



Blowin' in the wind of an invisible killer: long-term exposure to ozone and respiratory mortality in the USA

Ziheng Liu¹ · Xi Chen^{2,3} · Qinan Lu⁴

Received: 5 June 2024 / Accepted: 30 May 2025

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Abstract

In light of the lower public awareness of ozone pollution than other forms of pollution and the potential health threats posed by long-term ozone exposure, this study estimates the causal effect of long-term ozone exposure on respiratory mortality. By employing an instrumental variable approach based on the transmission of ozone from upwind neighbor counties, we discover that an increase in the average concentrations of ozone in the preceding 5 years significantly increases respiratory mortality. The findings show that long-term ozone exposure increases mortality from both acute and chronic respiratory diseases and has significant adverse effects on vulnerable groups. Our bootstrap simulation results suggest that if ozone concentrations in the preceding 5 years decrease by 10%, 11,498 annual deaths from respiratory diseases could be avoided in the USA, accruing health benefits valued at around \$75.50–80.32 billion. Our further estimates suggest that, consistent with general respiratory diseases, long-term ozone exposure also contributes to deaths from COVID-19 during the pandemic.

Keywords Long-term exposure · Ozone pollution · Respiratory diseases · Health benefits

Responsible editor: Shuaizhang Fen

✉ Qinan Lu
qinan.lu@ruc.edu.cn

Ziheng Liu
liuziheng97@hotmail.com

Xi Chen
xi.chen@yale.edu

¹ China Academy for Rural Development (CARD), School of Public Affairs, Zhejiang University, Hangzhou, China

² Department of Health Policy and Management, Yale School of Public Health, New Haven, USA

³ Department of Economics, Yale University, New Haven, USA

⁴ School of Agricultural Economics and Rural Development, Renmin University of China, Beijing, China

JEL Classification I15 · J14 · Q53

1 Introduction

The coronavirus disease 2019 (COVID-19) pandemic prompted renewed attention to respiratory health. Prior to the pandemic, two out of the ten leading causes of death in the USA were attributed to respiratory diseases (Heron 2021). According to the *Multiple Cause-of-Death Mortality* dataset, approximately 260,000 lives—almost 10% of the total death toll—were lost annually because of respiratory diseases from 2008 to 2019 in the USA.¹ This situation was aggravated after the coronavirus outbreak: in the USA, over a million individuals lost their lives as a result of COVID-19, which was projected to cost more than \$16 trillion (Cutler and Summers 2020; Murphy et al. 2021; Ahmad et al. 2022).

As the primary constituent of smog, surface-level ozone pollution is recognized as a persistent challenge and a major health hazard (Speight 2007). Unlike particulate matter, the most common subject of the literature estimating the health effects of air pollutants (Jha and Muller 2018; DeCicca and Malak 2020; He et al. 2020; Deschenes et al. 2020; Zhang et al. 2023; Shen and Sun 2025), surface-level ozone pollution is invisible (Kong et al. 2022). Public awareness of ozone is low due to its invisibility, and protective measures preventing ozone inhalation have been ignored. Additionally, as ozone has a pleasant odor, what is commonly perceived as “fresh air” may be produced by high ozone concentrations. For example, the fresh scent after a thunderstorm stems from the surface-level ozone attributed to lightning nitrogen oxides (NO_x) emissions (Kang et al. 2020). These features and misperceptions about ozone reinforce the importance of studying its health effects.

While contemporaneous effects of ozone exposure on respiratory health have been well established (Peden et al. 1995; Kim et al. 2011), studies examining the long-term effects are limited and should be interpreted as correlations rather than causal impacts (Jerrett et al. 2009; Hao et al. 2015; Turner et al. 2016; EPA 2020). In this study, we define “long-term exposure” as exposure to ambient ozone over a number of years prior to death, rather than early-life exposure, because of the limited early-life information available in the death records and the county-level data structure of this study.² The long-term effects of ozone on respiratory health are different from their short-term counterparts. Long-term ozone exposure may cause considerably more hazardous respiratory infections and endanger human life by delaying or disrupting the self-recovery process via chronically increased systemic inflammation (Perera et al. 2007). Furthermore, examining the long-term effects of ozone exposure is more in line with real-world experiences, as we breathe all the time and are continuously exposed to ozone.

¹ See the Data section for more information on the *Multiple Cause-of-Death Mortality* dataset.

² The data set is at the county-by-year level, and county residents might have migrated from different locations in their early life.

Identifying the causal effect of long-term ozone exposure on respiratory mortality is challenging owing to measurement errors and omitted variable bias. For example, as ozone precursors, NO_x and volatile organic compounds (VOCs) correlate with a variety of economic confounders, including industrial and vehicle emissions, which are linked to regional income (Anenberg et al. 2017; Gaudel 2022), influencing the quantity and quality of health facilities. Additionally, measurement errors may emanate from the fact that the exact location and the exact ozone exposure of each deceased person are unidentified, as the smallest identifiable geographical unit is the county, and the ozone pollution data are constructed from monitors in fixed locations (Schlenker and Walker 2016; Deschenes et al. 2020).

We address these empirical challenges and identify the causal effect of long-term ozone exposure on respiratory mortality by employing upwind ozone as an instrumental variable (IV). This approach relies on the evidence that surface-level ozone can be transferred by wind over a long distance (Brankov et al. 1998; Wang et al. 2022). As an arguably exogenous shock to local health, upwind ozone within a certain distance range influences local respiratory mortality only through its impact on local ozone levels. We also flexibly control for weather and include the county, state-by-month, and season-by-year fixed effects to control for county-specific characteristics, state-specific seasonality, and nationwide time-varying shocks.

We discover that long-term ozone exposure significantly increases mortality from respiratory diseases by employing a two-stage least squares (2SLS) estimator. Specifically, an increase of 1 ppb in the average ozone levels over the preceding 5 years increases deaths from respiratory diseases by 0.089–0.097 per 100,000. Consistent with expectations, such effects are not significant if the exposure window is set to be overly short (i.e., less than 3 years), suggesting that several years of exposure to ozone pollution are necessary to lead to deaths from respiratory diseases.³ Our bootstrap simulation results suggest that if ozone concentrations in the preceding 5 years decreased by 10%, 11,498 deaths from respiratory diseases could be avoided annually, and the respiratory health benefits would be \$75.50–80.32 billion, which account for 0.35–0.37% of the 2019 US gross domestic product (hereafter, GDP).

We then study the heterogeneous effects of long-term ozone exposure on respiratory mortality across diseases and age-by-sex groups. We discover that ozone pollution in the preceding 5 years increases mortality from both acute and chronic illnesses, and that ozone has considerably greater effects on older adults.

This study contributes to the literature in the following ways. First, whereas some epidemiological studies associate long-term ozone exposure with deaths from respiratory diseases (Jerrett et al. 2009; Hao et al. 2015; Turner et al. 2016; Lim et al. 2019), evidence supporting a causal relationship is scarce, and there is no consensus about the duration of the exposure window over which ozone pollution affects health in the long term. This study employs a causal inference framework to assess the long-term causal effects of ozone exposure on respiratory health outcomes. By constructing an IV based on the transmission of ozone from upwind neighbor counties,

³ The exposure window is defined as the time duration over which ozone pollution affects mortality from respiratory diseases.

our estimated long-term ozone effects on respiratory health outcomes are arguably free of biases from measurement errors and omitted variables.

Qiu et al. (2024), which looks into the ozone effect on mortality rates in China, is the closest study to ours. While using a similar IV strategy, our study is different from theirs in the following ways. Unlike Qiu et al. (2024), this study focuses on the US healthcare access, population health baselines, and the primary focus of air pollution regulations in the USA differ from China. The contextual distinctions underscore the importance of investigating ozone's impact on mortality in the USA. In addition, our data coverages differ. We rely on a full-sample dataset that includes all fatalities in the USA, which ensures population representation and increases the reliability and validity of our results. We also investigate the effects on a number of specific diseases and mortality risks, including respiratory illnesses such as pneumonia and chronic obstructive pulmonary disease, as well as diseases from other categories, such as gastrointestinal diseases, suicide, diabetes, falls, and drowning. The disease-specific analysis gives information on the mechanisms via which ozone exposure impacts health.

Second, this study contributes to the literature estimating the effects of pollution on COVID-19, the most substantial economic and public health crisis in the past few decades (Coker et al. 2020; Cole et al. 2020; Persico and Johnson 2021; Ispording and Pestel 2021). However, these studies only assess the contemporaneous impacts of air pollution on COVID-19. To the best of our knowledge, our extension analysis provides the first evidence of the long-term effects of ozone exposure on COVID-19 mortality. It indicates that, had average ozone concentrations in the 5 years prior to the pandemic dropped by 1 ppb, 7856 fewer deaths per month in the US population could have been achieved during the pandemic.

