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DISCUSSION PAPER SERIES

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ABSTRACT

Blowin' in the Wind of an Invisible Killer: Long-Term Exposure to Ozone and Respiratory Mortality in the United States

In light of the low public awareness of ozone 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 based on the long-distance transmission of ozone from upwind neighbor counties, we discover that an increase of one standard deviation in the average concentrations of ozone in the preceding five years increases respiratory mortality by 0.062–0.066 standard deviations. The findings indicate that long-term ozone exposure increases mortality from both acute and chronic respiratory diseases and has significant adverse effects on vulnerable groups. Furthermore, we discover that the respiratory mortality rate responds to long-term ozone exposure nonlinearly, and that there is a critical threshold at which the adverse effects of ozone exposure commence. Our bootstrap simulation results suggest that if ozone concentrations in the preceding five years decrease by 10 percent, 11,391 deaths from respiratory diseases could be avoided in the United States annually, with resulting health benefits valued at around $106.85–113.67 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.

JEL Classification: I15, J14, Q51, Q53

Keywords: long-term exposure, ozone, respiratory diseases, nonlinear responses, health benefits, COVID-19

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1 Introduction

The coronavirus disease 2019 (COVID-19) pandemic ravaging the world prompted renewed attention to respiratory health. Prior to the pandemic, two out of the ten leading causes of death in the United States were attributed to respiratory diseases (Heron, 2021). According to the *Multiple Cause-of-Death Mortality* dataset,[1] approximately 260,000 lives[2] were lost annually because of respiratory diseases from 2008 to 2019 in the US. This situation was aggravated after the coronavirus outbreak: in the US, more than a million individuals lost their lives because 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; Wang et al., 2022). 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), surface-level ozone pollution is invisible (Kong et al., 2022; Wang et al., 2022). Public awareness of ozone is low due to its invisibility, and protective measures preventing ozone inhalation have been ignored (Wang et al., 2022). Additionally, because ozone has a characteristic pleasant odor, the "fresh air" in common perception 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.

The contemporaneous effects of ozone exposure on respiratory diseases are well established: 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 the resolution of edema and inflammation, cell proliferation, and tissue remodeling (González-López and Albaiceta, 2012). The acute damages caused by ozone pollution may lead to a contemporaneous increase in hospitalizations for respiratory illness (Neidell, 2004; Janke, 2014).

[1] See section 4.1 for more detailed information.
[2] Deaths from respiratory diseases account for 9.81% of the total death toll.

However, studies examining the long-term effects of ozone exposure on respiratory health 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). 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). Moreover, studying the long-term effects of ozone exposure is more consistent with real-world experiences, as we are continuously exposed to ozone as we breathe.

Identifying the causal effect of long-term ozone exposure on respiratory mortality is challenging owing to the potential for classical measurement errors and omitted variable bias. For example, as the precursors of ozone, NO_x and volatile organic compounds (VOCs) correlate with numerous economic confounders, such as industrial and vehicle emissions, which are associated with regional income (Anenberg et al., 2017; Gaudel, 2022), determining the quantity and quality of health facilities. Additionally, the potential for classical measurement errors emanates 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; Wang et al., 2022).

We address these empirical challenges and identify the causal effect of long-term ozone exposure on respiratory mortality by employing ozone transferred from upwind neighbor counties within a certain distance range as an instrumental variable (IV). This approach is based on the scientific 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, ozone transferred from upwind neighbor counties within a certain distance range influences local respiratory mortality only through its impact on local ozone levels. We also flexibly control for weather characteristics and include the county, state-by-month, and season-by-year fixed effects to control for county-specific characteristics, the seasonal association between ozone pollution and respiratory health varying by state, 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 one standard deviation in the average concentration of ozone in the preceding five years increases respiratory mortality by 0.062–0.066 standard deviations. Consistent with expectations, such effects are not significant if the exposure window[3] is set to be overly short (i.e., less than three years), suggesting that several years of exposure to ozone pollution is necessary to cause deaths from respiratory diseases. Our bootstrap simulation results suggest that if ozone concentrations in the preceding five years decreased by 10 percent, 11,391 deaths from respiratory diseases could be avoided annually, and the respiratory health benefits would be $106.85–113.67 billion, which account for 0.50–0.53 percent of the 2019 US GDP.

We then scrutinize the nonlinear effects of long-term ozone exposure on respiratory mortality by fitting a step function. We discover that mortality responds to ozone pollution nonlinearly: the step function plot shows that the critical threshold where ozone significantly increases respiratory mortality is 31 ppb. We also study the heterogeneous effects of long-term ozone exposure on respiratory mortality across various diseases and age-by-sex groups. We discover that ozone pollution in the preceding five years increases mortality from either acute or chronic illness, and that ozone has considerably greater effects on the elderly and males.

This study contributes to the literature in three respects. First, whereas some epidemiological studies find that long-term ozone exposure correlates to 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 lacking, and there is no consensus about the duration of the exposure window over which ozone pollution affects health in the long term. To the best of our knowledge, this study is the first to employ a causal inference framework to estimate the impacts of long-term ozone exposure on respiratory health outcomes. By constructing an IV based on the long-distance transmission of ozone from upwind neighbor counties, our 2SLS estimates suggest that OLS estimates can underestimate deaths from respiratory diseases caused by ozone pollution. We discover that exposure to ozone pollution over several years leads to deaths from respiratory diseases by varying the exposure window from half a year to six years and letting the data determine the appropriate duration.

Second, this study contributes to the literature that assesses the nonlinear health effects of air pollutants (Chen et al., 2018; Jans et al., 2018). Numerous studies have examined the health

[3] The duration over which ozone pollution affects mortality from respiratory diseases.

effects of air pollutants; however, only a few studies estimate the nonlinear responses of health outcomes to air pollution, specifically ozone. This study is among the first to scrutinize the nonlinear impacts of ozone pollution on health outcomes (Mullins, 2018; Marcus, 2021; Wang et al., 2022). We visually show a critical threshold above which the detrimental effect of ozone commences by the innovation of introducing the step function commonly employed by the literature on environmental stressors (Schlenker and Roberts, 2009; Cui, 2020).

Third, this study contributes to the emerging literature estimating the effects of pollution on COVID-19, which is 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; Isphording 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, which suggests that 28,706 deaths per month could have been avoided in the US population in the pandemic if the average concentrations of ozone in the preceding five years had decreased by one standard deviation.

The remainder of this paper is organized as follows. Section 2 presents the background of surface-level ozone pollution and respiratory diseases in the US. Section 3 describes the empirical models used in this study. Further, Section 4 introduces data and provides summary statistics, and Section 5 presents the estimation results. Section 6 presents robustness checks and heterogeneity analyses. Section 7 presents the extension analyses and, finally, Section 8 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; Wang et al., 2022). VOCs and NO_x are emitted from both industrial (i.e., gasoline and power plants) and natural sources (i.e., deciduous trees) (Auffhammer and Kellogg, 2011). In regions where VOC levels are high, ozone formation is determined by NO_x concentrations, the so-called "NO_x-limited," which is generally located in rural areas. In regions where NO_x concentrations are relatively high, ozone formation is determined by VOC concentrations, the so-called "VOC-limited," which is mostly located in

urban areas (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 long distance (Brankov et al., 1998; Wang et al., 2022).

A series of regulations have been made because ozone pollution adversely impacts labor productivity (Wang et al., 2022) and human health (Neidell, 2004; Currie, 2009). NO_x becomes the regulation target because ozone cannot be directly controlled by regulation (Deschenes et al., 2017) and VOC emissions are mostly from biogenic sources (Auffhammer and Kellogg, 2011). Efforts targeted at reducing NO_x in the U.S. include the Reid vapor pressure regulation, the Federal reformulated gasoline, and the 1990 Clean Air Act Amendments (Henderson, 1996; Auffhammer and Kellogg, 2011; Walker, 2013). VOC concentrations become relatively high compared to NO_x because of such regulations; therefore, most parts of the U.S. are currently in a NO_x-limited condition (Jung et al., 2022).

2.2 Respiratory Diseases

Two out of ten leading death causes in the U.S. 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).

3 Empirical Strategy

3.1 Panel-Fixed Effects Model

Based on the panel structure of our data sample, we estimate the impacts of long-term ozone exposure on respiratory mortality using the following fixed effects model:

$$Y_{cym} = \beta_0 + \beta_1 Ozone_{cym} + \boldsymbol{W}_{cym}\varphi + \gamma_c + \delta_{sm} + \theta_{yz} + \varepsilon_{cym} \quad (1)$$

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 eight-hour, 12-hour, and 24-hour ozone over a certain time duration, which account for maximum, daytime, and all-day exposures to surface-level ozone, respectively. No consensus about the duration over which ozone pollution may affect health in the long term has been reached (EPA, 2020; Wang, 2022). Following Deschenes et al. (2020), we vary the time window from half a year to six years and let the data ascertain the appropriate exposure window, rather than having a priori assumed duration over which ozone causes deaths from respiratory diseases. Figure 3 shows the primary coefficients of interest for all exposure windows varying from half a year to six years. As explained later in Section 6.1.8, a five-year duration is an appropriate exposure window and is employed here. For example, for the deceased in county A in December 2010, we employ the average ozone concentrations from January 2006 to December 2010 for that county.

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 $\boldsymbol{W}_{cym}$, a set of weather conditions over the same five-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. Following Deschenes et al. (2020), we further control for the quadratic polynomials of the aforementioned weather characteristics to model their nonlinear effects on respiratory health.

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 ozone and respiratory health. 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), and the significance levels of our estimates are not sensitive to alternative clustering settings; we discuss this in Section 6.1.2.

3.2 2SLS Estimation

Although the fixed effects strategy, in conjunction with the rich weather controls, alleviates the endogeneity concern of our specification, we cannot conclusively rule out the possibility for omitted-variable bias, which poses a challenge to identifying such a causal effect. As introduced in Section 2.1, NOx 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; Deschenes et al., 2020; Wang et al., 2022).

To address the endogeneity concern stemming from the omitted variable bias and classical measurement errors, we construct an IV based on the scientific evidence that ozone can be transferred by wind over a long distance (Brankov et al., 1998; Wang et al., 2022), which is a property similar to particulate matters (Schlenker and Walker, 2016; Chen et al., 2021). Specifically, 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 good predictor of local ozone concentrations. Figure 1 shows the construction procedure of the IV. The triangle denotes a focal county, and the circles denote neighbor counties at distances of 100-300 km from the focal county. Only the counties within this radius band of 100-300 km are considered when computing the ozone transferred from upwind counties for a focal county. We set the radius band between 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 downwind 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 good predictor of ozone concentrations in the downwind focal county. We set the radius band as 150-300 km in Section 6.1.3, and our estimates remain robust.

As shown in Figure 1, we define α as the angle between wind flow and the east direction, and we define θ as the angle between the east direction and the vector connecting a neighbor

county centroid and a focal county centroid. Among the three neighbor counties in the 100-300 km radius band in Figure 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, following Wang et al. (2022), we assign a weight

$$w_{nc,t} = \frac{\frac{cos(\theta_{nc,t}-\alpha_{nc,t})}{d_{nc}} \cdot \mathbf{1}\{cos(\theta_{nc,t}-\alpha_{nc,t})>0\}}{\sum_{k=1}^{m} \frac{cos(\theta_{kc,t}-\alpha_{kc,t})}{d_{kc}} \cdot \mathbf{1}\{cos(\theta_{kc,t}-\alpha_{kc,t})>0\}}$$ [4]

for each neighbor county n within the 100-300 km radius band. Using the assigned weight $w_{nc,t}$, we then compute the weighted average of upwind ozone from all neighbor counties n within the 100-300 km radius band for focal county c. Finally, we sum the ozone concentrations from upwind neighbor counties in the preceding week (Wang et al., 2022) because the long-range transmission of ozone pollution is time-consuming.

Equation (1) is rewritten after instrumenting for ozone concentrations:

$$Ozone_{cym} = \beta_0 + \beta_1 Ozone_{IV,cym} + \boldsymbol{W}_{cym}\varphi + \gamma_c + \delta_{sm} + \theta_{yz} + \mu_{cym} \quad (2)$$

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

Equations (2) and (3) are the first and second stages, respectively, of the 2SLS model. 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, ozone from the upwind neighbor counties can affect the respiratory health of residents living in the focal county only through its impact on focal county ozone concentrations. Averaged to the same exposure window as $Ozone_{cym}$ and weather characteristics, $Ozone_{IV,cym}$ denotes the five-year average of ozone from upwind neighbor counties within the 100-300 km radius band of county c in the preceding week. All other variables follow Equation (1).

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] d_{nc} refers to the distance between the centroid of neighbor county n and the centroid of focal county c.

4.1 Deaths from Respiratory Diseases

We obtain respiratory death data from the *Multiple Cause-of-Death Mortality* dataset collected by the National Center for Health Statistics[5]. This dataset is the most comprehensive nationwide dataset that reports all deaths occurring in the US. The dataset contains information on the underlying death causes, age, sex, and county of residence. We aggregate the total number of resident deaths from respiratory diseases, identified by the underlying causes of deaths with ICD-10[6] codes J00-J99, to the county-month level between 2008 and 2019. Furthermore, 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. Additionally, the demographic data were obtained from the National Cancer Institute[7]. 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. The right plot of Figure A1 shows respiratory mortality from 2008 to 2019, suggesting that the respiratory mortality rate has been gradually increasing in the past few years.

