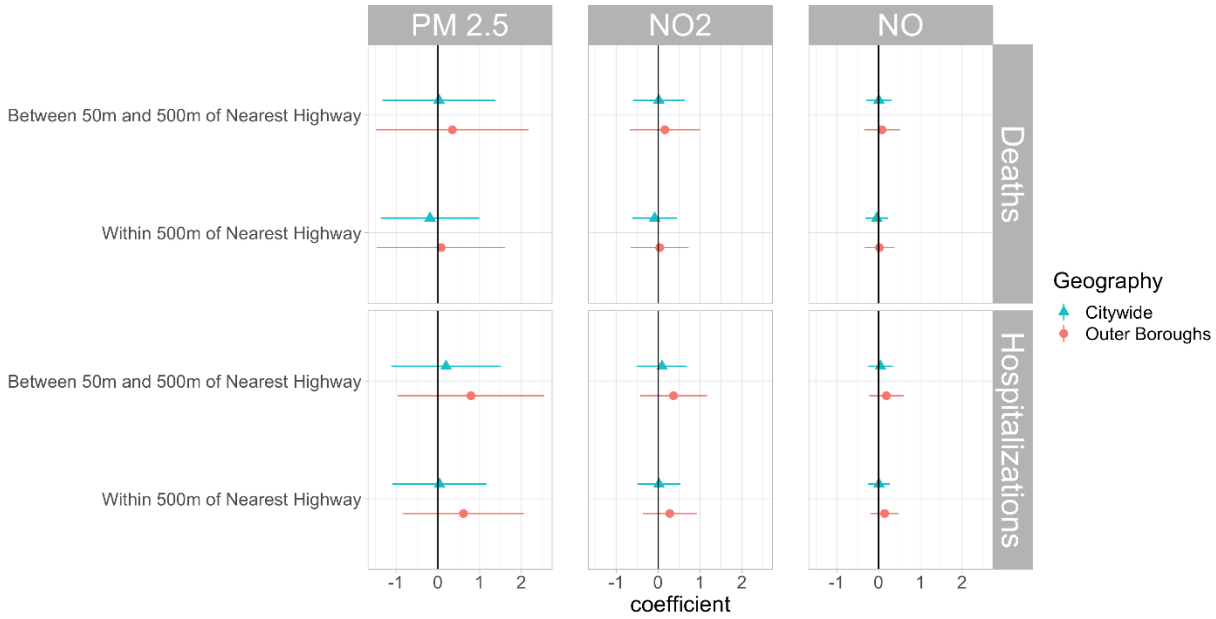

Figure 3. This figure presents the estimated coefficients and associated 95% confidence intervals on our instrumented measure of ambient pollutant concentration from the second stage of our instrumental variable log-linear models across our two geographic samples for all tracts that lie within 500 meters of the nearest highway and the subset of these tracts that are between 50 and 500 meters of the nearest highway.

We also perform quantile IV models to explore how the effects of air quality operate across the distribution of tracts by COVID-19 deaths. Figure 4 shows the quantile regression estimates for our Citywide sample, which are also consistently near zero across tracts based on their position in the distribution of COVID-19 deaths.