The rest of this paper is organized as follows. "Background" Section presents the background of surface-level ozone pollution and respiratory diseases in the USA. "Empirical strategy" Section shows the empirical strategy used in this study. "Data" Section introduces data and provides summary statistics. "Results" Section shows the estimation results. "Robustness and heterogeneity of effects" Section presents robustness checks and heterogeneity analyses. "Extension analyses" Section presents the extension analyses and, finally, "Conclusions" Section concludes the paper.

2 Background

2.1 Surface-level ozone pollution

Surface-level ozone is a secondary product of its two precursors, VOCs and NO_x (Auffhammer and Kellogg 2011). VOCs and NO_x are emitted from both industrial (e.g., gasoline and power plants) and natural sources (e.g., deciduous trees). The ozone formation in high-VOC regions is determined by NO_x concentrations; these regions are referred to as "NO_x-limited," generally located in rural areas. By contrast, the so-called VOC-limited, which is primarily found in metropolitan areas, is where NO_x levels are relatively high, and the ozone formation is determined by VOC concentrations (Sillman 1999; Auffhammer and Kellogg 2011). Additionally, sharing the same property as

particulate matters, surface-level ozone can also be transferred by wind over a certain distance (Brankov et al. 1998).

Because ozone pollution reduces worker productivity (Wang et al. 2022) and harms human health (Neidell 2004; Currie et al. 2009), a number of regulations have been enacted. NO_x becomes the regulation target, as ozone cannot be directly regulated (Deschenes et al. 2017) and VOC emissions are mostly from biogenic sources (Auffhammer and Kellogg 2011). Efforts to reduce NO_x in the USA include the Reid vapor pressure regulation, the Federal reformulated gasoline, and the 1990 Clean Air Act Amendments (Henderson 1996; Walker 2013). As a result of such regulations, VOC concentrations become relatively high in comparison to NO_x , and much of the USA is currently NO_x -limited (Jung et al. 2022).

2.2 Respiratory diseases and biological mechanisms

Two out of ten leading death causes in the USA were respiratory-disease-related. These diseases include influenza/pneumonia and chronic lower respiratory diseases (Heron 2021). According to the *Multiple Cause-of-Death Mortality* dataset, more than three million lives were lost in the past decade due to respiratory diseases. Owing to the threats posed to human health, respiratory diseases have drawn considerable attention in the existing literature. Numerous studies have examined the various factors associated with respiratory diseases, including cigarette smoking (Mullahy and Portney 1990), diesel emissions (Beatty and Shimshack 2011), avoidance behavior (Janke 2014), family income (Kuehnle 2014), agricultural fire (He et al. 2020), and natural gas flaring (Blundell and Kokoza 2022).

The contemporaneous effects of ozone exposure on respiratory diseases are well studied: short-term ozone exposure affects respiratory health by triggering allergen-induced responses, retarding pulmonary function, and increasing neutrophilic airway inflammation (Peden et al. 1995; Kleeberger et al. 1997; Kim et al. 2011); while a self-recovery ability of the respiratory system after acute injury involves inflammation resolution, cell proliferation, and tissue remodeling (González-López and Albaiceta 2012). The acute damage effects produced by ozone pollution may lead to a simultaneous increase in hospitalizations for respiratory illnesses (Neidell 2004; Janke 2014).

Contrary to the well-established contemporaneous effects, the long-term respiratory health effects of ozone exposure remain to be elucidated (Hao et al. 2015; EPA 2020). Different from the short-term effects of ozone exposure on respiratory health, the long-term ozone exposure may cause respiratory illnesses that are considerably more dangerous and threaten human lives by delaying or destroying the self-recovery process through persistently heightened systemic inflammation (Perera et al. 2007). As a result of persistent and recurring inflammation, long-term ozone exposure is likely to cause severe and serious respiratory diseases.

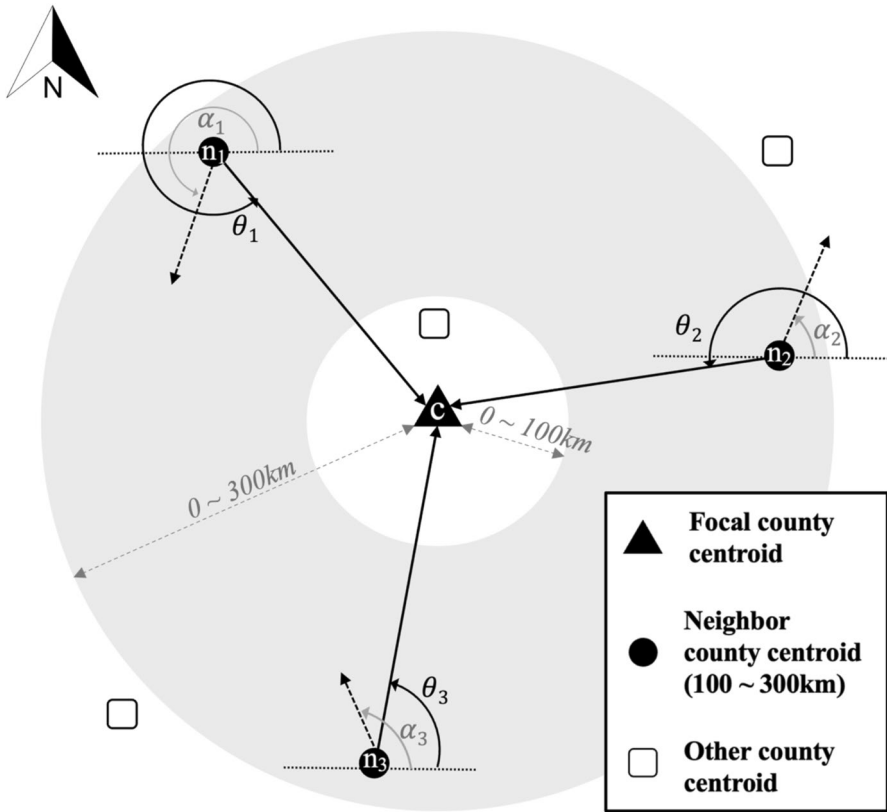


Fig. 1 Instrumental variable strategy. Notes: This figure illustrates an example of the long-distance transmission of surface-level ozone from upwind counties within a distance radius of 100 ~300 km to a focal county. The triangle represents a focal county. The circles represent neighbor counties distanced 100 ~300 km from the focal county. The squares refer to other counties. The solid line arrows represent vectors connecting neighbor counties from a range of 100 ~300 km to a focal county. The dashed line arrows represent wind flows

3 Empirical strategy

The possibility for omitted-variable bias poses a challenge to identifying the ozone effects on respiratory mortality. As introduced in the “Background” section, NO_x and VOCs correlate with numerous economic confounders, including industrial and vehicle emissions (Anenberg et al. 2017; Gaudel 2022). These confounders tend to be highly correlated with regional income, which determines the quantity and quality of health facilities accessible to residents. Additionally, because we could only identify the residence county of the deceased and are using ozone pollution data constructed from fixed monitors, the exact home address and the exact ozone exposure of the deceased are unidentified; this leads to classical measurement errors (Schlenker and Walker 2016).

To address the endogeneity concern stemming from the omitted variables and measurement errors, we build an IV based on the fact that ozone could be transferred by wind over a certain distance (Brankov et al. 1998), which is a property similar to particulate matters (Schlenker and Walker 2016; Chen et al. 2021). Specifically, referring to Liu and Lu (2023) and Wang et al. (2022), we rely on the long-range transmission of surface-level ozone from upwind counties to construct an IV that is both an exogenous shock to local respiratory health and a predictor of local ozone levels. Figure 1 shows the construction procedure of the IV. The triangle denotes a focal county, and the dots represent neighbor counties at distances of 100–300 km from the focal county. Only the counties within this 100–300-km band are considered when computing the ozone transferred from upwind counties for a focal county. We set the band as 100–300 km following Chen et al. (2021). The instrument exclusion restriction criteria would not hold if such a radius band was set considerably close to a focal county. This is because the ozone from nearby upwind neighbor counties may affect residents living in the focal county through channels other than long-range pollution transmissions. If such a radius band was set considerably far from a focal county, the instrument relevance criteria would fail because ozone from upwind neighbor counties would no longer be a predictor of ozone levels within the focal county. We alternatively set the radius band as 150–300 km and 200–350 km in the “Results” section, and our estimates remain robust.

