

Online Appendix

“The Long-run Effect of Air Pollution on Survival”

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A Supplementary information and analysis

A.1 Data

Monitor-level data for sulfur dioxide (SO_2), total suspended particulates (TSP), nitrogen dioxide (NO_2), O_3 , and carbon monoxide (CO) for the years 1972–1988 were obtained by email request from the US Environmental Protection Agency (EPA). Each SO_2 observation provides a sample measurement, usually recorded over a period of one hour. We dropped SO_2 observations with durations longer than 24 hours or values above 1000 parts per billion (ppb) or below -2 ppb.¹ We dropped CO, NO_2 , and O_3 observations with negative values. All TSP observations were non-negative. We then aggregated pollutant data to the monitor-day level, weighting by the time duration of each measurement. Finally, data were aggregated to the county-day level by averaging across all monitors within each county.

Figure A.1 displays the population-weighted concentrations and the number of counties with at least one operational monitor, by year for each pollutant. With the exception of O_3 , the population-weighted means for all pollutants decline substantially during our sample period. CO data are readily available from the mid-1970s onward, covering about 225 counties annually, while O_3 data are unavailable prior to 1980. NO_2 coverage is high for most of the 1970s but drops sharply by the late 1980s. SO_2 data offer broader spatial coverage than most other pollutants, with over 400 counties monitoring SO_2 each year and about half the US population living in a county with at least one SO_2 monitor. While TSP monitors cover more counties than SO_2 monitors, they collect data less frequently and are more often located in sparsely populated areas. At the county-day level, we have 62 percent fewer population-weighted TSP observations than SO_2 observations.

Figure A.3 shows the locations of the 4,740 active SO_2 pollution monitors during our 1972–1988 sample period, spanning 1,041 counties nationwide. After merging the pollution data with our mortality, weather, and demographic datasets, our main estimation sample—summarized in Table 1—includes 1,026 counties.

A.2 Short-run empirical results: additional analyses and robustness checks

Complier analysis

Table A.1 describes complier characteristics by regressing county-level measures obtained from the Regional Economic Information System and SEER datasets on the strength of the first stage (BEA 1988; SEER

¹According to the AQS Data Coding Manual version 2.38 (February 2, 2010), the maximum allowable sample value for SO_2 is 1000 ppb. The EPA informed us by email that small negative values can arise due to noise and should be included in sample averages to avoid bias. We chose -2 as the bottom cutoff because it appeared to be the minimum allowable sample value.

1988). The unit of observation is a county-year. Regressions include year fixed effects and are weighted by the county population in each year. The strength of the first stage is not significantly associated with population size, the percent of Black residents, per-capita income, or the employment rate. Counties with stronger first stages have higher per-capita government transfers, but the magnitude of the coefficient is small: a \$55 difference for each 1 ppb difference in the first stage, relative to a mean of \$1,290. They also have a slightly higher share of the population that is 65 and older: 0.19 percentage points per 1 ppb difference in the first stage, relative to a mean of 11. The largest difference is for mean SO₂ concentrations: 1.6 ppb higher for each 1 ppb difference in the first stage relative to a mean of 9.5 ppb.

Controlling for autocorrelation in air pollution and weather

Because wind direction is autocorrelated, our IV estimates for the effect of 1-day exposure may inadvertently capture the effects of multi-day exposure. To assess this possibility, Table A.2 regresses tomorrow's SO₂ levels on today's (instrumented) SO₂ levels and baseline controls, varying the number of leads and lags of the wind instrument. Without including any leads or lags, the correlation between predicted SO₂ today and SO₂ tomorrow is 0.27, which is large and statistically significant. However, including a single lead reduces this correlation to 0.033, and adding additional leads or lags reduces it even further and renders it statistically insignificant. These results indicate that our preferred specification, which includes two leads and two lags, adequately accounts for autocorrelation in pollution. Table A.3 confirms that our one-day estimate is largely insensitive to the numbers of instrument leads and lags included in the regression. Table A.4 shows modest sensitivity to the inclusion of leads and lags of our weather controls. Our longer-run (>1 day) specifications, which always include leads, account for any influence of subsequent weather on mortality.

Ruling out composition bias

The observed differences in mortality trends by cause of death (Figure 3) could potentially be driven by composition bias. For example, if cancer-related deaths occurring weeks after exposure are misattributed to cardiovascular causes, we might incorrectly observe a rising trend in cardiovascular mortality in place of a true increase in cancer deaths. This type of misclassification is especially plausible among people with multiple chronic conditions. To investigate this possibility, we estimate the effect of acute exposure on deaths where cancer was listed as either the underlying *or* secondary (contributing) cause of death. Those estimates, shown in Figure A.7, are very similar to our main estimates, indicating that misclassification in the underlying cause of death does not explain the cancer pattern in Figure 3.