4.2 Surface-Level Ozone and Other Air Pollutants

Our core analysis focuses on ground-level daily maximum eight-hour, 12-hour, and 24-hour ozone. We obtain hourly ozone data from the Environmental Protection Agency (EPA) of the US. The left plot of Figure A1 depicts ozone concentrations from 2003 to 2019, showing the relative concentrations of the three measurements of ozone. Following Zhang et al. (2017), Qiu et al. (2020), and Chen and Gong (2021), we interpolate ozone concentrations of all monitoring stations located less than 100 km from each county centroid using the inverse-distance-weighted (IDW) method. We then average the ozone concentrations to the five-year exposure window for each county.

The other criteria pollutants that we further control for in the robustness check include carbon monoxide (CO), particulate matters ($PM_{2.5}$, PM_{10}), sulfur dioxide (SO_2), and nitrogen

[5] See https://www.cdc.gov/nchs/data_access/ftp_data.htm for further information.
[6] ICD-10 refers to the International Classification of Diseases, 10th Revision.
[7] See https://seer.cancer.gov/popdata/download.html for further information.

dioxide (NO_2). We obtain $PM_{2.5}$ data from EPA and retrieve data of CO, PM_{10}, SO_2, and NO_2 from the fourth-generation European Centre for Medium-Term Weather Forecasting (ECMWF) global reanalysis of atmospheric composition (EAC4)[8]. We interpolate their concentrations of all grid cells located within 100 km of each county centroid using the same aforementioned IDW method.

Studies relying on the reanalysis data from ECMWF include Cai et al. (2016), Giaccherini et al. (2021), and Wang et al. (2022). We employ the reanalysis data because the geographic coverage of EPA data for CO, PM_{10}, SO_2, and NO_2 is considerably smaller compared to O_3 and $PM_{2.5}$. If we used the EPA data for CO, PM_{10}, SO_2, and NO_2, a large number of counties would be excluded from our data sample. Considering that we aim to conduct a nationwide analysis, a considerably smaller sample size would restrict our analysis and findings to a significantly less scale. To evaluate the validity of the ECMWF reanalysis data, we obtain monitoring stations data of CO, PM_{10}, SO_2, and NO_2 from the EPA and plot them in Figure A2. As shown in Figure A2, data from both sources follow a similar trend and are quite close in magnitude.

4.3 Weather Characteristics

We control for a series of weather characteristics because 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[9] on a grid of 0.25° × 0.25°. We interpolate weather characteristics to each county using the aforementioned IDW method and aggregate to the five-year exposure window. Referring to Deschenes et al. (2020), we control for the quadratic polynomials of all the weather characteristics to account for the potential nonlinear effects of weather.

4.4 Summary Statistics

[8] The EAC4 dataset reports every 3-hour on a grid of 0.75° × 0.75°. See Inness et al. (2019) for further information.
[9] The name of the dataset is the fifth-generation ECMWF reanalysis data (ERA5). See Hersbach et al. (2019) for further information.

Covering 2633 counties[10] from 2008 to 2019, the final data sample used in the analysis consists of 254,120 observations at the county-by-month level. The primary explanatory variables are daily maximum eight-hour, 12-hour, and 24-hour 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 five-year exposure window.

Table 1 presents the summary statistics from our final data sample. The average of our dependent variable, the monthly deaths from respiratory diseases, is 8.109 per 100,000. The average of daily maximum eight-hour, 12-hour, and 24-hour ozone over the five-year exposure window, our primary explanatory variables, are 38.801 ppb, 34.707 ppb, and 30.618 ppb, respectively. Our IV, the average of one-week cumulative ozone concentrations from upwind neighbor counties over the five-year exposure window, is 200.761 ppb.

5 Results

5.1 First-Stage Estimates: Effects of Ozone from Upwind Neighbor Counties on Ozone in the Focal County

Table 2 presents the estimated effects of ozone from upwind neighbor counties in the 100-300 km radius band (the constructed IV) on ozone concentrations in the focal county. First, we discover that upwind ozone from neighbor counties is a good predictor of ozone concentrations in the focal county: the estimated coefficients are statistically significant at 1% for daily maximum eight-hour, 12-hour, and 24-hour ozone, which demonstrates that the instrument relevance criterion is fulfilled. The estimates suggest that an additional ppb of ozone from upwind neighbor counties increases ozone concentrations in the focal county by 0.019, 0.017, and 0.018 ppb, respectively; thus, reassuring the validity of our IV. Additionally, the Kleibergen-Paap F-statistics for all three measurements of ozone are far above the Stock–Yogo threshold value of 16.38 (Stock and Yogo, 2005), suggesting that our constructed IV does not involve a weak instrument problem.

[10] The overlapping counties of all data sources.

5.2 Effects of Long-Term Ozone Exposure on Respiratory Mortality

Panel A of Table 3 presents results from OLS estimates. Col. (1) – (3) show that an additional ppb in the average concentrations of ozone in the preceding five years increases 0.030-0.035 deaths per 100,000 from respiratory diseases. They are statistically not significant and smaller in magnitude compared to their 2SLS counterparts for two reasons. First, the exact home address of the deceased is unknown, and the accurate ozone concentrations level is unidentified as we could only identify the residence county of the deceased and we used county-level ozone pollution data constructed from fixed monitors. Thus, the OLS estimates underestimate the impact of ozone pollution on respiratory mortality because of these classical measurement errors (Schlenker and Walker, 2016; Deschenes et al., 2020; Wang et al., 2022). Second, the confounding factors correlated with both ozone and respiratory health; similarly, regional economic status could also underestimate the effect. For example, the quantity and quality of health facilities may be high in metropolitans where the concentrations of the precursors of the ozone are high, downward biasing the OLS estimates.

In line with our expectation, the 2SLS estimates correct the biases in OLS estimates; therefore, we focus on the 2SLS estimates for the remaining analyses. Panel B of Table 3 presents 2SLS estimates where ozone from upwind neighbor counties is employed as an IV for ozone concentrations. Col. (1) – (3) show that an additional ppb in the average concentrations of daily maximum eight-hour, 12-hour, and 24-hour ozone in the preceding five years increases deaths per 100,000 from respiratory diseases by 0.089, 0.097, and 0.094, respectively. Using a magnitude of standard deviations, the estimates suggest that one standard deviation increase in the average concentrations of daily maximum eight-hour, 12-hour, and 24-hour ozone in the preceding five years increases respiratory mortality by 0.063 ($= 0.089 \times \frac{4.483}{6.325}$), 0.066 ($= 0.097 \times \frac{4.327}{6.325}$), and 0.062 ($= 0.094 \times \frac{4.200}{6.325}$) standard deviations, respectively. These estimates are consistent with our expectations that while contemporaneous ozone pollution only increases hospitalizations from respiratory illness (Neidell, 2004; Janke, 2014), long-term ozone exposure can lead to more severe and dangerous respiratory diseases.

5.3 Comparison with the Literature

As this study is 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 concentrations of ozone in the preceding five years increases 0.094 deaths per 100,000 from respiratory diseases, which corresponds to a 1.2% 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 three years affects 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 affects 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 caused a 0.4% increase in the respiratory mortality over the period of 1982-2000. The difference in magnitude is consistent with our expectation because our estimates are more plausibly causal: estimates from the existing epidemiological literature should at least partially be interpreted as associational and might thus, suffer from attenuation bias and endogeneity issues.

6 Robustness and Heterogeneity of Effects

6.1 Robustness Checks

We verify the sensitivity of our estimates to alternative fixed effects, alternative clustering methods, alternative IV approaches, alternative weather controls, possible omitted air pollutants, possible omitted socioeconomic factors, and restrict the data sample to the east of the 100^{th} meridian. 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 4 and Table B1, which demonstrates the invariance and robustness of our estimates to different fixed effects. Col. (1) of Table 4 presents the baseline model, in which we include county, season-by-year, and state-by-

month fixed effects to control for county-specific characteristics, the state-varying seasonal association between ozone and mortality, and nationwide time-varying shocks. In Col. (2) of Table 4 and Col. (2) of Table B1, 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 B1, we replace the state-by-month fixed effects with state-by-season fixed effects as an alternative control for the state-varying seasonal association between ozone and mortality. In Col. (4) – (6) of Table B1, 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 4 and Table B1, these estimates are qualitatively similar to their baseline counterparts.

6.1.2 Alternative Clustering Methods

Table 4 and Table B2 report the estimates under alternative clustering methods of standard errors. Col. (1) of Table 4 presents the baseline estimates, allowing for autocorrelation in the errors within each state, with clustered standard errors at the state level. Col. (3) of Table 4 and Col. (2) of Table B2 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) of Table B2 uses two-way clustering at the state-by-year and county levels, which allows for the autocorrelation within each state-by-year cell and the autocorrelation within the same county across time. Col. (4) of Table B2 maintains the county clustering and replaces the state-by-year clustering with state-season-year clustering, restricting autocorrelation to the errors within a state-season-year cell. Col. (5) of Table B2 also maintains the county clustering but changes the state-by-year clustering to state-month-year clustering, further restricting the autocorrelation to the errors within a state-month-year cell. All these estimates remain robust compared to the baseline estimate.

6.1.3 Alternative Instrumental Variable

We employ alternative IVs as robustness checks in Table 4; Col. (1) of Table 4 presents the baseline estimate, which uses ozone from upwind neighbor counties in the 100-300 km

radius band as an IV. Setting the radius band considerably close to the focal county in a manner wherein ozone pollution from neighbor counties may share the same emission source as ozone pollution in the focal county raises a concern. To test the sensitivity of our results to the spatial range of IV, Col. (4) of Table 4 employs an alternative IV by excluding counties within 150 km of the focal county when constructing the IV. Additionally, rather than solely depending on wind direction, following Chen et al. (2021) and Wang et al. (2022), we employ another alternative IV in Col. (5) by also accounting for wind speed in the construction of the IV. This is because the long-distance transmission of ozone may reach the focal county faster, and may hence, contribute more to the ozone concentrations in the focal county when wind speed is higher. Specifically, we update the weight assigned to neighbor counties when computing the weighted average of ozone from them. The updated weight equation is

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

Overall, all estimates using the two alternative IVs are qualitatively similar to our baseline estimate.

6.1.4 Alternative Weather Controls

Table 4 and Table B3 report 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[11] in Col. (6) of Table 4 and with the quadratic polynomial of average temperature in Col. (7) of Table 4. We further remove all weather controls one at a time in Col. (2) – (8) of Table B3. Overall, all estimates under various specifications of weather controls are qualitatively similar to their baseline counterparts.

[11] Following Deschenes et al. (2020), we control for the number of days within each five-degree 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.

6.1.5 Controlling for Additional Air Pollutants

There may be a concern that other air pollutants could confound our estimates on ozone. To show that our estimates are not driven by omitted air pollutants, we include additional air pollutants, one at a time, in Col. (8) of Table 4 and in Table B4. We do not include them simultaneously due to the fact that air pollutants tend to be highly correlated, and the simultaneous inclusion of multiple air pollutants may cause multicollinearity (Maddison, 2005). Consequently, all estimates remain statistically significant at the 5% or 1% level and are similar in magnitude to the baseline estimates.

6.1.6 The 100th Meridian

There may be a concern that our baseline estimates are driven by counties west of the 100th meridian, as such counties are considerably different from counties east of the 100th meridian in climate conditions (Schlenker et al., 2006). For example, the 100th meridian is the 20-inch rainfall line in the US (Schlenker et al., 2006). Furthermore, as shown in Figure 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[12] in Col. (9) of Table 4, and our estimates in Col. (9) are qualitatively similar to the baseline estimates in Col. (1), suggesting that our results are not driven by counties west of the 100th meridian.

6.1.7 Controlling for Additional Socioeconomic Factors

Although we employ an instrumental variable based on the long-range transmission of ozone by wind force, our estimates may be sensitive to the inclusion of socioeconomic factors. To mitigate this concern, we include a series of socioeconomic variables in Table B5. Col. (2) – (4) of Table B5 add unemployment rate, average annual salary, and poverty rate[13] as control

[12] There are 358 counties west of the 100th meridian in our data sample.
[13] County-level data on unemployment rate, average annual salary, and poverty rate are obtained from the U.S. 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/wagesearnings. For more details about poverty rate, see https://www.census.gov/programs-surveys/saipe.html.

variables, respectively. Our estimates in Col. (2) – (4) are similar in magnitude to their baseline counterparts and remain statistically significant at the 5% level, which suggests that our estimates are not confounded by employment status, income level, or poverty. Col. (5) of Table B5 adds the ratio of population to primary care physicians[14] as a control variable, and our estimates in Col. (5) remain qualitatively similar to their baseline counterparts, showing that our estimates are not confounded by healthcare access.

6.1.8 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 six years. Recall that in the baseline specification, we specify the exposure window as five years. Following Deschenes et al. (2020), we plot the 2SLS estimates on ozone using Equation (3) by varying the exposure window from half a year to six years in Figure 3. Each dot denotes the coefficient of interest from a separate 2SLS estimation. The primary explanatory variables are daily maximum eight-hour ozone, 12-hour ozone, and 24-hour ozone, respectively, for the left, middle, and right plots in Figure 3.