As shown in Fig. 1, among the three neighbor counties in the 100–300-km radius band in Fig. 1, n_1 and n_3 are upwind counties to focal county c , whereas n_2 is not an upwind county to focal county c , as wind blows in a direction opposite to county c . Specifically, we assign the following weight (e.g., Eq. (1)) for each neighbor county n within the 100–300-km radius band.⁴ In Eq. (1), d_{nc} represents the distance between a focal county and its neighbor county. The term $1\{\cos(\theta_{nc,t} - \alpha_{nc,t}) > 0\}$ excludes neighbor counties (n_2 in the case of Fig. 1) where the wind does not blow toward the focal county.

$$w_{nc,t} = \frac{\frac{\cos(\theta_{nc,t} - \alpha_{nc,t})}{d_{nc}} \cdot 1\{\cos(\theta_{nc,t} - \alpha_{nc,t}) > 0\}}{\sum_{k=1}^m \frac{\cos(\theta_{kc,t} - \alpha_{kc,t})}{d_{kc}} \cdot 1\{\cos(\theta_{kc,t} - \alpha_{kc,t}) > 0\}}. \tag{1}$$

Using the assigned weight $w_{nc,t}$, we then calculate the weighted average of upwind ozone from all neighbor counties n distancing 100–300 km to the focal county c . Finally, we sum the weighted average over the preceding week, as the transmission of ozone pollution is time-consuming.

The 2SLS model estimating the effect of long-term ozone exposure on respiratory mortality is as follows:

$$Ozone_{cym} = \eta_0 + \eta_1 Ozone_{IV,cym} + W_{cym}\varphi + \gamma_c + \delta_{sm} + \theta_{yz} + \mu_{cym}, \tag{2}$$

⁴ The weight is constructed referring to Wang et al. (2022).

$$Y_{cym} = \beta_0 + \beta_1 \widehat{Ozone}_{cym} + W_{cym}\varphi + \gamma_c + \delta_{sm} + \theta_{yz} + \varepsilon_{cym}. \quad (3)$$

Equations (2) and (3) are the first and second stages, respectively, of the 2SLS model. In this model, Y_{cym} denotes deaths per 100,000 from respiratory diseases at residence county c in month m and year y . The primary parameter of interest is β_1 , the coefficient on the average concentrations of daily maximum 8-h, 12-h, and 24-h ozone over a certain time duration, which account for maximum, daytime, and all-day exposures to surface-level ozone, respectively. Given that no consensus has been reached about the duration over which ozone pollution may affect health in the long term (EPA 2020), we vary the time window from half a year to five and a half years and allow the data to ascertain the exposure time window, instead of having a priori assumed duration over which ozone causes deaths from respiratory diseases.⁵ The primary coefficients of interest for all exposure windows varying from half a year to five and a half years will be presented later in the “Results” section. As explained later, a 5-year duration is an appropriate exposure window and is employed here. For example, for the deceased in county A in December 2010, the ozone exposure is calculated as the county’s average ozone concentrations from January 2006 to December 2010.

Some weather characteristics correlate with surface-level ozone pollution and may also affect respiratory health (Barreca 2012; Yu et al. 2019). Therefore, we control for W_{cym} , a set of weather conditions over the same 5-year exposure window. Specifically, the weather controls include maximum temperature, wind direction, maximum wind speed, net solar radiation, total precipitation, surface atmospheric pressure, and relative humidity. We further control for the quadratic polynomials of the aforementioned weather characteristics to model their nonlinear effects on respiratory health (Deschenes et al. 2020).

Referring to Deryugina et al. (2019), our specification includes the county (γ_c), state-by-month (δ_{sm}), and season-by-year (θ_{yz}) fixed effects. The county-fixed effects absorb time-invariant and county-specific features that may correlate with ozone pollution and respiratory health. The state-by-month fixed effects absorb any state-varying seasonal association between environmental factors and respiratory diseases, such as the effects of pollen. The season-by-year fixed effects control for nationwide time-varying shocks. Finally, ε_{cym} refers to the idiosyncratic error. We cluster standard errors in the baseline specification at the state level (Wooldridge 2003).

The identification assumption of the 2SLS model is that, after including the county fixed effects, state-by-month fixed effects, season-by-year fixed effects, and a rich set of weather controls, upwind ozone could affect the respiratory health of residents living within the focal county only through its impact on the focal-county ozone concentrations. Averaged to the same exposure window as $Ozone_{cym}$ and weather characteristics, $Ozone_{IV,cym}$ denotes the 5-year average of the 100–300-km upwind ozone for county c over the preceding week.

⁵ This approach was initially used by Deschenes et al. (2020).

4 Data

We combine respiratory deaths data with information on surface-level ozone, weather characteristics, county population size, and other air pollutants at the month-county level. Here, we provide an overview of our matched dataset.

4.1 Deaths from respiratory diseases

We obtained respiratory death data from the National Center for Health Statistics' *Multiple Cause-of-Death Mortality* dataset.⁶ This is the most complete national dataset, reporting all deaths in the USA. The dataset includes information about the underlying causes of death, as well as age, gender, and county of residence. Our sample excludes observations in which the county of residence and county of death do not match. We aggregate the total number of resident deaths from respiratory diseases, identified by the underlying causes of deaths with ICD-10 codes J00-J99,⁷ to the county-month level between 2008 and 2019. Additionally, we conduct heterogeneity analysis by further aggregating the total number of deaths from respiratory diseases by age-by-sex groups to the county-month level. Furthermore, the demographic data were obtained from the National Cancer Institute.⁸ We calculate the respiratory disease deaths per 100,000 at the county-month level using the total population counts and population counts by age-by-sex groups of each county.

4.2 Air pollutants

Our explanatory variables of interest are ground-level daily maximum 8-h, 12-h, and 24-h ozone. We obtain hourly ozone data from the Environmental Protection Agency (EPA) of the USA. Following Chen and Gong (2021), Qiu et al. (2020), and Zhang et al. (2017), we interpolate ozone concentrations of all monitoring stations located less than 100 km from each county centroid using the inverse-distance-weighted (IDW) method.⁹ We then average the ozone concentrations to the 5-year exposure window for each county.

The other criteria pollutants that we further control for in the robustness checks include carbon monoxide (CO), particulate matters (PM_{2.5}, PM₁₀), sulfur dioxide (SO₂), and nitrogen dioxide (NO₂). We obtain PM_{2.5} data from EPA and retrieve data of CO, PM₁₀, SO₂, and NO₂ from the European Centre for Medium-Term Weather Forecasting's (ECMWF) fourth-generation global reanalysis of atmospheric composition (EAC4).¹⁰ We interpolate their concentrations of all grid cells located within 100 km of each county centroid using the same aforementioned IDW method.

⁶ See https://www.cdc.gov/nchs/data_access/ftp_data.htm for more information of the Multiple Cause-of-Death Mortality dataset.

⁷ ICD-10 refers to the International Classification of Diseases, 10th Revision.

⁸ See <https://seer.cancer.gov/popdata/download.html> for further information about the demographic data.

⁹ We have also matched the data from the nearest monitoring stations to each county centroids, and the results are similar to the results relying on the IDW method.

¹⁰ See Inness et al. (2019) for further information.

Studies relying on the reanalysis data from ECMWF include Axbard (2016), Cai et al. (2016), Colmer (2021), Giaccherini et al. (2021), and Koppensteiner and Menezes (2024). We employ the reanalysis data, as the geographic coverage of EPA data for CO, PM₁₀, SO₂, and NO₂ is considerably smaller compared to O₃ and PM_{2.5}. If we used the EPA data for CO, PM₁₀, SO₂, and NO₂, a large number of counties would be excluded from our data sample. Considering that we aim to conduct a nationwide analysis, a smaller sample size would restrict our analysis and findings to a considerably smaller scale.

4.3 Weather

We control for a series of weather characteristics, as respiratory health is also correlated with weather conditions (Barreca 2012; Yu et al. 2019). The data on temperature, wind direction, and wind speed are obtained from the EPA. The data on total precipitation, surface atmospheric pressure, relative humidity, and net solar radiation are collected from ECMWF on a grid of $0.25^\circ \times 0.25^\circ$.¹¹ We interpolate weather characteristics to each county using the aforementioned IDW method and aggregate to the 5-year exposure window. Referring to Deschenes et al. (2020), we control for the quadratic polynomials of all the weather characteristics to take the potential nonlinear effects of weather into account.