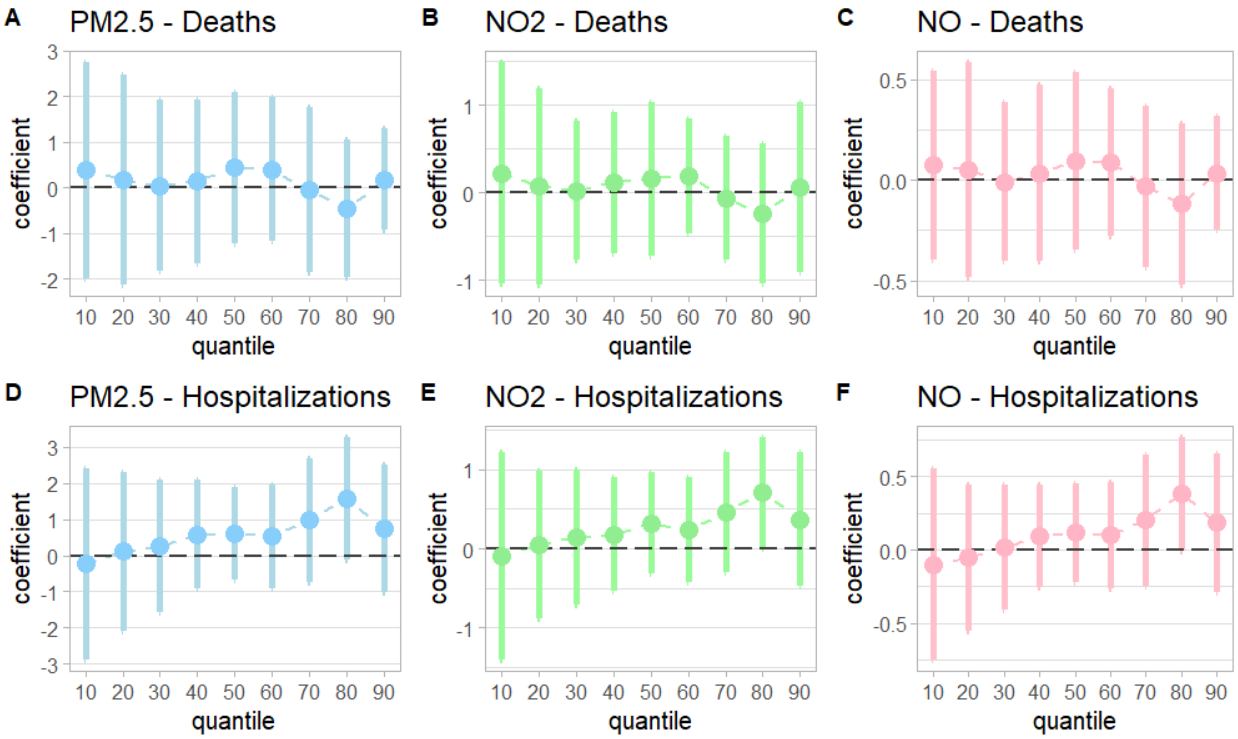

Figure 4. Quantile regression results for our focal COVID-19 outcomes in the sample of New York City census tracts that lies between 50 meters and 500 meters from the nearest highway.

While the point estimates are still statistically insignificant, we see evidence that the effect on COVID-19 hospitalizations is more pronounced in the tracts with a higher number of hospitalizations, which are located in the Washington Heights neighborhood, with three times the recorded population of an average tract and about 80% of the population being Hispanic. This finding is consistent with those from our stratified samples: when we stratify our samples by 24 age and race groups (see table S10), we observe a significant increase in COVID-19 hospitalizations among Hispanics who are at least 65 years old, driven by those tracts with large numbers of Hispanic residents. For all other 23 stratified demographic groups, we find no effect of air quality on COVID-19 deaths or hospitalizations. These results may occur by chance due to the large number of subgroups analyzed, however, they warrant further investigation.

Figure 5 presents coefficient estimates for various models exploring the impact of increased ambient NO concentration on COVID-19 deaths and hospitalizations. The left panels are estimated with ordinary least squares (OLS) models of rate-based outcomes and the right panels are estimated with count-based IV regressions, our preferred models. With an OLS model that controls for a representative set of demographic variables, we find that increasing NO concentration by 1 ppb leads to an imprecisely estimated additional 8 deaths per 100,000 in our sample tracts (model 2). This finding is smaller than, but comparable to the 11% increase in mortality risk from a 1ppm increase in $PM_{2.5}$ found in (6). However, this positive significant effect diminishes when we account for population weights in running the OLS

model (model 3), emphasizing that inclusion of control variables can bias estimation by conditioning on post-treatment variables, as mentioned previously. Finally, when we incorporate the Safegraph mobility data to adjust for the mortality rate (model 4), we find a null effect, which is qualitatively consistent with our main finding, as shown in the right panels (model 5).