Figure 3 shows the legitimacy of setting the exposure window as five years in our baseline specification. The estimates can be divided into three stages, as shown in Figure 3. First, when the exposure window is shorter than three years, the estimates of ozone pollution on respiratory mortality are not statistically significant and are close to zero. As explained in earlier, it takes time for the self-recovery mechanism of the respiratory system to be damaged by persistent and recurring inflammation (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 three and five years, the coefficients of interest are significantly positive, and their magnitude becomes larger when the exposure window enlarges. This suggests that the effects of ozone on respiratory diseases become more evident when the exposure time to ozone pollution becomes longer. This is

[14] 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.

consistent with our expectation; longer exposure to ozone pollution may induce more inflammation, which would destroy the self-recovery mechanism of the respiratory system (Perera et al., 2007). Third, when the exposure window exceeds five years, the estimated effects shrink as the exposure window becomes longer. The shrinkage is 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 could be matched to the resident deaths data based on the place of death, which may introduce measurement errors due to moving and attenuated effects. Overall, it takes approximately five years for ozone pollution to cause fatal respiratory system illnesses. The damages caused by ozone pollution would be insufficient to cause 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

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 influenza/pneumonia and chronic lower respiratory diseases for two reasons. First, they are among the top ten death causes in the US. (Heron, 2021). Second, they are representatives of acute and chronic respiratory diseases, respectively.

Col. (1) – (3) of Table 5 present estimation results of long-term ozone exposure on mortality from influenza/pneumonia. The estimates suggest that a one standard deviation increase in the average concentration of ozone in the preceding five years increases mortality from influenza/pneumonia by 0.136–0.144 standard deviations[15]. Col. (4) – (6) of Table 5 present

[15] A one standard deviation increase in the average concentrations of daily maximum eight-hour ozone, 12-hour ozone, and 24-hour ozone in the preceding five years increases mortality from influenza/pneumonia by 0.136

estimates of long-term ozone exposure on mortality from chronic lower respiratory diseases. The estimates suggest that a one standard deviation increase in the average concentrations of ozone in the preceding five years increases mortality from chronic respiratory diseases by 0.070–0.074 standard deviations[16].

The various estimates on influenza/pneumonia and chronic lower respiratory diseases 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 test the effects of long-term ozone exposure on gastrointestinal mortality in Col. (7) – (9) of Table 5 as a placebo test because gastrointestinal illness is presumably not related to air pollution (Chen et al., 2018). Thus, the estimated effects of long-term ozone exposure on gastrointestinal mortality 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. (7) – (9) of Table 5, consistent with our expectation, the estimated effects of all three measurements of ozone pollution on gastrointestinal mortality 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 five-year exposure window on respiratory mortality separately for each age-by-sex group. Figure 4 shows the findings; it suggests that 1) long-term ozone exposure only has impacts on the most vulnerable groups (i.e., the older ages of 65-79

(=0.091×4.483/3.006), 0.144 (=0.100×4.327/3.006), and 0.136 (=0.097×4.200/3.006) standard deviations, respectively.

[16] A one standard deviation increase in the average concentrations of daily maximum eight-hour ozone, 12-hour ozone, and 24-hour ozone in the preceding five years increases mortality from chronic respiratory diseases by 0.070 (=0.083×4.483/5.317), 0.074 (=0.091×4.327/5.317), and 0.070 (=0.089×4.200/5.317) standard deviations, respectively.

years and those of 80 years or over), and that 2) long-term ozone exposure has larger impacts on males than on females. Specifically, one standard deviation increase in ozone concentrations in the preceding five years increases the respiratory mortality for males aged 80 years or over, females aged 80 years or over, and males aged 65 to 79 years by 0.129–0.138[17], 0.114–0.123[18], and 0.033–0.034 standard deviations[19], respectively. As shown in Figure 4, males aged 80 years or over, females aged 65 to 79 years, and males aged 65 to 79 years are the only groups who are significantly affected by ozone pollution; the effects for all other groups are not statistically significant.

These differences across various age-by-sex groups are reasonable. First, 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. Second, the difference across sex could be due to the difference in time spent outdoors by males and females. Figure A3 plots time spent outdoors across age-by-sex groups from 2003 to 2019 using data from the American Time Use Survey (ATUS).[20] The blue bars denote time spent outdoors by males, and the red bars denote time spent outdoors by females. As shown in Figure A3, males spend longer time outdoors than females across all age groups, suggesting that males may suffer from more exposure to ozone pollution. Given that 1) we do not have longitudinal health data at the individual level and that 2) the sample size of ATUS data does not support analysis at the county level (US Bureau of Labor Statistics, 2018), the aforementioned reasons are only hypotheses, and we leave it for future research to test the exact mechanisms of such differences.

7 Extension Analyses

7.1 Nonlinear Responses

[17] One standard deviation increase in daily maximum eight-hour, 12-hour, and 24-hour ozone over the five-year exposure window increases respiratory mortality for males aged 80 or over by 0.130 (=5.676×4.483/195.831), 0.138 (=6.228×4.327/195.831), and 0.129 (=5.993×4.200/195.831) standard deviations, respectively.
[18] One standard deviation increase in daily maximum eight-hour, 12-hour, and 24-hour ozone over the five-year exposure window increases respiratory mortality for females aged 80 or over by 0.118 (=3.651×4.483/138.978), 0.123 (=3.947×4.327/138.978), and 0.114 (=3.777×4.200/138.978) standard deviations, respectively.
[19] One standard deviation increase in daily maximum eight-hour, 12-hour, and 24-hour ozone over the five-year exposure window increases respiratory mortality for males aged 65–79 by 0.033 (=0.381×4.483/51.055), 0.034 (=0.405×4.327/51.055), and 0.034 (=0.414×4.200/51.055) standard deviations, respectively.
[20] For more details, see https://stats.bls.gov/tus/.

There is a trend in the recent literature to illustrate a nonlinear response by fitting a step function (Schlenker and Roberts, 2009; Cui, 2020). In this section, we assess the nonlinear effects of long-term ozone exposure on respiratory mortality by plotting a step function that fits a separate point estimate for each one-ppb ozone bin. Specifically, we replace $Ozone_{cym}$ in Equation (1) with a set of one-ppb ozone bins[21]. Figure 5 shows the nonlinear effects of the average concentrations of 24-hour ozone on respiratory mortality in the preceding five years by plotting the step function. The dark blue line and the light blue band indicate the step function and 95% confidence band, respectively. Figure 5 shows that the critical threshold point is 31 ppb, and the point estimates can be categorized into two stages. When ozone concentrations are below 31 ppb, the effects of ozone on respiratory morality are not statistically significant. In contrast, when ozone concentrations exceed 31 ppb, ozone pollution begins to increase respiratory mortality significantly, showing evidence that ozone only leads to fatal respiratory diseases after reaching a certain concentration level. Furthermore, the step functions of daily maximum eight-hour ozone and 12-hour ozone are shown in Figure A4. They show a similar trend to the step function of 24-hour ozone in Figure 5, buttressing our finding that the detrimental effects of ozone exposure on respiratory mortality would commence only after reaching a certain threshold level.

7.2 Welfare Analysis

In this section, we predict deaths per 100,000 from respiratory diseases under four different scenarios using bootstrap simulations. Specifically, we fit Equation (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 6 presents the predicted deaths from respiratory diseases using bootstrap simulations, indicating that long-term ozone exposure results in 7.946, 7.823, 7.701, and 7.579 deaths per 100,000 per month from respiratory diseases when the five-year average of ozone concentrations are 95, 90, 85, and 80% of the current concentration level. These predictions suggest that a 5, 10, 15, and 20% reduction in ozone

[21] We employ a set of 1-ppb ozone dummies ranging from 25 to 39 ppb (25–26, 26–27, …, 38–39, 39+) where 25 ppb is the 5th percentile of 24-hour ozone concentrations and 39 ppb is the 95th percentile of 24-hour ozone concentrations.

concentrations can save 6,492, 11,391, 16,249, and 21,109 lives,[22] respectively, from respiratory diseases per year in the US. By converting to the value of a statistical life (VSL)[23], the respiratory health benefits from a 10% reduction in ozone pollution will be $106.85–113.67 billion[24] annually, which accounts for 0.50%–0.53% of the 2019 US GDP.

7.3 Evidence from COVID-19

The ongoing COVID-19 pandemic has given rise to the largest economic and public health crisis in the past few decades (Isphording and Pestel, 2021). It is expedient to adequately prepare for future pandemics by understanding how ozone pollution worsens the adverse effects on respiratory health that poses threats to human lives through SAS-COV-2.

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 assess the effects of long-term ozone exposure. If long-term ozone exposure, especially pre-pandemic exposure, 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. Therefore, we next estimate the impact of long-term ozone exposure on mortality from COVID-19.

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

$$Ozone_{cym} = \beta_0 + \beta_1 Ozone_{IV\ cym} + \boldsymbol{X}_{cym}\omega + \boldsymbol{W}_{cym}\varphi + \gamma_c + \delta_{sm} + \theta_{yz} + \mu_{cym} \quad (4)$$

[22] 6,492 lives {= (8.109-7.946) × 331,900,000 / 100,000 ×12}; 11,391 lives {= (8.109-7.823) × 331,900,000 / 100,000 ×12}; 16,249 lives {= (8.109-7.701) × 331,900,000 / 100,000 ×12}; 21,109 lives {= (8.109-7.579) × 331,900,000 / 100,000 ×12}.

[23] 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/.

[24] 106.85 billion dollars {≈ 9,380,000×11,391}; 113.67 billion dollars {≈ 9,979,014×11,391}.

$$Y_{cym} = \beta_0 + \beta_1 \widehat{Ozone_{cym}} + \boldsymbol{X}_{cym}\omega + \boldsymbol{W}_{cym}\varphi + \gamma_c + \delta_{sm} + \theta_{yz} + \varepsilon_{cym} \quad (5)$$

In this model, Y_{cym} denotes the deaths per 100,000 from COVID-19 at county c in month m and year y.[25] $\boldsymbol{X}$ 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[26]. All other variables follow Equations (2) and (3).

Table 6 presents the estimated effect of ozone exposure in the preceding five years on deaths per 100,000 from COVID-19. The estimates indicate that an additional ppb of the average concentrations of ozone in the preceding five years significantly increases 2.367–2.871 deaths per 100,000 per month from COVID-19. A back-of-envelope calculation shows that 28,706 deaths[27] per month could be avoided among the US population in the pandemic if the average concentrations of 24-hour ozone in the preceding five years decreased by one standard deviation.

8 Conclusion

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 transferred from upwind neighbor counties. We demonstrate that an increase of one standard deviation in the average concentrations of ozone in the preceding five years increases respiratory mortality by 0.062–0.066 standard deviations. If ozone concentrations decreased by 10 percent, 11,391 deaths from respiratory diseases would be avoided annually, which is equivalent to a VSL of $106.85–113.67 billion.

Further analyses reveal deeper insights into how long-term ozone exposure affects respiratory health. First, by varying the exposure window from half a year to six years, our estimates suggest that several years of exposure to ozone pollution is 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

[25] We obtain nationwide COVID-19 infection rate, mortality rate, and vaccination rate data in 2020 and 2021 from the CDC.
[26] 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.
[27] 28,706 deaths {=2.367×331,900,000/100,000×3.654}.

more significant effects on vulnerable groups. Third, we discover that the causal effect of ozone pollution on respiratory mortality is nonlinear: that is, there is a critical threshold above which the detrimental effects of ozone exposure commence. Fourth, our extension analysis on COVID-19 indicates that, if ozone concentrations had been decreased by one standard deviation in the preceding five years, 28,706 deaths per month could have been avoided among the US population in the pandemic.

The policy and research implications of this study are as follows. First, our estimates suggest that policymakers should focus on vulnerable populations (e.g., older people), raising public 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., diabetes and cognitive disorders) that might be affected by long-term ozone exposure. We leave this work for future studies.

This study has a few limitations. First, we assumed that the deceased had been living in their county of residence for the entire five years before death. Owing to the nature of our dataset, we could not identify whether the deceased resided out of the area during the five-year exposure window. As a result, the effects we have estimated may be understated. This suggests that our findings should be interpreted as the lower bounds of the long-term ozone exposure effects on respiratory mortality. Second, our empirical strategy was limited by the variables available in the dataset. An ideal dataset would include longitudinal hospitalization information for the deceased, which would enable us to test the underlying mechanism by which long-term ozone exposure weakens self-recovery ability and exacerbates respiratory diseases.