4.4 Summary statistics

The final data sample used in the analysis is at the county-by-month level. The primary explanatory variables are daily maximum 8-h, 12-h, and 24-h ozone, accounting for daily maximum, daytime, and all-day exposures to surface-level ozone. The weather controls include the quadratic polynomials of maximum temperature, total precipitation, relative humidity, net solar radiation, surface pressure, wind direction, and maximum wind speed. Both the primary explanatory variables and weather controls are aggregated to the 5-year exposure window.

Table B1 shows the summary statistics. The average of our dependent variable, the monthly deaths from respiratory diseases, is 8.2 per 100,000. The average values of daily maximum 8-h, 12-h, and 24-h ozone, our explanatory variables of interest, are 38.8 ppb, 34.8 ppb, and 30.8 ppb, respectively. Our IV, the average of 1-week cumulative ozone concentrations from upwind neighbor counties over the 5-year exposure window, is 204.9 ppb.¹²

¹¹ The name of the dataset is the fifth-generation ECMWF reanalysis data (ERA5). See Hersbach et al. (2019) for further information.

¹² This number is approximately seven times as high as the explanatory variable of interest because it represents the cumulative value over 1 week.

5 Results

5.1 First-stage estimates: effects of ozone from upwind neighbor counties on ozone in the focal county

Panel A of Table 1 shows the estimated effects of upwind ozone (the constructed IV) on ozone levels in the focal county. First, we discover that upwind ozone from neighbor counties is predictive of ozone concentrations within the focal county: the estimated coefficients are statistically significant at 1% for daily maximum 8-h, 12-h, and 24-h ozone, which demonstrates that the instrument relevance criterion is fulfilled. The estimates suggest that an additional ppb of upwind ozone significantly increases ozone levels within the focal county; thus, reassuring the validity of our IV. Additionally, the Kleibergen-Paap F-statistics for all three

Table 1 Effects of ozone pollution on mortality from respiratory diseases

	Respiratory diseases deaths per 100,000		
	Max 8-h ozone (1)	12-h ozone (2)	24-h ozone (3)
Panel A: 2SLS (first stage)			
Ozone concentrations	0.019*** (0.000)	0.017*** (0.000)	0.018*** (0.000)
Panel B: 2SLS (second stage)			
Ozone concentrations	0.089** (0.040)	0.097** (0.044)	0.094** (0.042)
KP F-statistics	2818	2511	4319
Observations	254,120	254,120	254,120
County FE	Yes	Yes	Yes
State-month FE	Yes	Yes	Yes
Year-season FE	Yes	Yes	Yes
Weather controls	Yes	Yes	Yes

This table reports the estimated effects of 5-year average concentrations of ozone on respiratory diseases deaths per 100,000. Panel A reports the first stage of the 2SLS results. Panel B reports the second stage of the 2SLS results, in which we use ozone from upwind counties in the range of 100 ~ 300 km as the instrumental variable. The dependent variable is respiratory diseases deaths per 100,000. The primary explanatory variables are the 5-year average concentrations of maximum 8-h ozone, 12-h ozone, and 24-h ozone in Col. (1)–(3), respectively. The weather controls include the quadratics of maximum temperature, relative humidity, precipitation, solar radiation, maximum wind speed, wind direction, and surface atmospheric pressure. We include the county fixed effects, state-by-month fixed effects, and year-by-season fixed effects in this table. Standard errors are clustered at the state level

* $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$

measurements of ozone are far above the Stock-Yogo value of 16.38 (Stock and Yogo 2005), suggesting that our constructed IV is not weak.

5.2 Effects of long-term ozone exposure on respiratory mortality

Panels B of Table 1 presents results from the second stage of the 2SLS estimates.¹³ As aforementioned, the 2SLS estimates mitigate the biases from measurement errors and omitted variables: First, the exact home address of the deceased is unknown, and the accurate ozone concentration level is unidentified, as we could only identify the residence county of the deceased and use county-level ozone pollution data constructed from fixed monitors. Second, confounding factors associated with both ozone and respiratory health, such as regional economic status, may bias the estimated effects as well.

Specifically, as indicated by the results in panel B, an additional ppb in the average concentrations of daily maximum 8-h, 12-h, and 24-h ozone in the preceding 5 years increases deaths per 100,000 from respiratory diseases by 0.089, 0.097, and 0.094, respectively. Converting to a magnitude of standard deviations, the estimates show that a one-standard-deviation rise in the average daily maximum 8-h, 12-h, and 24-h ozone in the preceding 5 years increases respiratory mortality by 0.055–0.058 standard deviations. These estimates are consistent with our expectations that while contemporaneous ozone pollution increases hospitalizations from respiratory illness (Neidell 2004; Janke 2014), long-term ozone exposure can cause more serious and dangerous respiratory diseases.

5.3 Comparison with the literature

As this study is among the first to use a causal inference framework to examine the causal effects of long-term ozone exposure on respiratory mortality, we compare our estimates with the epidemiological literature assessing the association between the two in this section. Our estimates suggest that an additional ppb of the average 24-h ozone concentrations in the preceding 5 years increases 0.094 deaths per 100,000 from respiratory diseases, which corresponds to a 1.1% increase in respiratory mortality, assuming the effect is linear. The closest estimate in the existing literature is from Turner et al. (2016), who discovered that an additional ppb of average ozone concentrations in the preceding 3 years resulted in a 1.2% increase in the respiratory mortality. Considerably lower than our estimates, Lim et al. (2019) discovered that an additional ppb of average ozone concentrations in the preceding year led to a 0.4% increase in the respiratory mortality. Jerrett et al. (2009) discovered that an additional ppb of average ozone concentrations over the period of 1977–2000

¹³ We also conduct an analysis that allows for a nonlinear response and observe a roughly linear and stable increase in respiratory mortality across the entire range of ozone indicators. The coefficients across the full range of ozone indicators are positive, with no sharp jumps in magnitude. This implies that there is no clear evidence of a nonlinear relationship between ozone and respiratory mortality, which is most likely because our ozone metric is averaged over a 5-year exposure window.

was associated with a 0.4% increase in the respiratory mortality over the period of 1982–2000. The difference in magnitude is consistent with our expectation, as our estimates are more plausibly causal: estimates from the existing epidemiological literature should at least partially be interpreted as associational and might suffer from attenuation bias and endogeneity issues.

6 Robustness and heterogeneity of effects

6.1 Robustness checks

We check the robustness of our estimates to alternative fixed effects, alternative standard-error clustering levels, alternative IV approaches, alternative weather controls, possible omitted air pollutants, and possible omitted socioeconomic factors. Additionally, we restrict the data sample to the east of the 100th meridian and remove the seasonality in ozone concentrations. We also present evidence to support the validity of our exposure window by exploring how alternative exposure windows affect our baseline estimates.

6.1.1 Alternative fixed effects

We employ alternative fixed effects as robustness checks in Table 2 and Table B2, which demonstrates the robustness of our estimates to different fixed effects. Col. (1) of Table 2 shows the baseline estimates, in which we include county, season-by-year, and state-by-month fixed effects to absorb county-specific characteristics, the state-varying seasonal correlation of ozone and mortality, and nationwide time-varying shocks. In Col. (2) of Table 2 and Col. (2) of Table B2, we replace the season-by-year fixed effects with year fixed effects and month-by-year fixed effects, respectively, as alternative controls for unobserved nationwide temporal shocks. In Col. (3) of Table B2, we replace the state-by-month fixed effects with state-by-season fixed effects as an alternative control for the state-varying seasonal correlation of ozone and mortality. In Col. (4)–(6) of Table B2, we assume such an association as county-specific by including county-by-month fixed effects rather than assuming the seasonal association between ozone and mortality as state-specific. As shown in Table 2 and Table B2, these estimates are qualitatively similar to their baseline counterparts.