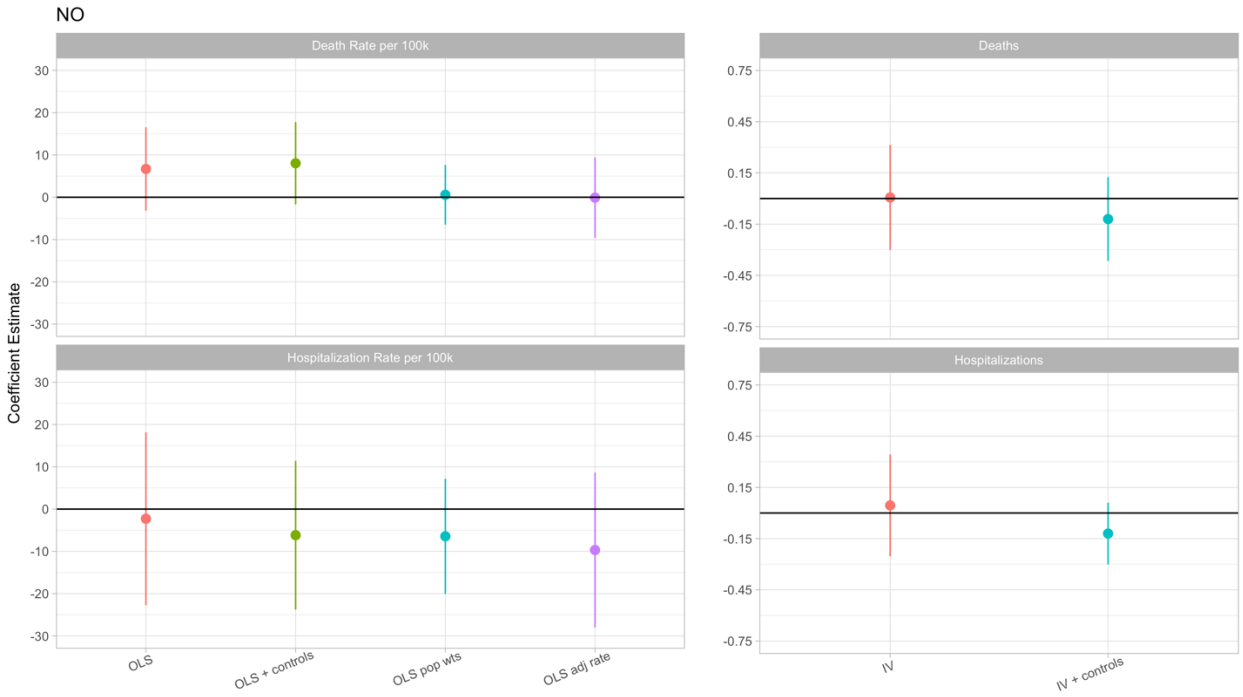

Figure 5. This figure presents coefficient estimates for various models exploring the impact of increased ambient NO concentration on COVID-19 deaths and hospitalizations. OLS models do not capture the causal effect of air pollution due to its endogeneity. These rate-based models are also impacted by measurement error in the dependent variable that is correlated with unobservables that determine COVID-19 outcomes. These rate-based models are still impacted by measurement error in the dependent variable, while the inclusion of covariates can bias estimation by conditioning on post-treatment variables. The 2SLS models capture the causal effect of air pollution on COVID-19 deaths, through both direct and indirect channels in models without covariates.

Discussion

Work exploring links between chronic ambient pollutant concentrations and health outcomes, including COVID-19 outcomes, such as existing observational studies, is usually confronted by three challenges. In this section, we provide an in-depth discussion of the identification challenges faced in this strand of the literature and replicate the results from existing studies that ignore these key identification issues.

Challenge 1: "Cleaning" Air Pollutant Measures

A key feature in our model is the ability to identify the variation in air pollutant concentration that is purged of correlation with demographic characteristics. It has been well documented that low-income and majority non-white neighborhoods are often subjected to higher concentrations of air pollutants (12-16), which suggests that poverty or other correlated characteristics could confound the results from recent observational studies about the relationship between air quality and COVID-19 outcomes (17). Including demographic controls in these models does not solve this issue (18, see table S11). These endogenous

measures of air pollution will bias all coefficient estimates in such regressions (19), due to the causal link between air pollution and several individual characteristics (e.g., income, pre-existing health conditions). Importantly, it is difficult to know a priori the direction of the bias - correlational studies could either over or underestimate the effect of pollution on health.

While we reject a causal link between air quality and COVID-19 outcome using exogenously identified chronic pollutant concentration measures, we are able to replicate the results of observational studies that find a significant positive relationship by using a larger sample: census tracts that lie within 1000 meters of the nearest highway, rather than 500 meters (see table S7 and S8). However, in this larger sample, our instrument is correlated with several observable demographic characteristics that also determine COVID-19 outcomes (see table S3), thus failing the exclusion restriction necessary to establish a causal relationship in the IV estimation. This correlation may arise due to the fact that at the farther distance, tracts that are surrounded by highways appear to be downwind more often, and these tracts may differ in important ways. By confining ourselves to a more restricted and cleaner sample, we are confident that we have an appropriate set-up for identifying a causal relationship between air quality and COVID-19 outcomes.