Estimates by detailed causes of death

Figure A.8 shows cause-specific estimates for the mortality effect of SO₂ over time, disaggregating cardiovascular and other diseases into 5 and 21 subcategories, respectively. Three cardiovascular subcategories—heart disease, cerebrovascular disease, and atherosclerosis—show significant same-day and monthly mortality increases. We also find strong and growing effects for deaths from chronic obstructive pulmonary disease (COPD), pneumonia and influenza, and other respiratory diseases, which collectively account for

over 99 percent of all deaths from respiratory illness (Table A.15). By contrast, we find no significant monthly effects for conditions not previously linked to air pollution exposure, such as stomach ulcers and appendicitis.

Longer mortality window by age

Figure A.9 and Table A.7 report age-specific mortality estimates for outcome windows up to one month (28 days) following exposure. Among individuals aged 65 and over, the monthly estimates are substantially larger than the 1-day estimates, indicating that any short-run mortality displacement is more than offset by delayed effects of acute exposure. By contrast, while the 1-day estimates are statistically significant for ages 20–64, the corresponding monthly estimates become statistically insignificant and imprecisely estimated. This pattern suggests that, unlike among older age groups, acute pollution exposure increases mortality among young adults primarily by accelerating death among those who would have died within a month regardless.

Alternative fixed effects and clustering levels

Table A.10 investigates the sensitivity of our estimates to including alternative sets of fixed effects. Column (1) reproduces our baseline estimate, while Columns (2)–(6) present five alternative specifications. The estimates are fairly similar in most specifications, although they decline by 15–20 percent when we include state-by-calendar-week-by-year (Column 5) or state-by-day-of-year (Column 6) fixed effects.

Table A.11 shows that clustering standard errors at broader geographic levels, such as state or geographic group, or applying two-way clustering at the county and geographic-group-by-year levels, has little effect on their magnitude.

Assessing potential violations of monotonicity

We interpret our IV estimate as a weighted average treatment effect where the weights are non-negative, which requires monotonicity of air pollution in the instruments (Angrist, Graddy and Imbens, 2000). In other words, if the instruments increase SO₂ levels in one county, then SO₂ levels in other counties assigned to the same geographic group cannot fall. This assumption could be violated if the relationship between wind direction and SO₂ varies substantially across counties located within the same group. We investigate this possibility with two alternative specifications that allow the effect of wind direction on SO₂ to vary over either a larger or smaller geographic area. Columns (2)–(3) of Table A.12 show that these alternatives produce estimates similar to our main estimate (Column 1), suggesting that violations of the monotonicity assumption are unlikely to drive our findings.

Columns (4)–(6) of Table A.12 show that our estimates change little if we employ the following non-parametric specification for $f^g(\theta_{cd})$:

$$f^g(\theta_{cd}) = \sum_{i=1}^b \gamma_g^i 1[G_c = g] \times 1 \left[\frac{360}{b}(i-1) \leq \theta_{cd} < \frac{360}{b}i \right]$$

where the first indicator function, $1[G_c = g]$, is equal to 1 if county c is a member of group g and 0 otherwise. The second indicator function is equal to 1 if the local wind direction, θ_{cd} , now expressed in degrees rather than radians, lies between $\frac{360}{b}(i-1)$ and $\frac{360}{b}i$ for some fixed value b . For example, when $b = 4$ and $i = 1$, the indicator function will equal 1 if the wind direction lies between 0 and 90 degrees. Columns (4)–(6) report estimates when we set b equal to 9, 6, and 4, respectively.

Assessing potential weak instrument bias

Our reported F -statistics in Table 2 exceed 600 and our 2SLS estimates differ significantly from OLS estimates, indicating that weak instrument bias is unlikely. As an additional check, Table A.13 shows that our 2SLS and LIML estimates are identical to two significant figures, reflecting only negligible differences at higher precision. Because LIML is approximately median unbiased even in the presence of many weak instruments, the similarity of the two estimators further confirms the strength of our instrument. We also conduct a placebo exercise using randomly generated wind directions in place of the actual wind direction (Table A.14). The resulting first-stage F -statistics never exceed 4, confirming that actual wind direction captures meaningful variation in SO_2 levels rather than spurious correlations.

A.3 Model calibration

The dynamic production model of health given by Equation (3) depends on seven parameters:

$\{\alpha, \delta, I, \sigma_\varepsilon, \mu_H, \sigma_H, \underline{H}\}$. To achieve identification, we normalize two parameters: $\underline{H}^* = 0$ and $\sigma_H^* = 1$. The five remaining parameters are calibrated using a 1972 period life table, as described in Section 5.2. We assume that pollution exposure only affects the parameters δ and $\underline{H}$, and denote their post-exposure-change values as $\{\tilde{\delta}, \tilde{\underline{H}}\}$.