6.1.2 Alternative clustering methods

Table 2 and Table B2 report the estimates under alternative clustering methods of standard errors. Col. (1) of Table 2 presents the baseline estimates, allowing for autocorrelation in the errors within each state, with standard errors clustered at the state level. Col. (3) of Table 2 and Col. (2) of Table B3 employ two-way clustering and cluster standard errors at the state and month-by-year (season-by-year for the latter) levels, allowing for autocorrelation in the errors within each state and a month-by-year (season-by-year for the latter) cell. Col. (3) and (4) of Table B3

Table 2 Robustness checks

	Baseline		Alt. FE		Alt. CL		Alt. weather controls		Add pollut		East of the 100th	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)				
Panel A: maximum 8-h ozone												
Max eight-hour ozone	0.089** (0.040)	0.089** (0.040)	0.089** (0.040)	0.085** (0.042)	0.085** (0.040)	0.092** (0.039)	0.112** (0.053)	0.098** (0.044)				
Observations	254,120	254,120	254,120	254,120	254,120	254,120	224,695	254,120				
Weather controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes				
KP F-statistics	2818	2866	2818	2572	2856	2889	2478	2554				
Panel B: 12-h ozone												
12-h ozone	0.097** (0.044)	0.098** (0.044)	0.097** (0.044)	0.093** (0.046)	0.093** (0.044)	0.100** (0.042)	0.134** (0.063)	0.105** (0.048)				
Observations	254,120	254,120	254,120	254,120	254,120	254,120	224,695	254,120				
Weather controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes				
KP F-statistics	2511	2560	2511	2320	2543	2662	1805	2222				
Panel C: 24-h ozone												
24-h ozone	0.094** (0.042)	0.094** (0.042)	0.094** (0.042)	0.089** (0.044)	0.089** (0.042)	0.096** (0.041)	0.137** (0.064)	0.100** (0.045)				
Observations	254,120	254,120	254,120	254,120	254,120	254,120	224,695	254,120				

Table 2 (continued)

	Baseline		Alt. FE		Alt. CL		Alt. weather controls		Add pollut		East of the 100th meridian		Mean center by month	
	(1)	(2)	Cnty, StMth, Year	Yes	St, YrMth	Yes	Temp bins	Quadratic avg temp	PM ₁₀	(6)	(7)	(8)		
Weather controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
KP F-statistics	4319	4385	4319	3882	4413	4750	3273	3841						

The dependent variable is respiratory diseases deaths per 100,000. The primary explanatory variables are maximum 8-h ozone concentrations, 12-h ozone concentrations, and 24-h ozone concentrations in panels A, B, and C, respectively. Col. (1) is the baseline estimate which uses ozone from upwind counties in the range of 100 ~ 300 km as the instrumental variable. It controls for the county fixed effects, state-by-month fixed effects, and season-by-year fixed effects. The weather controls in Col. (1) include the quadratics of maximum temperature, relative humidity, precipitation, solar radiation, maximum wind speed, wind direction, and surface atmospheric pressure. Standard errors are clustered at the state level in Col. (1). Col. (2) replaces the fixed effects in Col. (1) with the county fixed effects, state-by-month fixed effects, and year fixed effects. Col. (3) clusters standard errors at the state and year-by-month level (two way). Col. (4) replaces the maximum temperature by days within each 5 °C bin. Col. (5) replaces the quadratic polynomial of maximum temperature with the quadratic polynomial of average temperature. Col. (6) adds PM₁₀ as a control variable. Col. (7) only includes counties east of the 100th meridian. Col. (8) means center ozone by month to remove seasonality

* $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$

use two-way clustering at the state-by-year (state-by-year-month for the latter) and county levels, which allows for the autocorrelation within state-by-year (state-by-year-month for the latter) cells and the autocorrelation within the same county over time. All these estimates remain robust compared to the baseline estimate.

6.1.3 Alternative weather controls

Table 2 reports estimates under various specifications of weather controls. Recall that our baseline estimates control for the quadratic polynomials of maximum temperature, solar radiation, maximum wind speed, wind direction, relative humidity, precipitation, and surface pressure to ensure that ozone pollution is the only channel by which ozone from upwind neighbor counties affects respiratory mortality. To test whether our point estimates are sensitive to the inclusion of maximum temperature, we replace the quadratic polynomial of maximum temperature with temperature bins in Col. (4) of Table 2 and with the quadratic polynomial of average temperature in Col. (5) of Table 2.¹⁴ Both of the two estimates under various specifications of weather controls are qualitatively similar to the baseline.

6.1.4 Controlling for additional air pollutants

There may be a concern that other air pollutants could confound our estimates on ozone. To alleviate this concern, we include additional pollutants, one at a time, in Col. (6) of Table 2, Table B4, and Table B5. We do not include them simultaneously, as air pollutants tend to be highly correlated, and the simultaneous inclusion of multiple air pollutants may cause multicollinearity (Maddison 2005). Consequently, all estimates are still significant at the 5% or 1% level and close to the baseline results. Particulate matter contributes to the global mortality burden. To further validate our baseline specification model, we show the results for PM_{2.5}, instrumented by upwind PM_{2.5}, in Table B5. The estimated coefficients, in Table B5, show that a 1 µg/m³ increase in PM_{2.5} leads to an additional 0.043–0.044 deaths per 100,000 from respiratory diseases (amounting to 0.52–0.54% of the mean), which is substantially consistent with earlier findings in the literature. For example, He et al. (2020) and Fan et al. (2020) found that a 1 µg/m³ rise in PM_{2.5} leads to a 0.4% and 0.29% increase of cardiorespiratory mortality in China, respectively. In the USA, Deryugina et al. (2019) found that a 1 µg/m³ increase in PM_{2.5} causes an additional 0.069 deaths per 100,000; Wang et al. (2020) found that an elevation of 1 µg/m³ in PM_{2.5} is associated with a 0.56% rise in respiratory mortality. These estimates are broadly consistent with our findings, with minor variations caused by differences in datasets, time durations, exposure windows, settings, and disease classifications.

¹⁴ Referring to Chen et al. (2020), Deschenes et al. (2020), and Zhang et al. (2024), we control for the number of days within each temperature bins. The temperature bins include 0–5°C, 5–10°C, 10–15°C, 15–20°C, 20–25°C, 25–30°C, and greater than 30°C.

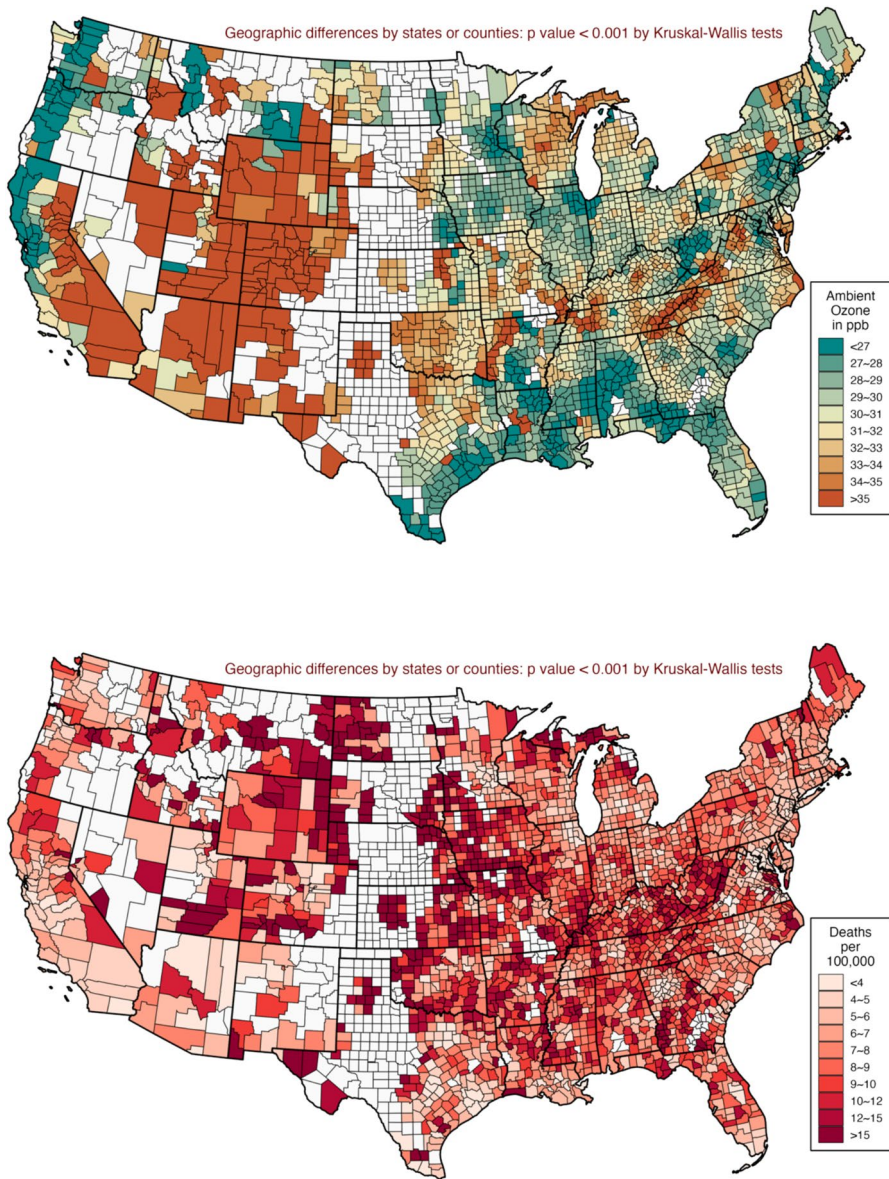


Fig. 2 Ozone concentrations and respiratory mortality. Notes: This top figure depicts the average ozone concentrations in the USA. The bottom figure depicts the average deaths per 100,000 from respiratory diseases in the USA. The p -values of Kruskal–Wallis tests, by either states or counties, are smaller than 0.001 for both ozone concentrations and deaths from respiratory diseases

6.1.5 The 100th meridian

There may be a concern that our estimated baseline results are driven by counties west of the 100th meridian, as such counties are considerably different from counties east of the 100th meridian in climatic conditions. For example, the 100th meridian is the 20-inch rainfall line in the USA (Schlenker et al. 2006). Furthermore, as shown in Fig. 2, ozone concentrations in counties west of the 100th meridian are higher in magnitude compared to their counterparts in counties east of the 100th meridian, and our estimates may be sensitive to such a difference. To address these concerns, we exclude all counties west of the 100th meridian in Col. (7) of Table 2, and our estimates in Col. (7) are qualitatively similar to the baseline estimates in Col. (1), suggesting that our results are not driven by high concentrations in the west counties.