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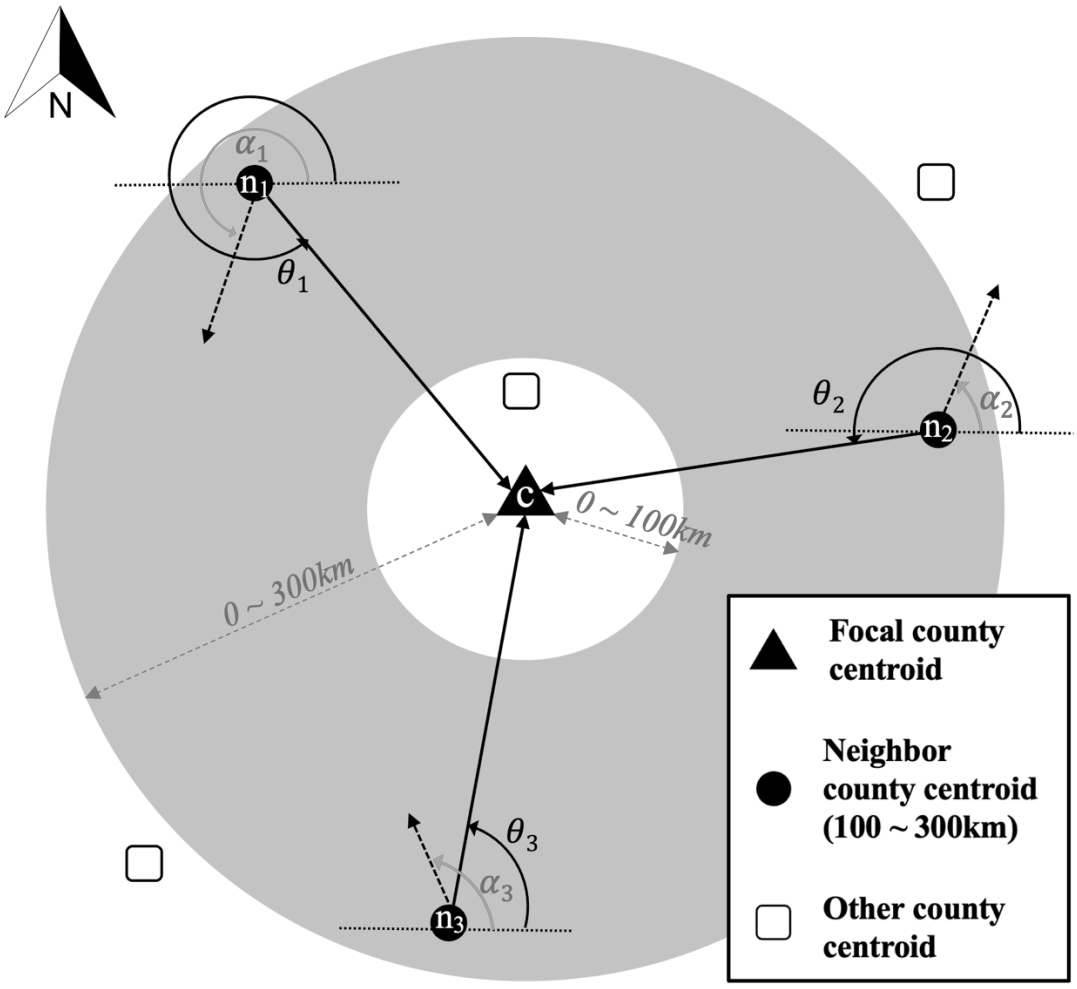
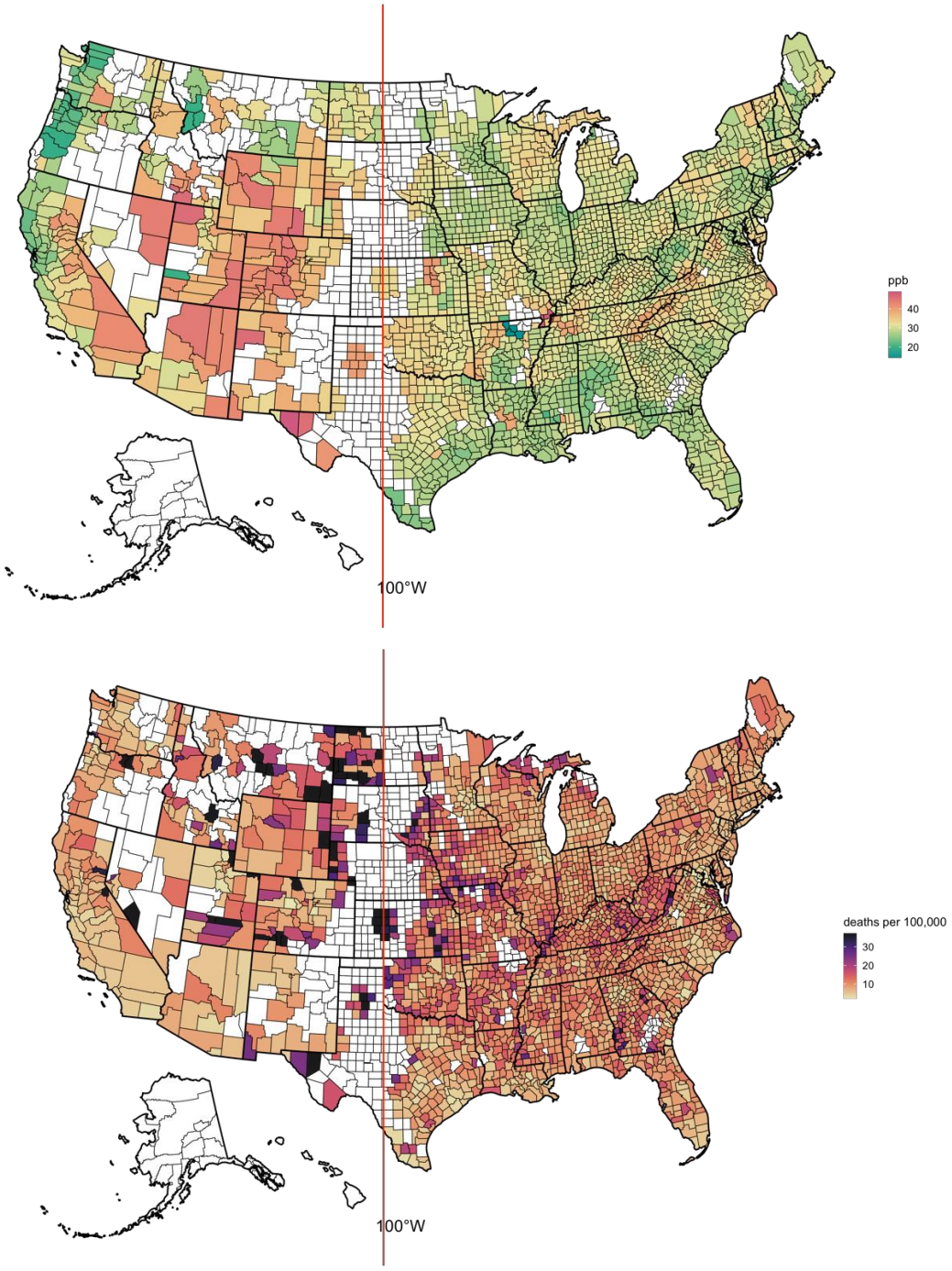
Figures and Tables

Figure 1: Illustration of Instrumental Variable Strategy



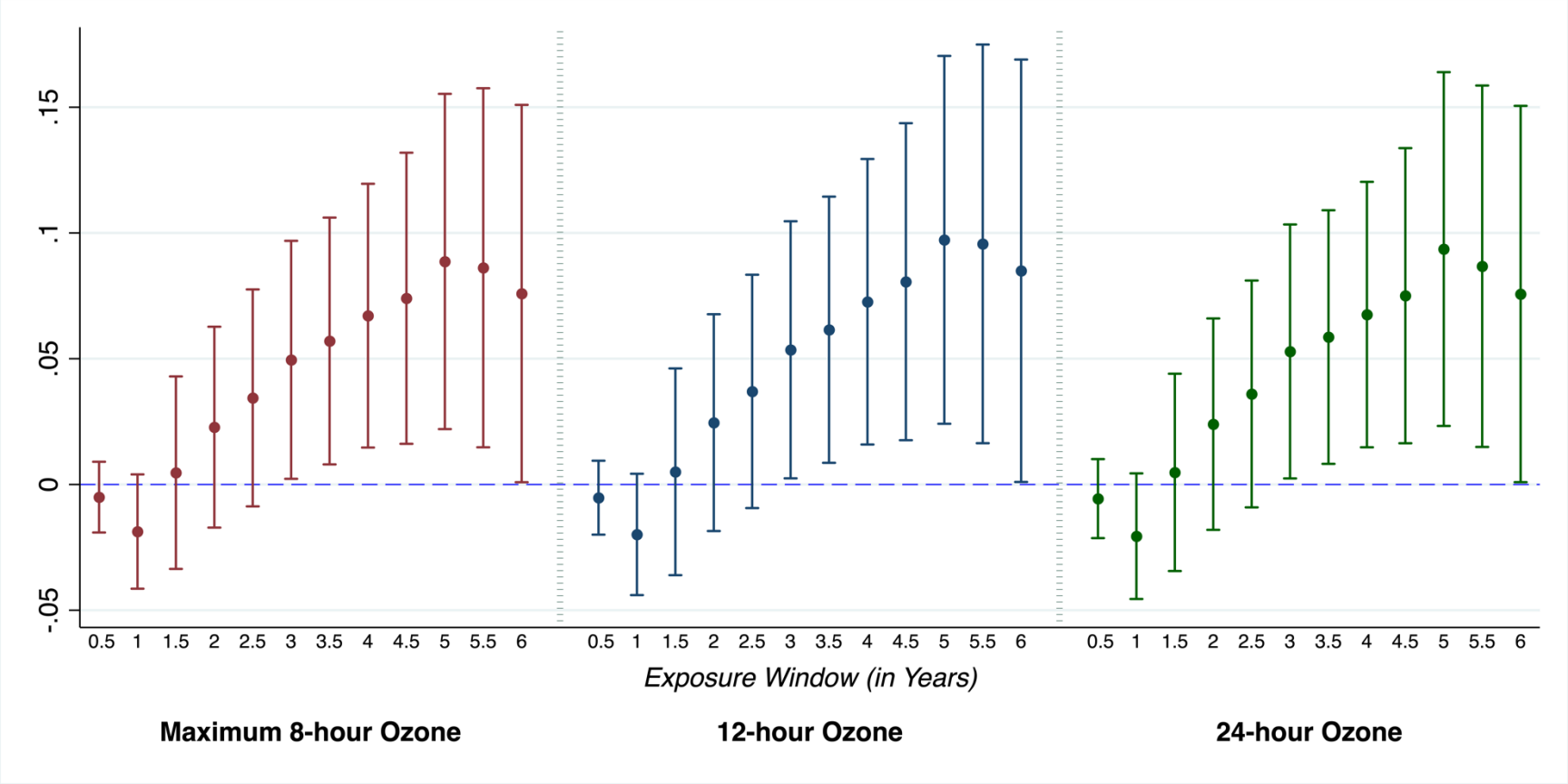
Notes: This figure illustrates an example of the long-distance transmission of surface-level ozone from upwind counties within a distance radius of 100~300 km to a focal county. The triangle represents a focal county. The circles represent neighbor counties distancing 100~300 km from the focal county. 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.

Figure 2: Spatial Distributions of Ozone Concentrations and Respiratory Mortality



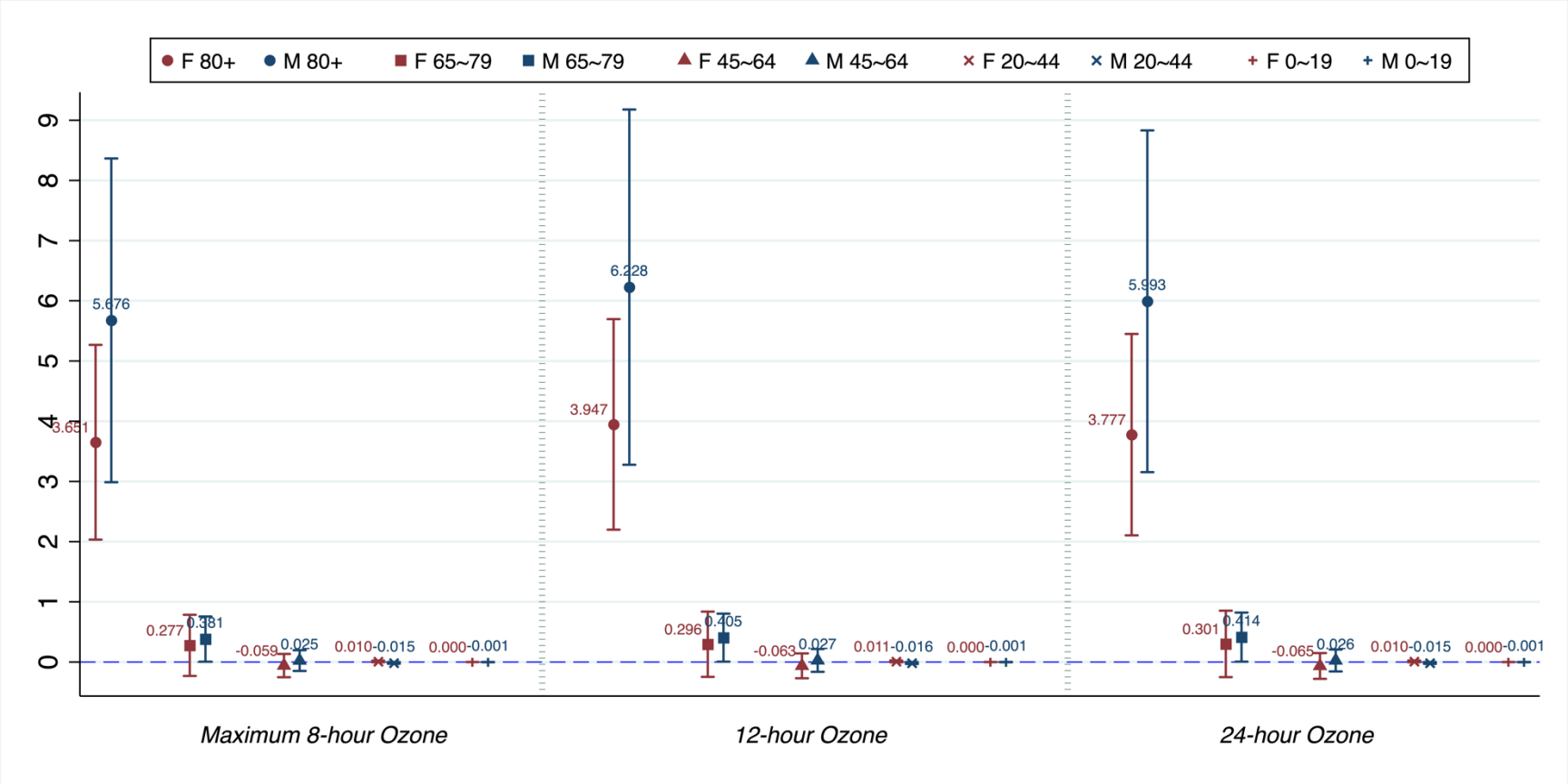
Notes: This top figure depicts the average ozone concentrations in the US. The bottom figure depicts the average deaths per 100,000 from respiratory diseases in the US. The red lines denote the 100th meridian.