Challenge 2: Defensive Behaviors Threaten the Validity of Rate-Based Models

As detailed above, studies relying on administrative population data to construct estimates of mortality and hospitalization rates during disease outbreaks that threaten public health are subject to systematic measurement errors. Such errors are likely correlated with determinants of an individual's susceptibility to the disease (e.g., income, ability to temporarily relocate), as well as pollution levels, which would bias regression results with rate-based dependent variables and studies relying on administrative data to create comparison groups (see e.g., 20). Further, the standard practice of using population weights to account for heteroskedasticity in these rate-based models exacerbates the existing measurement error problem.

This error is potentially a substantial problem for previous studies on this topic, as COVID-19 caused many people to leave cities, which tend to have higher pollution levels. Failure to account for differential availability of defensive measures, including the ability to temporarily move away from infection hotspots, can lead to systematic measurement error in commonly used rate-based measures of disease outcomes, in our context, COVID-19, that can bias regression results from both observational and causal studies.

While the spatial patterns of air quality and demographic characteristics in NYC differ from other parts of the nation, our findings reinforce the point that disparities exist across incomes and racial groups regarding the ability to mitigate exposure to environmental and public health hazards, a point emphasized by a recent study of ambient air quality conditions in California (21). The Safegraph data suggest that the wealthier, whiter population of Manhattan below 110th Street was not less susceptible to COVID-19 or air pollution, but appeared to experience lower death and hospitalization rates than other groups largely due to defensive behaviors and temporary relocation that were not reflected in administrative data.

Challenge 3: Ambient Pollutant Concentrations Are Not Measures of Exposure, Especially at Coarse Spatial Scales

Aggregated ambient pollutant concentrations are typically used as a proxy for exposure, a particularly strong assumption given the coarse spatial resolution of most pollutant concentration measures, whether derived from sparse governmental monitoring networks or satellite estimates. Note that existing studies have tended to use county-level pollutant data aggregated or calibrated using EPA monitors, which usually bear the issues of being sparsely and strategically sited, resulting in known limitations of the EPA monitoring network and concentration estimates derived from satellite measurements (22-25).

Our study was conducted at a much finer spatial resolution across a smaller geographical area than existing work in this field. The NYCCAS monitoring network provides fine-scaled and high-quality pollution measurements at about 100 city locations active between 2009-2018, as compared to the 12 EPA monitors in the city. NYC is one of the few places in the United States with such a concentrated network of monitors, and our results suggest that there is great value in the establishment of a national network at similar scales. Additionally, our data on census tract level COVID-19 outcomes allow for much more precise attribution of exposure than county-level data. Our results, which vary from those reported in several observational studies, highlight the importance of improved indoor and outdoor pollutant monitoring to acknowledge differential access to defensive behaviors across segments of our society.

Conclusion

We have attempted to carefully identify the causal relationship between chronic ambient concentrations of several air pollutants and the intensity of COVID-19 disease. Using an IV approach to develop exogenous measures of chronic ambient air quality, we find that increases in the average chronic concentration of three traffic-related air pollutants do not have meaningful impacts on COVID-19 outcomes in NYC.

That we do not observe a causal link between chronic ambient air pollutant concentrations and COVID-19 outcomes in NYC does not mean that such a causal impact might not exist in other contexts. Our estimated effects are observed at chronic ambient pollutant concentrations that fall below the NAAQS thresholds for $PM_{2.5}$ (three-year annual average of 12 $\mu g/m^3$ for the primary standard) and NO_2 (annual average of 53 ppb). Given the sparse spatial coverage of our national air-quality monitoring network, and its susceptibility to strategic siting, it is quite possible that further amendment of the Clean Air Act could increase its net benefits to society, which are already known to be substantial (26).

We emphasize that our estimate of air quality concentration might not fully capture the 10-year cumulative exposure to air pollution. Rather, it only captures the average ambient air pollutant concentration. This limitation relates to the challenge of proxying for a stimulus variable with ambient concentration measures, as the ability to moderate exposure to air pollution via defensive behaviors may differ across individuals. We have shown these behaviors to be very important in the context of COVID-19, which is just one of the respiratory ailments through which air-pollutant exposure might lead to premature death and reduced quality of life. This is a challenge for all existing work that has explored the health effects of air quality and their implications for behavior.

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