6.1.6 Removing seasonality

Given that ozone concentrations exhibit a seasonable pattern, there may be a concern that seasonality may influence our estimated ozone effect on respiratory mortality. To alleviate this concern, Col. (8) of Table 2 removes the seasonality in ozone concentrations. Specifically, prior to aggregating over the exposure window, we mean-center daily ozone concentrations by month, a common strategy to prevent time trends or seasonable patterns from driving the estimated results (Carter et al. 2021). As shown in Col. (8), our results after removing seasonality in ozone concentrations are similar to the baseline results, alleviating this concern.

6.1.7 Controlling for additional socioeconomic factors

Although we employ a 2SLS-estimation approach, our estimates may still be sensitive to socioeconomic factors. To mitigate this concern, we include a series of socioeconomic variables in Table B6. Col. (2)–(4) of Table B6 add unemployment rate, average annual salary, and poverty rate as control variables, respectively.¹⁵ Our estimates in Col. (2)–(4) are close to the baseline estimates and remain significant at the 5% level, which suggests that our estimates are not confounded by employment status, income level, or poverty. Col. (5) of Table B6

¹⁵ County-level data on unemployment rate, average annual salary, and poverty rate are obtained from the US Bureau of Labor Statistics. For more details about unemployment rate, see <https://www.bls.gov/lau/>. For more details about average annual salary, see https://www.dol.gov/general/topic/statistics/wages_earnings. For more details about poverty rate, see <https://www.census.gov/programs-surveys/saipe.html>.

adds the ratio of population to primary care physicians as a control variable,¹⁶ and our estimates in Col. (5) remain qualitatively similar to the baseline results, showing that our baseline estimation is not confounded by healthcare access.

6.1.8 Alternative IVs

We employ alternative IVs as robustness checks in Table B7; Col. (1) of Table B7 shows the baseline estimate, which uses upwind ozone distancing 100–300 km as an IV. Setting the radius band considerably close to the focal county in a manner wherein ozone pollution from neighbor counties could share emission sources with the focal county may raise a concern. To test the sensitivity of our results to the spatial range of the IV, Col. (2)–(3) of Table B7 use alternative IV specifications with radius bands of 150–300 km and 200–350 km. The results remain statistically significant, confirming that upwind ozone within certain distance ranges effectively predicts ozone concentrations in the focal county. However, as shown in Col. (4), the results using an IV based on the 450–600 km radius band are no longer statistically significant. This is likely because ozone from far counties cannot be effectively transmitted to the focal county by wind, reducing its predictive power for ozone levels within the focal county. Additionally, rather than solely depending on wind direction, referring to Chen et al. (2021) and Wang et al. (2022), we employ another alternative IV in Col. (5) by also taking account of wind speed in the IV construction. This is because higher wind speeds allow long-distance ozone transmission to reach the focal county faster, contributing more to the focal county's ozone concentrations. Specifically, we update the weight assigned to neighbor counties when computing the weighted average of ozone from them. The updated weight equation is as follows:

$$w_{nc,t} = \frac{\frac{\cos(\theta_{nc,t} - \alpha_{nc,t})}{d_{nc}} \cdot 1\{\cos(\theta_{nc,t} - \alpha_{nc,t}) > 0\} \cdot \text{windspeed}_{n,t}}{\sum_{k=1}^m \frac{\cos(\theta_{kc,t} - \alpha_{kc,t})}{d_{kc}} \cdot 1\{\cos(\theta_{kc,t} - \alpha_{kc,t}) > 0\} \cdot \text{windspeed}_{k,t}}. \quad (4)$$

The estimates using the alternative IV that takes account of wind speed are qualitatively similar to our baseline estimate.

6.1.9 Alternative exposure windows

We explore the effects of various exposure windows on our baseline estimates in this section by varying the exposure windows from half a year to five and a half years. Recall that in the baseline specification, we specify the exposure window as 5 years. Following Deschenes et al. (2020), we plot the 2SLS estimates on ozone by varying the exposure window from half a year to five and a half years in Fig A2. Each dot denotes the coefficient of interest from a separate 2SLS estimation. The primary

¹⁶ County-level data on the ratio of population to primary care physicians are obtained from the Population Health Institute at University of Wisconsin. For more details, see <https://www.countyhealthrankings.org/explore-health-rankings/rankings-data-documentation>.

explanatory variables are daily maximum 8-h ozone, 12-h ozone, and 24-h ozone, respectively, for the left, middle, and right plots in Fig A2.

Figure A2 shows the legitimacy of setting the exposure window as 5 years in our baseline specification. The estimates can be divided into three stages, as shown in Fig A2. First, when the exposure window is shorter than 3 years, the estimates of ozone pollution on respiratory mortality are not statistically significant and are close to zero. As previously stated, continuous and recurring inflammations damage the respiratory system's self-recovery process over time (Perera et al. 2007). When the time exposed to ozone pollution is insufficiently long, inflammations caused by ozone pollution may not be frequent and sufficient to destroy the self-recovery mechanism. Therefore, respiratory system illness may not be fatal in this case.

Second, when the exposure windows are between 3 and 5 years, the coefficients of interest are significantly positive, and their magnitude becomes larger when the exposure window enlarges. This suggests that the ozone effects on respiratory diseases become more evident when the exposure time to ozone pollution becomes longer. This is consistent with our expectation; longer exposure to ozone pollution may induce more inflammations, which would destroy the self-recovery mechanism of the respiratory system (Perera et al. 2007).

Third, when the exposure window exceeds 5 years, the estimated effects shrink as the exposure window becomes longer. The shrinkage may be attributed to two reasons. First, some patients may have already passed away after being exposed to ozone pollution for a long period. Second, ozone pollution data are matched to the death record based on the place of death, which may introduce measurement errors due to moving and attenuated effects.

Overall, it takes approximately 5 years for ozone pollution to cause fatal respiratory illnesses. The ozone-induced damage effects would be insufficient to lead to fatal illness if the exposure window was set considerably short; the estimated effects would shrink if the exposure window was set considerably long.

6.2 Heterogeneity analyses

We assess the heterogeneous effects of long-term ozone exposure across various diseases and different demographic characteristics to identify the susceptible diseases and the vulnerable population groups.

6.2.1 Effects across diseases and mortality risks

In this section, we estimate the baseline model across different types of diseases to explore whether long-term ozone exposure has heterogeneous effects on mortality from various respiratory diseases. We focus on pneumonia and chronic obstructive pulmonary diseases for two reasons, as they are representatives of acute and chronic respiratory diseases, respectively.