Figure 3: Alternative Exposure Windows



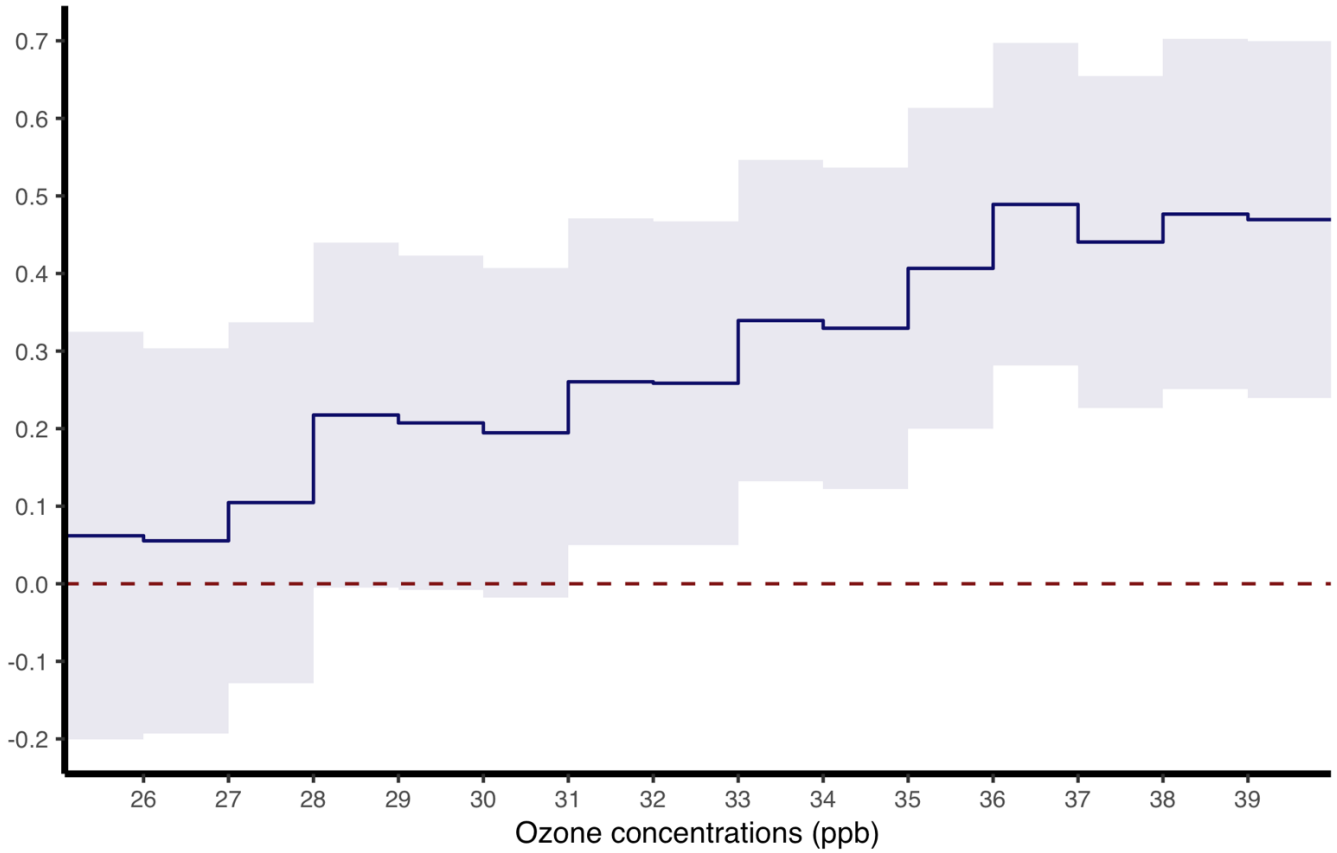
Notes: This figure depicts the impacts of maximum eight-hour ozone (left), 12-hour ozone (middle), and 24-hour ozone (right) on the deaths per 100,000 from respiratory diseases. The model is estimated using Equation 3. Ozone is calculated using average concentrations from the preceding half a year to the preceding six years. Each circle denotes a point estimate from a separate regression, and the whisker shows the 90% confidence intervals. The weather controls include the quadratics of maximum temperature, relative humidity, precipitation, solar radiation, maximum wind-speed, wind direction, and surface atmospheric pressure. Our specification includes the county fixed effects, state-by-month fixed effects, and year-by-season fixed effects. Standard errors are clustered at the state level.

Figure 4: Effects Across Age-by-Sex Groups



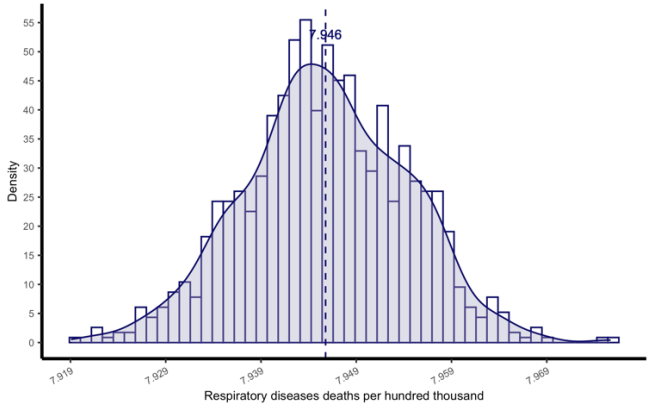
Notes: This figure depicts the impacts of maximum eight-hour ozone (left), 12-hour ozone (middle), and 24-hour ozone (right) on the deaths per 100,000 from respiratory diseases by different age-by-sex groups. The model is estimated using Equation (3). Ozone is calculated using the average concentrations from the preceding five years. Each symbol (a circle denotes the age group 80+, a square denotes 65~79, a triangle denotes 45~64, "×" denotes 20~44, and "+" denotes 0~19) represents a point estimate from a separate regression, and the whisker shows the 90% confidence intervals. The red color denotes the female group, and the blue color refers to the male group. The weather controls include the quadratics of maximum temperature, relative humidity, precipitation, solar radiation, maximum wind speed, wind direction, and surface atmospheric pressure. Our specification includes the county fixed effects, state-by-month fixed effects, and year-by-season fixed effects. Standard errors are clustered at the state level.

Figure 5: Nonlinear Effects of 24-Hour Ozone on Deaths from Respiratory Diseases

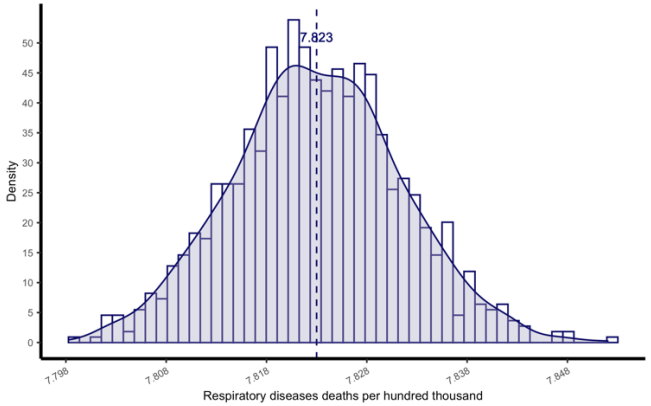


Notes: This figure depicts the nonlinear effects of 24-hour ozone on deaths per 100,000 from respiratory diseases. 24-hour ozone is calculated using the average concentrations from the preceding five years. The weather controls include the quadratics of maximum temperature, relative humidity, precipitation, solar radiation, maximum wind speed, wind direction, and surface atmospheric pressure. Our specification includes the county fixed effects, state-by-month fixed effects, and year-by-season fixed effects. The dark blue line indicates estimated coefficients for different ozone bins, and the light blue band represents the 95% confidence intervals. The red dashed line is a baseline of no effect. The reference bin is less than 25 ppb.

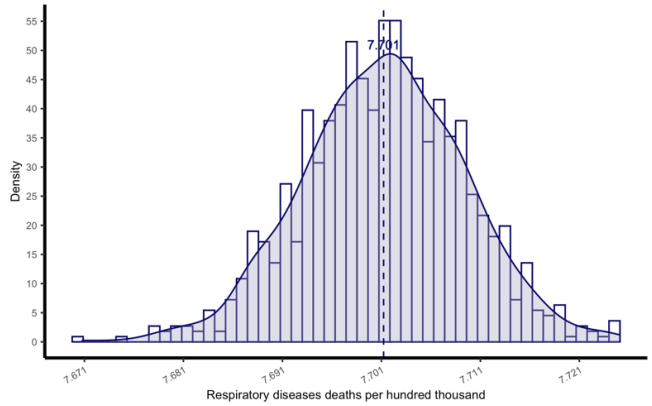
Figure 6: Predicted Deaths from Respiratory Diseases (24-Hour Ozone)



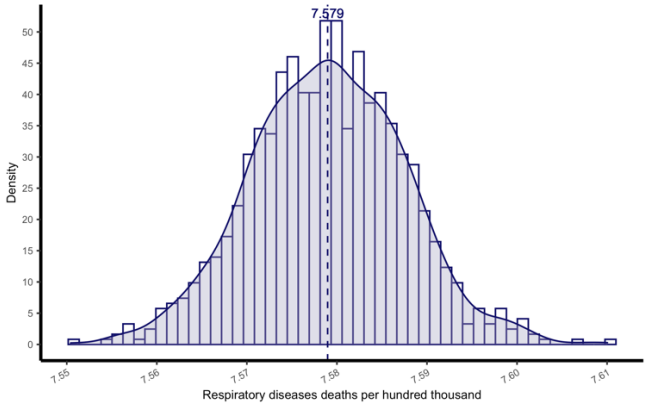
Scenario A: 5% Decrease in Ozone



Scenario B: 10% Decrease in Ozone



Scenario C: 15% Decrease in Ozone



Scenario D: 20% Decrease in Ozone

Notes: This figure depicts the predicted deaths per 100,000 from respiratory diseases caused by surface-level 24-hour 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 1,000 times. The x-axis denotes predicted deaths per 100,000 from respiratory diseases. The y-axis denotes the density within each bootstrapping predicted deaths bin.

Table 1: Summary Statistics

Variables	Unit	N	Mean	SD	Min	Max
Respiratory Diseases Deaths per 100,000	/	254,120	8.109	6.325	0.128	202.840
Maximum 8-Hour Ozone	ppb	254,120	38.801	4.483	0	74
12-Hour Ozone	ppb	254,120	34.707	4.327	0	74
24-Hour Ozone	ppb	254,120	30.618	4.200	0	67.500
Ozone from Upwind Counties (100~300 km)	ppb	254,120	200.761	41.345	0	359.858
Maximum Temperature	℃	254,120	17.948	4.880	-10.892	36.998
Maximum Wind-speed	m/s	254,120	4.085	1.734	0.343	35.754
Wind Direction	°	254,120	188.510	19.008	2.694	286.333
Solar Radiation	$10^3 MJ/m^2$	254,120	23.923	2.828	3.423	43.472
Precipitation	m	254,120	5.238	1.509	0.004	26.165
Surface Pressure	hPa	254,120	973.400	50.895	684.733	1,023.743
Relative Humidity	%	254,120	66.593	6.448	15.988	90.635

Notes: This table reports the summary statistics. All variables are at the county-month level. The data sample covers 2633 counties during 2008-2019 in the United States. Maximum eight-hour ozone, 12-hour ozone, 24-hour ozone, ozone from upwind counties (100~300km), maximum temperature, maximum wind-speed, wind direction, surface atmospheric pressure, and relative humidity are the average values over the 5-year exposure window. Solar radiation and precipitation are the cumulative values over the five-year exposure window.

Table 2: First-Stage Estimation: Effects of Ozone from Upwind Neighbor Counties on Ozone in the Focal County

	(1)	(2)	(3)
	Maximum Eight-Hour Ozone	12-Hour Ozone	24-Hour Ozone
Ozone from Upwind Counties (50~200 km)	0.019***	0.017***	0.018***
	(0.001)	(0.001)	(0.001)
Observations	256,387	256,387	256,387
County FE	YES	YES	YES
State-Month FE	YES	YES	YES
Year-Season FE	YES	YES	YES
Weather Controls	YES	YES	YES
KP F-Statistics	2,818	2,511	4,319

Notes: This table reports the first-stage estimation. The dependent variables are the five-year average concentrations of maximum eight-hour ozone, 12-hour ozone, and 24-hour ozone in Col. (1) - (3), respectively. The main explanatory variable is ozone from upwind counties in the radius band of 100~300 km. 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. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table 3: Effects of Ozone Pollution on Mortality from Respiratory Diseases

	(1)	(2)	(3)
	Respiratory Diseases Deaths per 100,000		
Panel A: OLS			
Ozone Concentrations	0.030	0.035	0.032
	(0.029)	(0.034)	(0.032)
Panel B: 2SLS			
Ozone Concentrations	0.089**	0.097**	0.094**
	(0.040)	(0.044)	(0.042)
KP F-Statistics	2,818	2,511	4,319
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

Notes: This table reports the estimated effects of five-year average concentrations of ozone on respiratory diseases deaths per 100,000. Panel A reports the OLS estimates in which ozone is not instrumented. Panel B reports the 2SLS estimates, 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 five-year average concentrations of maximum eight-hour ozone, 12-hour ozone, and 24-hour 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. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table 4: Robustness Checks

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
		Change FE	Alternative CL	Alternative IV		Change Weather Controls		Add Pollutants	
	Baseline	County, State-Month, Year	State, Year-Month	150~300 km	Account for Windspeed	Temp Bins	Quadratic Avg Temp	PM_{10}	East of the 100th Meridian
Panel A: Maximum 8-Hour Ozone									
Max Eight-Hour	0.089**	0.089**	0.089**	0.085**	0.087**	0.085**	0.085**	0.092**	0.114**
Ozone	(0.040)	(0.040)	(0.040)	(0.040)	(0.038)	(0.042)	(0.040)	(0.039)	(0.053)
Observations	254,120	254,120	254,120	254,120	254,120	254,120	254,120	254,120	225,799
Weather controls	YES	YES	YES	YES	YES	YES	YES	YES	YES
KP F-Statistics	2,818	2,866	2,818	2,770	2,810	2,572	2,856	2,889	2,172
Panel B: 12-Hour Ozone									
12-Hour Ozone	0.097**	0.098**	0.097**	0.094**	0.096**	0.093**	0.093**	0.100**	0.136**
	(0.044)	(0.044)	(0.044)	(0.044)	(0.043)	(0.046)	(0.044)	(0.042)	(0.064)
Observations	254,120	254,120	254,120	254,120	254,120	254,120	254,120	254,120	225,799
Weather controls	YES	YES	YES	YES	YES	YES	YES	YES	YES
KP F-Statistics	2,511	2,560	2,511	2,478	2,524	2,320	2,543	2,662	1,583
Panel C: 24-Hour Ozone									
24-Hour Ozone	0.094**	0.094**	0.094**	0.092**	0.090**	0.089**	0.089**	0.096**	0.137**
	(0.042)	(0.042)	(0.042)	(0.043)	(0.040)	(0.044)	(0.042)	(0.041)	(0.064)
Observations	254,120	254,120	254,120	254,120	254,120	254,120	254,120	254,120	225,799
Weather controls	YES	YES	YES	YES	YES	YES	YES	YES	YES
KP F-Statistics	4,319	4,385	4,319	4,196	4,796	3,882	4,413	4,750	2,807

Notes: The dependent variable is respiratory diseases deaths per 100,000. The primary explanatory variables are the five-year average concentrations of maximum eight-hour ozone, 12-hour ozone, and 24-hour ozone in Panel 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) by 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 IV used in Col. (1) by changing the distance radius of upwind counties to 150~300 km. Col. (5) replaces the IV used in Col. (1) by accounting for wind-speed when constructing the IV. Col. (6) replaces the maximum temperature by days within each 5°C bin. Col. (7) replaces the quadratic polynomial of maximum temperature by the quadratic polynomial of average temperature. Col. (8) adds PM_{10} as a control variable. Col. (9) only includes counties east of the 100th meridian. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table 5: Effects Across Diseases

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Influenza and Pneumonia Deaths per 100,000			Chronic Lower Respiratory Diseases Deaths per 100,000			*Placebo Test*: Gastrointestinal Diseases Deaths per 100,000		
Ozone	0.091***	0.100***	0.097***	0.083*	0.091*	0.089*	0.012	0.013	0.013
Concentrations	(0.024)	(0.026)	(0.025)	(0.044)	(0.049)	(0.048)	(0.010)	(0.011)	(0.010)
KP F-Statistics	2,818	2,511	4,319	2,818	2,511	4,319	2,818	2,511	4,319
Observations	254,120	254,120	254,120	254,120	254,120	254,120	254,120	254,120	254,120
County FE	YES	YES	YES	YES	YES	YES	YES	YES	YES
State-Month FE	YES	YES	YES	YES	YES	YES	YES	YES	YES
Year-Season FE	YES	YES	YES	YES	YES	YES	YES	YES	YES
Weather controls	YES	YES	YES	YES	YES	YES	YES	YES	YES

Notes: Col. (1) - (3) report the estimated effects of the five-year average concentrations of ozone on influenza/pneumonia deaths per 100,000. Col. (4) - (6) report the estimated effects of the five-year average concentrations of ozone on chronic lower respiratory diseases deaths per 100,000. Col. (7) - (9) report the estimated effects of the five-year average concentrations of ozone on gastrointestinal diseases deaths per 100,000. All columns use ozone from upwind counties in the range of 100~300 km as the instrumental variable. The primary explanatory variables are the five-year average concentrations of maximum eight-hour ozone (Col. (1), (4), and (7)), 12-hour ozone (Col. (2), (5), and (8)), and 24-hour ozone (Col. (3), (6), and (9)). 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. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table 6: Effects of Ozone Pollution on Mortality from COVID-19

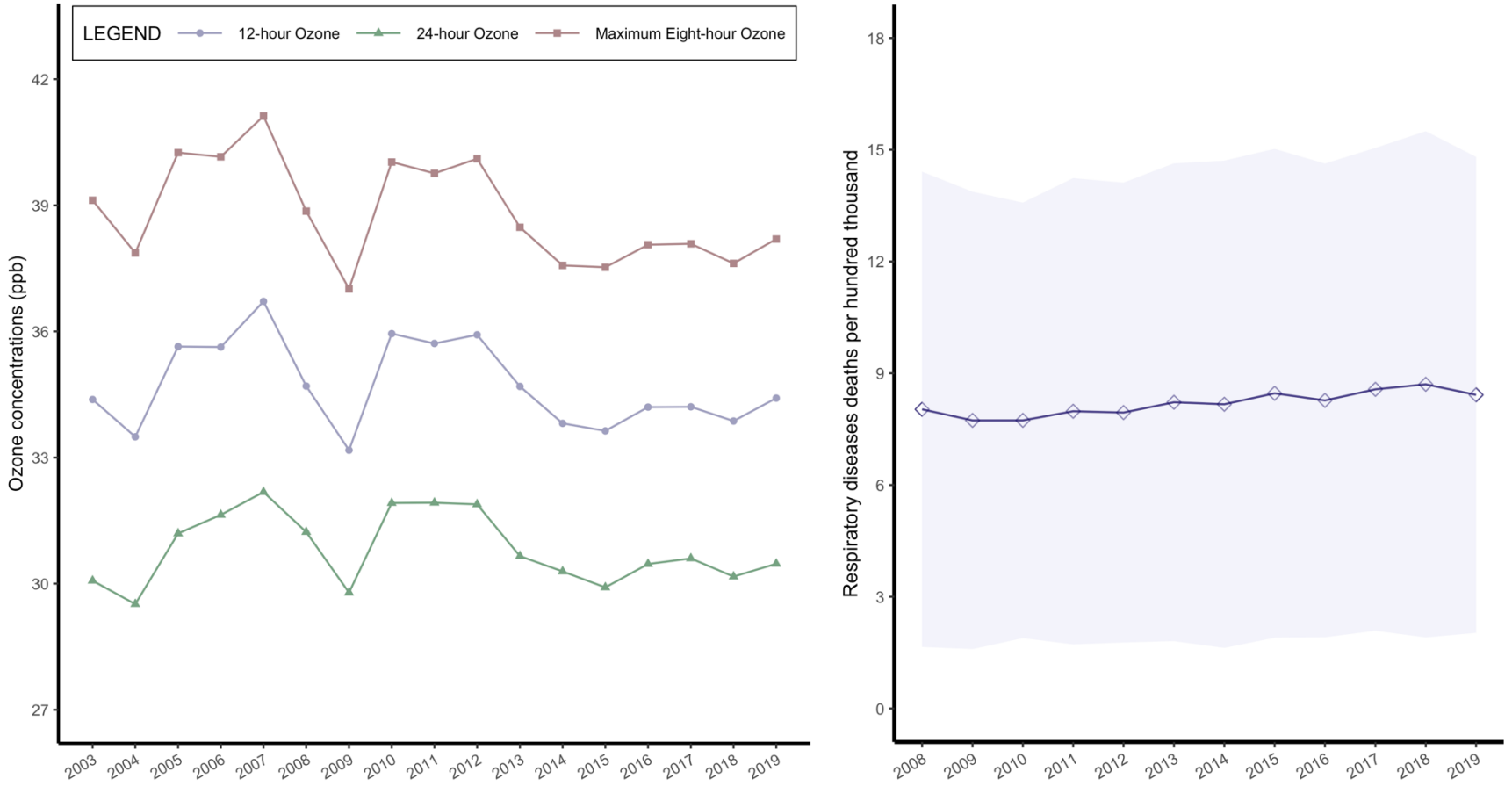
	(1)	(2)	(3)
	COVID-19 Deaths per 100,000		
Ozone Concentrations	2.800***	2.871***	2.367***
	(0.946)	(0.968)	(0.793)
KP F-Statistics	204.1	219.9	227.5
Observations	43,685	43,685	43,685
County FE	YES	YES	YES
State-Month FE	YES	YES	YES
Year-Season FE	YES	YES	YES
Weather controls	YES	YES	YES
Other controls	YES	YES	YES

Notes: This table reports the 2SLS estimates of five-year ozone concentrations on COVID-19 deaths per 100,000. The instrumental variable is ozone from upwind counties in the range of 100~300 km. The dependent variable is COVID-19 deaths per 100,000. The primary explanatory variables are the five-year average concentrations of maximum eight-hour ozone, 12-hour ozone, and 24-hour 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. Other controls include the COVID-19 infection rate, vaccination rate, the indicator for stay-at-home order, and the indicator for income support. 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. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Appendix - For Online Publication