Col. (1)–(2) of Table 3 present estimation results of long-term ozone exposure on mortality from pneumonia and chronic obstructive pulmonary diseases, respectively. The various estimates on pneumonia and chronic obstructive pulmonary diseases

Table 3 Effects across diseases and mortality risks

	Pneumonia	COPD	Placebo tests				
			Gastro	Suicide	Diabetes	Falls	Drowning
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel A: maximum 8-h ozone							
Max eight-hour ozone	0.059*** (0.015)	0.009*** (0.004)	0.012 (0.010)	- 0.012 (0.019)	- 0.019 (0.026)	0.010 (0.007)	- 0.001 (0.002)
Observations	254,120	254,120	254,120	254,120	254,120	254,120	254,120
Weather controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes
KP F-statistics	2818	2818	2818	2818	2818	2818	2818
Panel B: 12-h ozone							
12-h ozone	0.065*** (0.017)	0.010*** (0.005)	0.013 (0.011)	- 0.013 (0.020)	- 0.020 (0.029)	0.011 (0.007)	- 0.001 (0.002)
Observations	254,120	254,120	254,120	254,120	254,120	254,120	254,120
Weather controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes
KP F-statistics	2511	2511	2511	2511	2511	2511	2511
Panel C: 24-h ozone							
24-h ozone	0.063*** (0.016)	0.009*** (0.005)	0.013 (0.010)	- 0.012 (0.020)	- 0.020 (0.028)	0.010 (0.007)	- 0.001 (0.002)
Observations	254,120	254,120	254,120	254,120	254,120	254,120	254,120
Weather controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes
KP F-statistics	4319	4319	4319	4319	4319	4319	4319

Col. (1)–(7) report the estimated effects of 5-year ozone concentrations on the mortality rates from pneumonia, chronic obstructive pulmonary diseases, gastrointestinal diseases, suicide, diabetes, falls, and drowning (in deaths per 100,000), respectively. All columns use ozone from upwind counties in the range of 100 ~ 300 km as the instrumental variable. The primary explanatory variables are maximum 8-h ozone concentrations (in panel A), 12-h ozone concentrations (in panel B), and 24-h ozone concentrations (in panel C). The weather controls include the quadratics of maximum temperature, relative humidity, precipitation, solar radiation, maximum wind speed, wind direction, and surface atmospheric pressure. The fixed effects include the county fixed effects, state-by-month fixed effects, and year-by-season fixed effects. Standard errors are clustered at the state level

* $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$

lead us to cautiously conclude that, although long-term ozone exposure increases mortality from both acute and chronic respiratory diseases, the effect may be more evident for patients with acute respiratory diseases. As this study pioneers the examination of the causal effects of long-term ozone exposure on respiratory mortality by various diseases, we do not know the exact mechanisms by which long-term ozone exposure affects patients with acute and chronic respiratory diseases differently. Considering that we do not have longitudinal hospitalization data at the individual level, we leave the exact mechanism for future research.

We also conduct several placebo tests. Specifically, we examine the effects of long-term ozone exposure on mortality rates from gastrointestinal diseases, suicide,

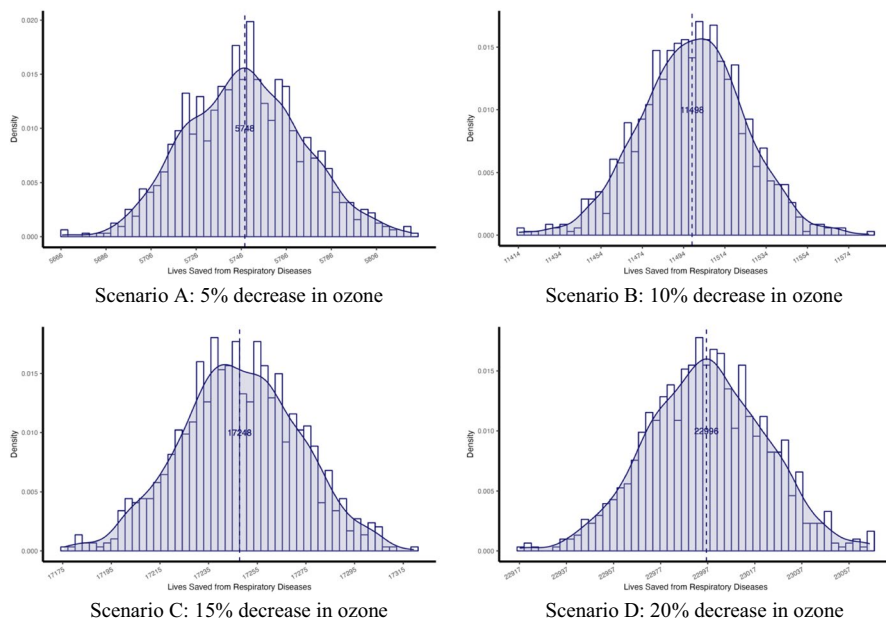


Fig. 3 Predicted lives saved from respiratory diseases (24-h ozone). Notes: This figure depicts the predicted lives saved from respiratory diseases driven by surface-level 24-h ozone under four different scenarios using bootstrap simulations. Scenario A is when ozone decreases by 5%; scenario B is when ozone decreases by 10%; scenario C is when ozone decreases by 15%; scenario D is when ozone decreases by 20%. Predictions under each scenario are bootstrapped 1000 times. The x-axis denotes lives saved from respiratory diseases. The y-axis denotes the density within each bootstrapping predicted deaths bin

diabetes, falls, and drowning, as they are arguably not directly affected by ozone pollution. Thus, the estimated effects of long-term ozone exposure on these diseases or mortality risks would not be statistically significant if our specification had validly isolated the effect of ozone pollution from other determinants of the mortality rate. As shown in Col. (3)–(7) of Table 3, consistent with our expectation, the estimated effects of all three measurements of ozone pollution on these diseases or mortality risks are not statistically significant, thus, further validating our specification.

6.2.2 Effects across age and sex groups

We investigate the heterogeneous effects across age-by-sex groups by assessing the impact of ozone pollution over the 5-year exposure window on respiratory mortality separately for each age-by-sex group. Figure A3 shows the findings that long-term ozone exposure only has impacts on the most vulnerable groups (i.e., the older adults aged 65–79 years and 80 years or over). Specifically, a one-standard-deviation rise in ozone levels over the preceding 5 years raises the respiratory mortality for males aged 80 years or above, females aged 80 years or above, and males aged 65–79 years by 0.129–0.137, 0.118–0.123, and 0.033–0.036 standard deviations, respectively.

As shown in Fig A3, males aged 80 years or above, females aged 65–79 years, and males aged 65–79 years are the only groups who are significantly affected by ozone pollution; the effects for all other groups are not statistically significant. A plausible reason for the difference across age groups is that older people have a weaker body condition compared to younger age groups, and thus have less tolerance to ozone pollution.

7 Extended analyses

7.1 Mechanism analysis

While the CDC's Multiple Cause-of-Death Mortality dataset does not provide information on behaviors and chronic health conditions prior to death, we are nonetheless interested in understanding how ozone contributes to mortality through these behavioral and physiological mechanisms. To explore this, we incorporate data from the Behavioral Risk Factor Surveillance System (BRFSS),¹⁷ a well-established, health-focused telephone survey. The outcome variables of interest include use of preventive services (e.g., receipt of flu and pneumonia shots), respiratory and related health conditions (e.g., diagnosis of asthma, stroke, or heart attack), and overall health status (e.g., whether poor physical or mental health limited individuals' ability to perform usual activities). Because county FIPS codes in the BRFSS are only available for data prior to 2012, we restrict our sample to pre-2012 observations. The data is aggregated to the county-month level to maintain consistency with the baseline specification, and other model specifications align with the 2SLS baseline model.

Table B8 presents the results of the mechanism analysis. Col. (1)–(2) show that long-term ozone exposure does not significantly affect the proportion of individuals who received flu or pneumonia shots in the past year. This is most likely because ozone is an invisible pollutant that people are often unaware of. Consequently, an increase in ozone concentrations does not appear to prompt greater use of preventive services, such as flu or pneumonia vaccinations.

Col. (3)–(6) indicate that long-term ozone exposure leads to adverse health outcomes. Specifically, a one-ppb rise in ozone levels over the preceding 5 years significantly raises the proportion of individuals diagnosed with asthma, stroke, and heart attack by 0.9–1.4, 0.5–0.8, and 0.6–0.9 percentage points, respectively. Regarding overall health conditions, a one-ppb rise in ozone levels significantly increases the proportion of individuals whose poor health prevents them from performing usual activities by 1.2–1.9 percentage points. These findings suggest that chronic health conditions are a likely mechanism through which long-term ozone exposure contributes to mortality from respiratory diseases.

¹⁷ The BRFSS dataset is available at https://www.cdc.gov/brfss/annual_data/annual_data.htm.

7.2 Welfare analysis

In this section, we predict lives saved from respiratory diseases under four different scenarios using bootstrap simulations. Specifically, we fit Eq. (3) and bootstrap 1000 times for predictions by preserving the distribution of ozone concentrations while reducing the mean by 5, 10, 15, and 20%, respectively. Figure 3 presents the predicted saved lives from respiratory diseases using bootstrap simulations, indicating that a 5, 10, 15, and 20% reduction in ozone concentrations can save 5748, 11,498, 17,248, and 22,996 lives, respectively, from respiratory diseases per year in the USA. By converting to the value of a statistical life (VSL),¹⁸ the respiratory health benefits from a 10% reduction in ozone pollution will be \$107.85–114.74 billion annually. Given that our estimates suggest that older adults disproportionately suffer from ozone-induced deaths, we follow an established method to further discount the VSL by 30% to take this age heterogeneity into account (Fan et al. 2020). The annual health benefits hence become \$75.50–80.32 billion, which accounts for 0.35–0.37% of the 2019 US GDP.