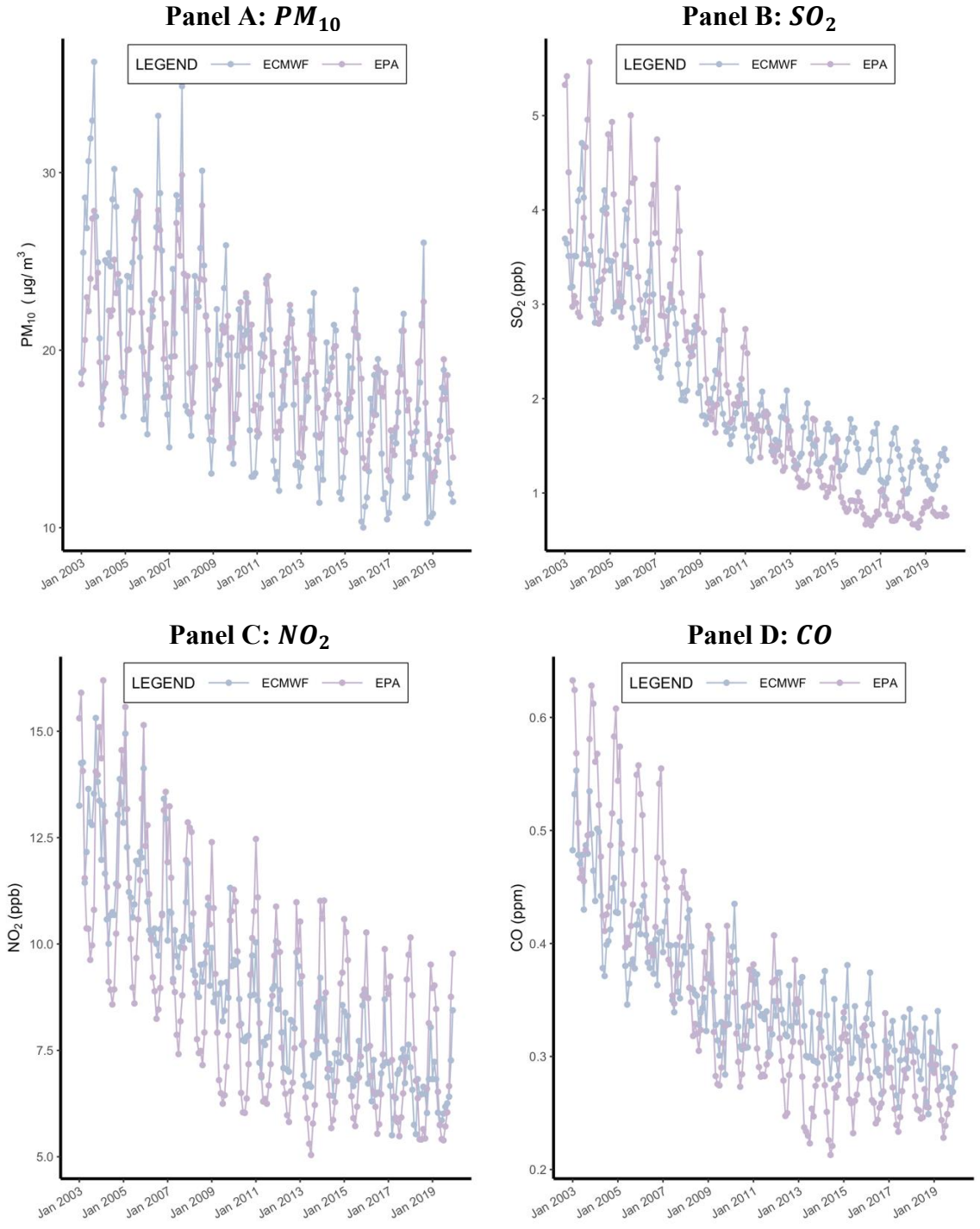
Appendix A. Additional Figures

Figure A1: Ozone Concentrations and Respiratory Mortality Over Time



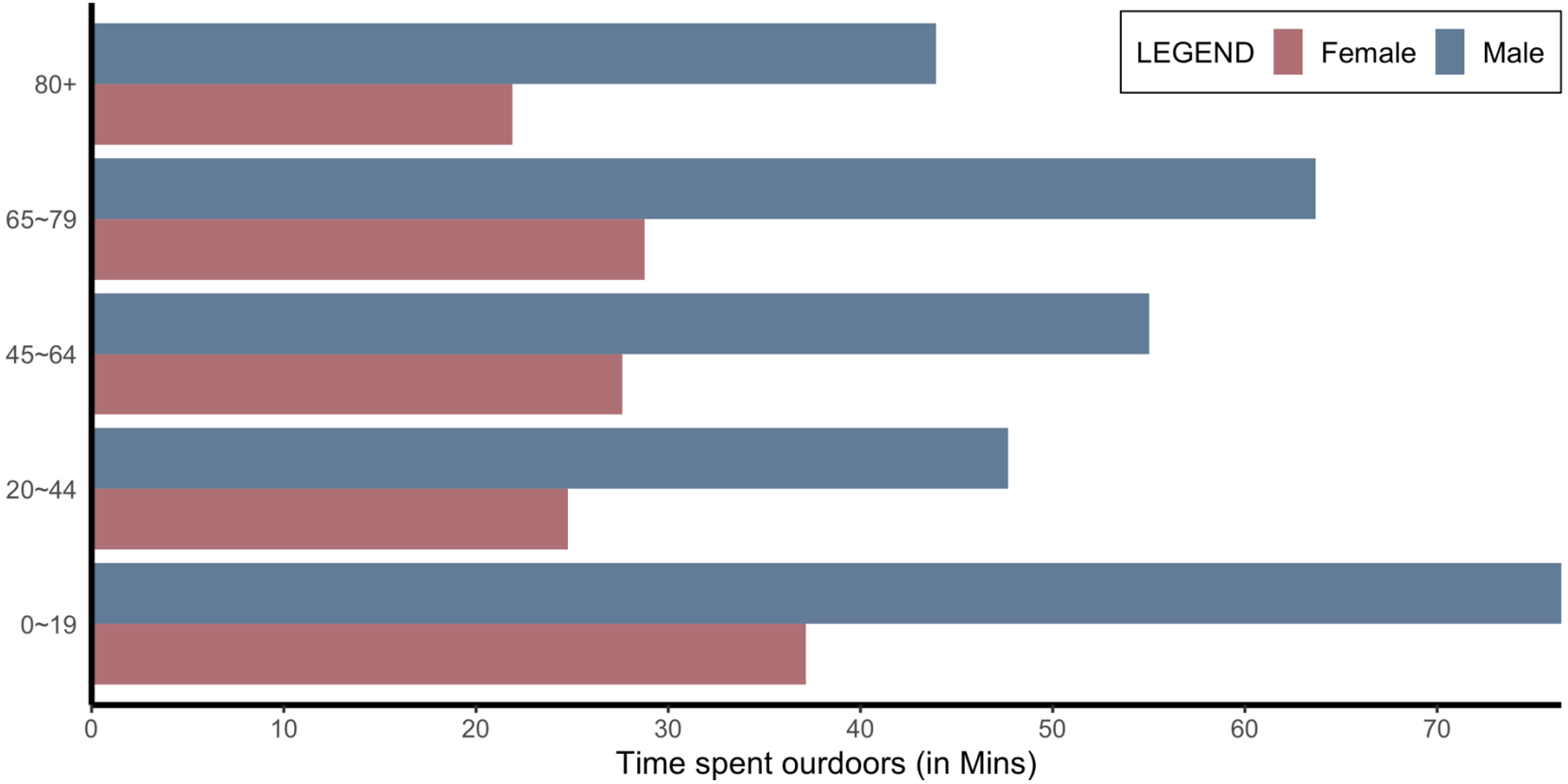
Notes: This left figure depicts ozone concentrations in the U.S. from 2003 to 2019. The red square denotes the maximum eight-hour ozone concentrations. The blue circle denotes 12-hour ozone concentrations. The green triangle denotes 24-hour ozone concentrations. The right figure depicts deaths per 100,000 from respiratory diseases in the U.S. from 2008 to 2019. The blue diamond indicates yearly average deaths per 100,000 from respiratory diseases. The shallow band represents plus or minus one standard deviation.

Figure A2: PM_{10}, SO_2, NO_2, and CO Over Time



Notes: This figure compares data from EPA and data from ECMWF. The blue circle denotes air pollutant concentrations from ECMWF, and the purple circle denotes air pollutant concentrations from EPA. Panel A, B, C, and D depict monthly PM_{10}, SO_2, NO_2, and CO concentrations over time, respectively.

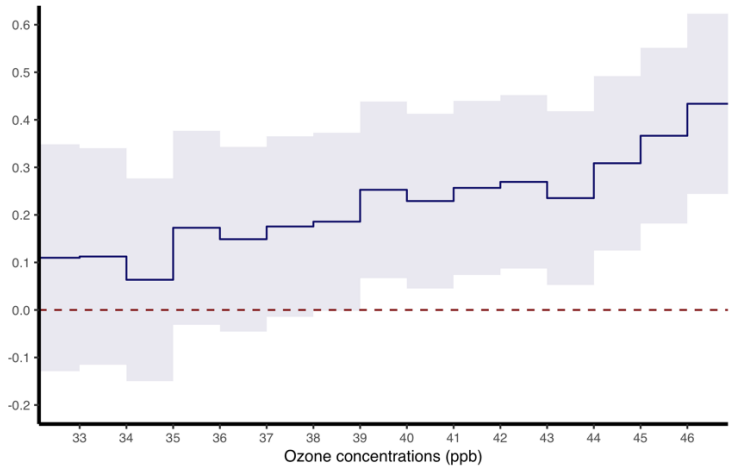
Figure A3: Time Spent Outdoors



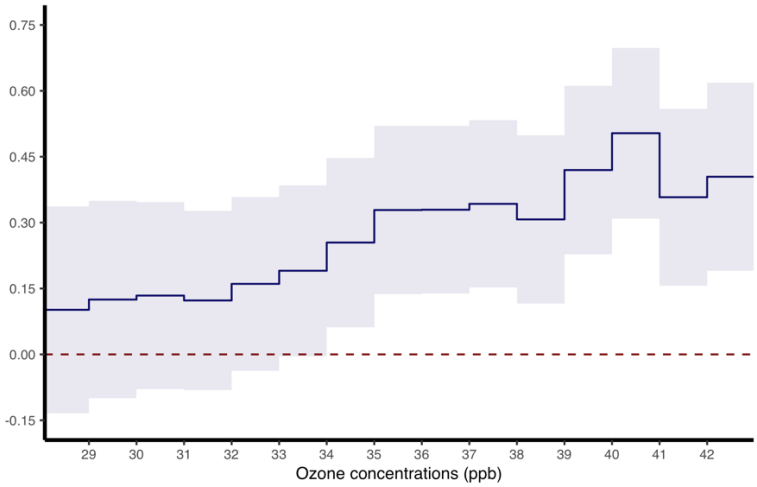
Notes: This figure depicts time spent outdoors by different age-by-sex groups over the time period of 2003 to 2019. The data source is ATUS. The x-axis denotes time spent outdoors in minutes. The y-axis presents age groups. The red bars denote time spent outdoors by females, and the blue bars refer to time spent outdoors by males.

Figure A4: Nonlinear Effects of Maximum eight-hour Ozone and 12-Hour Ozone on Deaths from Respiratory Diseases

Panel A: Maximum eight-hour Ozone



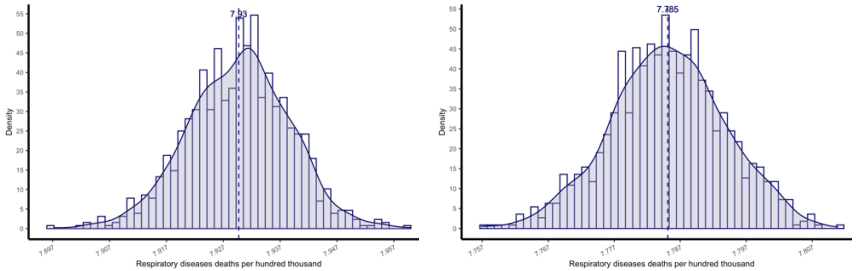
Panel B: 12-Hour Ozone



Notes: This top figure depicts the nonlinear effects of the average concentrations of maximum eight-hour ozone in the preceding five years on deaths per 100,000 from respiratory diseases. The bottom figure depicts the nonlinear effects of the average concentrations of 12-hour ozone in the preceding five years on deaths per 100,000 from respiratory diseases. Maximum eight-hour ozone and 12-hour ozone are calculated using average concentrations from the preceding five years. The weather controls include the quadratics of maximum temperature, relative humidity, precipitation, solar radiation, maximum wind speed, wind direction, and surface atmospheric pressure. Our specification includes the county fixed effects, state-by-month fixed effects, and year-by-season fixed effects. The dark blue line indicates estimated coefficients for different ozone bins, and the light blue band represents 95% confidence intervals. The red dashed line is a baseline of no effect. The reference bins are less than the 5th percentile of maximum eight-hour ozone concentrations and 12-hour ozone concentrations, respectively.

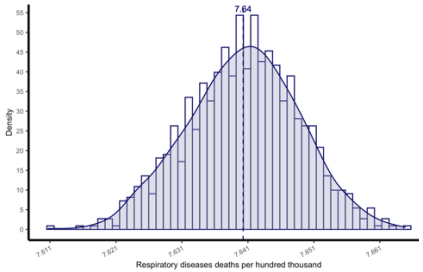
Figure A5: Predicted Deaths from Respiratory Diseases (Maximum eight-hour Ozone and 12-Hour Ozone)

Panel A: Maximum eight-hour Ozone

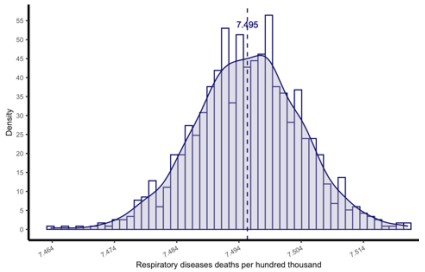


Scenario A:
5% Decrease in Ozone

Scenario B:
10% Decrease in Ozone

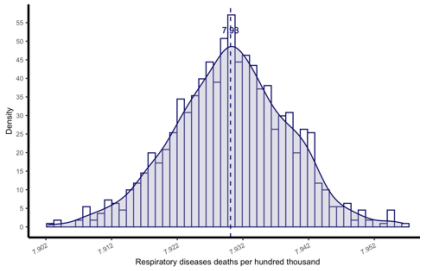


Scenario C:
15% Decrease in Ozone

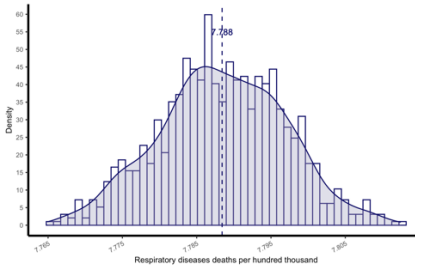


Scenario D:
20% Decrease in Ozone

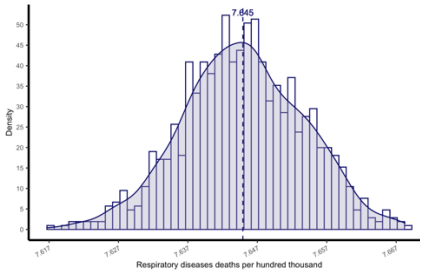
Panel B: 12-Hour Ozone



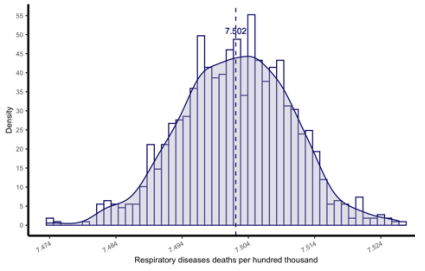
Scenario A:
5% Decrease in Ozone



Scenario B:
10% Decrease in Ozone



Scenario C:
15% Decrease in Ozone



Scenario D:
20% Decrease in Ozone

Notes: Panel A depicts the predicted deaths per 100,000 from respiratory diseases caused by surface-level maximum eight-hour ozone under four different scenarios using bootstrap simulations. Panel B depicts the bootstrapping predicted deaths per 100,000 from respiratory diseases caused by surface-level 12-hour ozone under four different scenarios. 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 predicted deaths per 100,000 from respiratory diseases. The y-axis denotes the kernel density within each bootstrapping predicted deaths bin.

Appendix B. Additional Robustness Checks

Table B1: Change Fixed Effects

	(1)	(2)	(3)	(4)	(5)	(6)
	Baseline	County, State-Month, Year-Month	County, State-Season, Year-Month	County-Month, Year	County-Month, Year-Month	County-Month, Year-Season
Panel A: Maximum Eight-Hour Ozone						
Max Eight-Hour Ozone	0.089**	0.078*	0.074*	0.089**	0.076**	0.088**
	(0.040)	(0.040)	(0.040)	(0.038)	(0.038)	(0.038)
Observations	254,120	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES	YES
KP F-Statistics	2,818	2,801	2,846	3,024	2,962	2,979
Panel B: 12-Hour Ozone						
12-Hour Ozone	0.097**	0.086*	0.081*	0.098**	0.083**	0.097**
	(0.044)	(0.044)	(0.044)	(0.042)	(0.042)	(0.042)
Observations	254,120	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES	YES
KP F-Statistics	2,511	2,490	2,528	2,709	2,640	2,661
Panel C: 24-Hour Ozone						
24-Hour Ozone	0.094**	0.082*	0.078*	0.095**	0.081**	0.094**
	(0.042)	(0.042)	(0.042)	(0.041)	(0.041)	(0.041)
Observations	254,120	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES	YES
KP F-Statistics	4,319	4,289	4,358	4,541	4,444	4,475

Notes: The dependent variable is respiratory diseases deaths per 100,000. The primary explanatory variables are the five-year average concentrations of maximum eight-hour ozone, 12-hour ozone, and 24-hour ozone in Panel A, B, and C, respectively. Col. (1) is the baseline estimate and includes the county fixed effects, state-by-month fixed effects, and year-by-season fixed effects. Col. (2) includes the county fixed effects, state-by-month fixed effects, and year-by-month fixed effects. Col. (3) includes the county fixed effects, state-by-season fixed effects, and year-by-month fixed effect. Col. (4) includes the county-by-month fixed effects and year fixed effects. Col. (5) includes the county-by-month fixed effects and year-by-month fixed effects. Col. (6) includes the county-by-month fixed effects and year-by-season fixed effects. The weather controls include the quadratics of maximum temperature, relative humidity, precipitation, solar radiation, maximum wind-speed, wind direction, and surface atmospheric pressure. The instrumental variable is ozone from upwind counties in the range of 100~300 km. Standard errors are clustered at the state level. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table B2: Cluster at Different Levels

	(1)	(2)	(3)	(4)	(5)
	Baseline	2-way Cluster: State, Year-Season	2-way Cluster: County, State-Year	2-way Cluster: County, State-Year-Season	2-way Cluster: County, State-Year-Month
Panel A: Maximum Eight-Hour Ozone					
Max Eight-Hour Ozone	0.089**	0.089**	0.089**	0.089**	0.089**
	(0.040)	(0.040)	(0.040)	(0.040)	(0.040)
Observations	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES
KP F-Statistics	2,818	2,818	2,812	2,812	2,812
Panel B: 12-Hour Ozone					
12-Hour Ozone	0.097**	0.097**	0.097**	0.097**	0.097**
	(0.044)	(0.044)	(0.044)	(0.044)	(0.044)
Observations	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES
KP F-Statistics	2,511	2,511	2,505	2,505	2,505
Panel C: 24-Hour Ozone					
24-Hour Ozone	0.094**	0.094**	0.094**	0.094**	0.094**
	(0.042)	(0.042)	(0.042)	(0.042)	(0.042)
Observations	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES
KP F-Statistics	4,319	4,320	4,309	4,309	4,309

Notes: The dependent variable is respiratory diseases deaths per 100,000. The primary explanatory variables are the five-year average concentrations of maximum eight-hour ozone, 12-hour ozone, and 24-hour ozone in Panel A, B, and C, respectively. Col. (1) is the baseline estimate and clusters standard errors at the state level. Col. (2) clusters standard errors at the state and season-by-year levels (two way). Col. (3) clusters standard errors at the county and state-year levels (two way). Col. (4) clusters standard errors at the county and state-year-season levels (two way). Col. (5) clusters standard errors at the county and state-year-month levels (two way). The weather controls include the quadratics of maximum temperature, relative humidity, precipitation, solar radiation, maximum wind-speed, wind direction, and surface atmospheric pressure. All specifications include the county fixed effects, state-by-month fixed effects, and year-by-season fixed effects. The instrumental variable is ozone from upwind counties in the range of 100~300 km. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table B3: Change Weather Controls

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Baseline	Remove Max Temp	Remove Windspeed	Remove Wind Direction	Remove Solar Radiation	Remove Precipitation	Remove Atmospheric Pressure	Remove Relative Humidity
Panel A: Maximum 8-Hour Ozone								
Max Eight-Hour Ozone	0.089**	0.091**	0.091**	0.077*	0.109***	0.087**	0.087**	0.083**
	(0.040)	(0.040)	(0.042)	(0.040)	(0.040)	(0.038)	(0.040)	(0.040)
Observations	254,120	254,120	254,120	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES	YES	YES	YES
KP F-Statistics	2,818	2,801	2,663	2,786	3,020	3,130	2,823	2,853
Panel B: 12-Hour Ozone								
12-Hour Ozone	0.097**	0.099**	0.102**	0.085*	0.119***	0.092**	0.095**	0.091**
	(0.044)	(0.044)	(0.047)	(0.044)	(0.043)	(0.042)	(0.044)	(0.044)
Observations	254,120	254,120	254,120	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES	YES	YES	YES
KP F-Statistics	2,511	2,507	2,244	2,443	2,683	2,812	2,514	2,509
Panel C: 24-Hour Ozone								
24-Hour Ozone	0.094**	0.093**	0.091**	0.082*	0.115***	0.089**	0.092**	0.088**
	(0.042)	(0.041)	(0.042)	(0.043)	(0.042)	(0.039)	(0.042)	(0.042)
Observations	254,120	254,120	254,120	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES	YES	YES	YES
KP F-Statistics	4,319	4,372	3,020	4,199	4,625	4,911	4,291	4,291

Notes: The dependent variable is respiratory diseases deaths per 100,000. The primary explanatory variables are the five-year average concentrations of maximum eight-hour ozone, 12-hour ozone, and 24-hour ozone in Panel A, B, and C, respectively. Col. (1) is the baseline estimate and controls for the quadratics of maximum temperature, relative humidity, precipitation, solar radiation, maximum wind-speed, wind direction, and surface atmospheric pressure. Col. (2) removes the quadratic polynomial of maximum temperature. Col. (3) removes the quadratic polynomial of maximum wind-speed. Col. (4) removes the quadratic polynomial of wind direction. Col. (5) removes the quadratic polynomial of solar radiation. Col. (6) removes the quadratic polynomial of precipitation. Col. (7) removes the quadratic polynomial of atmospheric surface pressure. Col. (8) removes the quadratic polynomial of relative humidity. All specifications include the county fixed effects, state-by-month fixed effects, and year-by-season fixed effects. The instrumental variable is ozone from upwind counties in the range of 100~300 km. Standard errors are clustered at the state level. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Table B4: Add Air Pollutants

	(1)	(2)	(3)	(4)	(5)
	Baseline	Add $PM_{2.5}$	Add SO_2	Add CO	Add NO_2
Panel A: Maximum 8-Hour Ozone					
Max Eight-Hour Ozone	0.089**	0.132***	0.086**	0.088**	0.092**
	(0.040)	(0.044)	(0.040)	(0.040)	(0.041)
Observations	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES
KP F-Statistics	2,818	2,856	2,870	2,848	2,770
Panel B: 12-Hour Ozone					
12-Hour Ozone	0.097**	0.144***	0.094**	0.096**	0.101**
	(0.044)	(0.048)	(0.043)	(0.043)	(0.045)
Observations	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES
KP F-Statistics	2,511	2,464	2,581	2,571	2,459
Panel C: 24-Hour Ozone					
24-Hour Ozone	0.094**	0.129***	0.091**	0.093**	0.097**
	(0.042)	(0.043)	(0.042)	(0.042)	(0.043)
Observations	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES
KP F-Statistics	4,319	4,602	4,529	4,601	4,422

Notes: The dependent variable is respiratory diseases deaths per 100,000. The primary explanatory variables are the five-year average concentrations of maximum eight-hour ozone, 12-hour ozone, and 24-hour ozone in Panel A, B, and C, respectively. Col. (1) is the baseline estimate and controls for the quadratics of maximum temperature, relative humidity, precipitation, solar radiation, maximum wind-speed, wind direction, and surface atmospheric pressure. Col. (2) adds $PM_{2.5}$ as a control variable. Col. (3) adds SO_2 as a control variable. Col. (4) adds CO as a control variable. Col. (5) adds NO_2 as a control variable. All specifications include the county fixed effects, state-by-month fixed effects, and season-by-year fixed effects. The instrumental variable is ozone from upwind counties in the range of 100~300 km. Standard errors are clustered at the state level. **** p<0.01, ** p<0.05, * p<0.1.*

Table B5: Add Socioeconomic Controls

	(1)	(2)	(3)	(4)	(5)
	Baseline	Add Unemployment Rate	Add Average Annual Salary	Add Poverty Rate	Add the Ratio of Population to Primary Care Physicians
Panel A: Maximum 8-Hour Ozone					
Max Eight-Hour Ozone	0.089**	0.094**	0.096**	0.089**	0.077*
	(0.040)	(0.040)	(0.040)	(0.040)	(0.040)
Observations	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES
KP F-Statistics	2,818	2,951	2,952	2,951	2,962
Panel B: 12-Hour Ozone					
12-Hour Ozone	0.097**	0.103**	0.105**	0.098**	0.084*
	(0.044)	(0.044)	(0.044)	(0.044)	(0.044)
Observations	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES
KP F-Statistics	2,511	2,637	2,643	2,632	2,665
Panel C: 24-Hour Ozone					
24-Hour Ozone	0.094**	0.099**	0.101**	0.094**	0.083*
	(0.042)	(0.042)	(0.042)	(0.042)	(0.043)
Observations	254,120	254,120	254,120	254,120	254,120
Weather controls	YES	YES	YES	YES	YES
KP F-Statistics	4,319	4,688	4,658	4,639	4,534

Notes: The dependent variable is respiratory diseases deaths per 100,000. The primary explanatory variables are maximum eight-hour ozone concentrations, 12-hour ozone concentrations, and 24-hour ozone concentrations in Panel A, B, and C, respectively. Col. (1) is the baseline estimate and controls for the quadratics of maximum temperature, relative humidity, precipitation, solar radiation, maximum wind-speed, wind direction, and surface atmospheric pressure. Col. (2) adds unemployment rate as a control variable. Col. (3) adds average annual salary as a control variable. Col. (4) adds poverty rate as a control variable. Col. (5) adds the ratio of population to primary care physicians as a control variable. All specifications include the county fixed effects, state-by-month fixed effects, and season-by-year fixed effects. The instrumental variable is ozone from upwind counties in the range of 100~300 km. Standard errors are clustered at the state level. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Appendix C. Additional Evidence of the Nonlinear Health Effects of Ozone Pollution

In this section, we explore how cumulative exposure to extreme ozone pollution affects respiratory mortality as additional evidence to further demonstrate that the health effects of ozone pollution could be nonlinear. Following McGrath et al. (2015), we adopt three cumulative indices of ozone, $AOT40$, $SUM06$, and $W126$ as measurements of cumulative exposure to extreme ozone pollution:

$$AOT40 = \sum_{h=1}^{n} C_h \text{ where } C_h = \begin{cases} O_h - 40, O_h > 40 \\ 0, \quad O_h \leq 40 \end{cases}, \tag{4}$$

$$SUM06 = \sum_{h=1}^{n} C_h \text{ where } C_h = \begin{cases} O_h, O_h > 60 \\ 0, \quad O_h \leq 60 \end{cases}, \tag{5}$$

$$W126 = \sum_{h=1}^{n} (O_h \cdot \frac{1}{1+4403 \cdot e^{-126 \cdot O_h}}), \tag{6}$$

where O_h is ozone in ppm-h for hour h, and n refers to the total number of hours over the exposure window. $AOT40$ sums hourly ozone concentrations exceeding 40 ppb; $SUM06$ sums hourly excessive ozone concentrations above 60 ppb; $W126$ sums hourly ozone concentrations where higher concentrations are assigned a greater weight (McGrath et al., 2015). To estimate the effects of cumulative exposure to extreme ozone pollution on respiratory mortality, we replace the five-year average of maximum eight-hour ozone, 12-hour ozone, and 24-hour ozone concentrations in Equation (3), respectively with $AOT40$, $SUM06$, and $W126$ in the same five-year exposure window. Table C1 presents how respiratory mortality responds to cumulative exposure to extreme ozone pollution, indicating that the point estimates on all three metrics of cumulative ozone ($AOT40$, $SUM06$, and $W126$) are significantly positive. In all three metrics of cumulative ozone, lower ozone concentrations are assigned less weight and higher ozone concentrations are assigned more weight (McGrath et al., 2015); therefore, the consistent estimates on all three metrics draw the same conclusion that higher ozone concentrations cause more damage to the respiratory health over the five-year exposure window compared to their lower counterparts.

Table C1: Effects of Cumulative Extreme Ozone Pollution on Mortality from Respiratory Diseases

	(1)	(2)	(3)
	Respiratory Diseases Deaths per 100,000		
Cumulative Ozone	0.005**	0.004**	0.005**
	(0.002)	(0.002)	(0.002)
KP F-Statistics	5,644	7,883	6,217
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

Notes: This table reports the estimated effects of cumulative five-year ozone concentrations (in ppm-h, 1ppm = 10^3 ppb) on respiratory diseases deaths per 100,000. The instrumental variable is ozone from upwind counties in the range of 100~300 km. The dependent variable is respiratory diseases deaths per 100,000. The primary explanatory variables are AOT40, SUM06, and W126 over the five-year exposure window 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. 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. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.