7.3 Evidence from COVID-19

The COVID-19 pandemic has given rise to the largest economic and public health crisis in the past few decades. While the existing epidemiological literature has focused on the contemporaneous effects of ozone exposure on mortality from COVID-19 (Isphording and Pestel 2021; Persico and Johnson 2021), none of the studies to our knowledge assesses the effects of long-term ozone exposure. If long-term ozone exposure prior to the pandemic significantly contributes to the deaths from COVID-19, an acute infectious disease, then our speculation about the mechanism through which long-term ozone exposure acts on the respiratory system may be further corroborated, as pre-pandemic exposure could not play a role in virus transmission. The most plausible channel through which long-term ozone exposure contributes to the deaths from COVID-19 is through medical pre-conditions, resulting from persistently heightened systemic inflammation induced by ozone. To prepare for future pandemics, it is important to understand how ozone pollution worsens the adverse effects on respiratory health that poses threats to human lives through SAS-COV-2, which will be discussed in this section.

Using the following 2SLS model, we examine the causal effect of ozone pollution over the 5-year exposure window on deaths per 100,000 from COVID-19:

$$Ozone_{cym} = \beta_0 + \beta_1 Ozone_{IVcym} + X_{cym}\omega + W_{cym}\varphi + \gamma_c + \delta_{sm} + \theta_{yz} + \mu_{cym}, \quad (5)$$

¹⁸ VSL refers to people's willingness to pay to reduce the dying risk. We obtain VSL in 2019 from EPA and USDA. For more information about the VSL from EPA, see <https://www.epa.gov/environmental-economics/mortality-risk-valuation>. Note that the VSL from EPA is 7.4 million dollars in 2006, equivalent to approximately 9.38 million dollars in 2019 after adjusting for inflation. For more information about the VSL from USDA, see <https://www.ers.usda.gov/data-products/cost-estimates-of-foodborne-illnesses/>.

Table 4 Effects of ozone pollution on mortality from COVID-19

	SARS-CoV-2 deaths per 100,000		
	(1)	(2)	(3)
Ozone concentrations	2.800*** (0.946)	2.871*** (0.968)	2.367*** (0.793)
KP F-statistics	204.1	219.9	227.5
Observations	43,685	43,685	43,685
County FE	Yes	Yes	Yes
State-month FE	Yes	Yes	Yes
Year-season FE	Yes	Yes	Yes
Weather controls	Yes	Yes	Yes
Other controls	Yes	Yes	Yes

This table reports the 2SLS estimates of 5-year ozone concentrations on SARS-CoV-2 deaths per 100,000. The instrumental variable is ozone from upwind counties in the range of 100 ~ 300 km. The dependent variable is SARS-CoV-2 deaths per 100,000. The primary explanatory variables are maximum 8-h ozone concentrations, 12-h ozone concentrations, and 24-h ozone concentrations in Col. (1)–(3), respectively. The weather controls include the quadratics of maximum temperature, relative humidity, precipitation, solar radiation, maximum wind speed, wind direction, and surface atmospheric pressure. Other controls include the SARS-CoV-2-confirmed rate, vaccination rate, the indicator for stay-at-home order, and the indicator for income support. We include the county fixed effects, state-by-month fixed effects, and year-by-season fixed effects in this table. Standard errors are clustered at the state level

*** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

$$Y_{cym} = \beta_0 + \beta_1 \widehat{Ozone}_{cym} + X_{cym}\omega + W_{cym}\varphi + \gamma_c + \delta_{sm} + \theta_{yz} + \varepsilon_{cym}. \tag{6}$$

In this model, Y_{cym} denotes the deaths per 100,000 from COVID-19 at county c in month m and year y .¹⁹ X represents a set of variables related to the pandemic, including the COVID-19 infection rate, COVID-19 vaccination rate, and policies in response to COVID-19.²⁰ All other variables follow Eqs. (2) and (3).

Table 4 presents the estimated effect of ozone exposure in the preceding 5 years on deaths per 100,000 from COVID-19. The estimates indicate that an additional ppb of average ozone concentrations in the preceding 5 years significantly increases 2.367–2.871 deaths per 100,000 per month from COVID-19. A back-of-envelope calculation shows that 7856 deaths per month could have been avoided among the US population in the pandemic if the average concentrations of 24-h ozone in the preceding 5 years decreased by 1 ppb.

¹⁹ We obtain nationwide COVID-19 infection rate, mortality rate, and vaccination rate data in 2020 and 2021 from the CDC.

²⁰ The set of policies includes the indicator for stay-at-home order and the indicator for income support. We obtain the data from the Center for Systems Science and Engineering at Johns Hopkins University.

8 Conclusions

In light of the low public awareness of ozone pollution and the potential health threats posed by long-term ozone exposure, this study quantifies the causal effects of long-term ozone exposure on respiratory mortality, utilizing exogenous changes in ozone concentrations induced by ozone originating from upwind counties. We demonstrate that an increase in the average concentrations of ozone in the preceding 5 years significantly increases respiratory mortality. If ozone concentrations decreased by 10%, 11,498 deaths from respiratory diseases would be avoided annually, which is equivalent to a VSL of \$75.50–80.32 billion.

Further analyses look into how long-term ozone exposure affects respiratory health. First, by varying the exposure window from half a year to five and a half years, our estimates suggest that several years of exposure to ozone pollution are necessary to lead to deaths from respiratory diseases. Second, evidence from heterogeneity analyses suggests that long-term ozone exposure increases mortality from both acute and chronic respiratory diseases and has more significant effects on vulnerable groups. Third, our extension analysis on COVID-19 indicates that, if ozone levels had been decreased by 1 ppb in the preceding 5 years, 7856 deaths per month could have been avoided among the US population in the pandemic.

The policy and research implications are as follows. First, our estimates suggest that policymakers should focus on vulnerable populations (e.g., older adults), raising awareness of ozone pollution to the same level as for particulate matters and establishing a stricter ozone pollution standard. This would reduce the socioeconomic costs resulting from long-term ozone exposure and prevent numerous deaths from respiratory diseases. Second, further studies could examine the exact mechanism by which long-term ozone exposure affects respiratory health when a more detailed longitudinal hospitalization dataset is available. This would provide more precise guidance on avoidance behaviors among vulnerable populations. Third, although we focus on respiratory diseases, our empirical strategies could be generalized to other diseases (e.g., cognitive disorders) that might be affected by long-term ozone exposure. We leave this work for future studies.

A limitation of this study is that we assumed that the deceased had been living in their county of residence for the entire 5 years prior to death. Owing to the nature of our dataset, we could not identify whether the deceased resided outside the area during the 5-year exposure window. As a result, the effects we have estimated may be understated, and our findings are more likely the lower bounds of the long-term ozone exposure effects on respiratory mortality. However, because ozone is an invisible pollutant, it is less likely to be a major source of concern for travel or immigration. People only respond to air pollution when they can perceive it, and the invisibility of ozone mitigates this issue.

Supplementary Information The online version contains supplementary material available at <https://doi.org/10.1007/s00148-025-01110-6>.

Acknowledgements The first authorship is shared equally by Ziheng Liu and Xi Chen. The authors thank the editor and two anonymous referees for their constructive comments and suggestions. The authors acknowledge helpful comments by participants and discussants at various conferences, seminars, and

workshops. The authors acknowledge the confidential Multiple Cause-of-Death Mortality dataset collected by the US National Center for Health Statistics (NCHS), Surface-level ozone data and other sources of weather and pollution information from the US Environmental Protection Agency (EPA), and the European Centre for Medium-Range Weather Forecasts (ECMWF).

Funding Qinan Lu acknowledges financial support from the National Natural Science Foundation of China (NSFC) under Project No. 72403240. Xi Chen acknowledges funding from the Yale PEPPEER Center Scholar Award (P30AG021342), two NIH/NIA Grants (R01AG077529; K01AG053408), and Yale Tobin Faculty Research Award.

Data availability Death data from the US National Center for Health Statistics (NCHS) are confidential in nature and cannot be shared by agreement with the NCHS to ensure that the identity of individuals and establishments cannot be disclosed.

Declarations

Conflict of interest The authors declare no competing interests.

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