Let $\Theta^* = \{\alpha^*, I^*, \mu_H^*, \sigma_H^*, \sigma_\varepsilon^*\}$ denote the calibrated baseline parameters before any changes in exposure. Consider a population of N individuals whose health capital evolves according to Equation (3). Let S be a random-number seed that fixes the initial stock of health capital, H_{i0} , and the evolution of the i.i.d. shock, ε_{it} , for all individuals. Define $M_t(\delta^*, \underline{H}^* | \Theta^*, N, S)$ as the deterministic mortality rate at time $t \geq 0$ in the absence of changes in pollution exposure.

We use age-specific IV estimates of the mortality effect of air pollution exposure to jointly recover the parameters $\{\tilde{\delta}, \tilde{\underline{H}}\}$. Let $\hat{\beta}_{a,c}^1$ denote the IV estimate for the effect of acute exposure on 1-day mortality for age group a and cause of death c , and let $[t_a^0, t_a^1]$ represent the age range (in days) spanned by that age group. For any age $t \in [t_a^0, t_a^1]$, we can solve for $\tilde{\underline{H}}_{at}$ as the implicit solution to:

$$\hat{\beta}_{a,\text{cancer}}^1 = M_t(\delta^*, \tilde{\underline{H}}_{at} | \Theta^*, N, S) - M_t(\delta^*, \underline{H}^* | \Theta^*, N, S) \quad (\text{A.1})$$

Holding $\tilde{\underline{H}}_{at}$ fixed, we then solve for $\tilde{\delta}$, defined implicitly by:²

$$\hat{\beta}_{a,\text{all}}^1 = M_t(\tilde{\delta}_{at}, \tilde{\underline{H}}_{at} | \Theta^*, N, S) - M_t(\delta^*, \underline{H}^* | \Theta^*, N, S) \quad (\text{A.2})$$

²Equivalently, subtracting (A.1) from (A.2) yields $\hat{\beta}_{a,\text{noncancer}}^1 = M_t(\tilde{\delta}_{at}, \tilde{\underline{H}}_{at} | \Theta^*, N, S) - M_t(\delta^*, \tilde{\underline{H}}_{at} | \Theta^*, N, S)$, since $\hat{\beta}_{a,\text{noncancer}}^1 \equiv \hat{\beta}_{a,\text{all}}^1 - \hat{\beta}_{a,\text{cancer}}^1$.

Because health capital is strictly decreasing in δ and death occurs when health falls below $\underline{H}$, the mortality rate, $M_t(\cdot)$, is monotonically increasing in both δ and $\underline{H}$. Consequently, the solutions $\{\tilde{\delta}_{at}, \tilde{\underline{H}}_{at}\}$ to Equations (A.1) and (A.2) are unique and can be computed using standard root-finding algorithms.³

We can solve for $\{\tilde{\delta}_{at}, \tilde{\underline{H}}_{at}\}$ for any $t \in [t_a^0, t_a^1]$. We use the approximate integer midpoint of each age bin, $t = \text{round}[(t_a^0 + t_a^1)/2]$.⁴ To reduce noise from the i.i.d. health shocks, we average over multiple daily ages near the center of each age bin. Specifically, we solve Equations (A.1) and (A.2) for 50 different days near the approximate midpoint of each age bin and take the average. For example, for $a = 65$ (the 65–69 age group), we solve for $\{\tilde{\delta}_{65,t}, \tilde{\underline{H}}_{65,t}\}$ using ages $t = 68\text{y}1\text{d}, t = 68\text{y}2\text{d}, \dots, t = 68\text{y}50\text{d}$.⁵ Figure A.13 shows the model-implied cumulative mortality effects of acute (1-day) exposure across the 50 daily ages in the 65–69 age group, using the calibrated $\{\tilde{\delta}_{65,t}, \tilde{\underline{H}}_{65,t}\}$ for each age t . By construction, the first-day effect matches the 1-day IV estimate for the 65–69 age group (see first row of Column (5) in Table A.7). The subsequent values report longer-run effects of exposure up to 90 days later. The “own-age prediction” in Figure 5 is the average of these 50 trajectories.

We compute the age-specific parameter solutions, $\{\tilde{\underline{H}}_a, \tilde{\delta}_a\}$, as the averages of these 50 solutions:

$$\begin{aligned}\tilde{\underline{H}}_a &= \frac{1}{50} \sum_t \tilde{\underline{H}}_{at} \\ \tilde{\delta}_a &= \frac{1}{50} \sum_t \tilde{\delta}_{at}\end{aligned}$$

Figure A.11 reports $\{\tilde{\underline{H}}_a, \tilde{\delta}_a\}$ for the five oldest age groups, expressed as deviations from the baseline calibrated values (i.e., $\tilde{\underline{H}}_a - \underline{H}^* = \tilde{\underline{H}}_a$ and $\tilde{\delta}_a - \delta^* = \tilde{\delta}_a$). The long-run survival projections shown in Figure 7 use the average across the five oldest age groups:

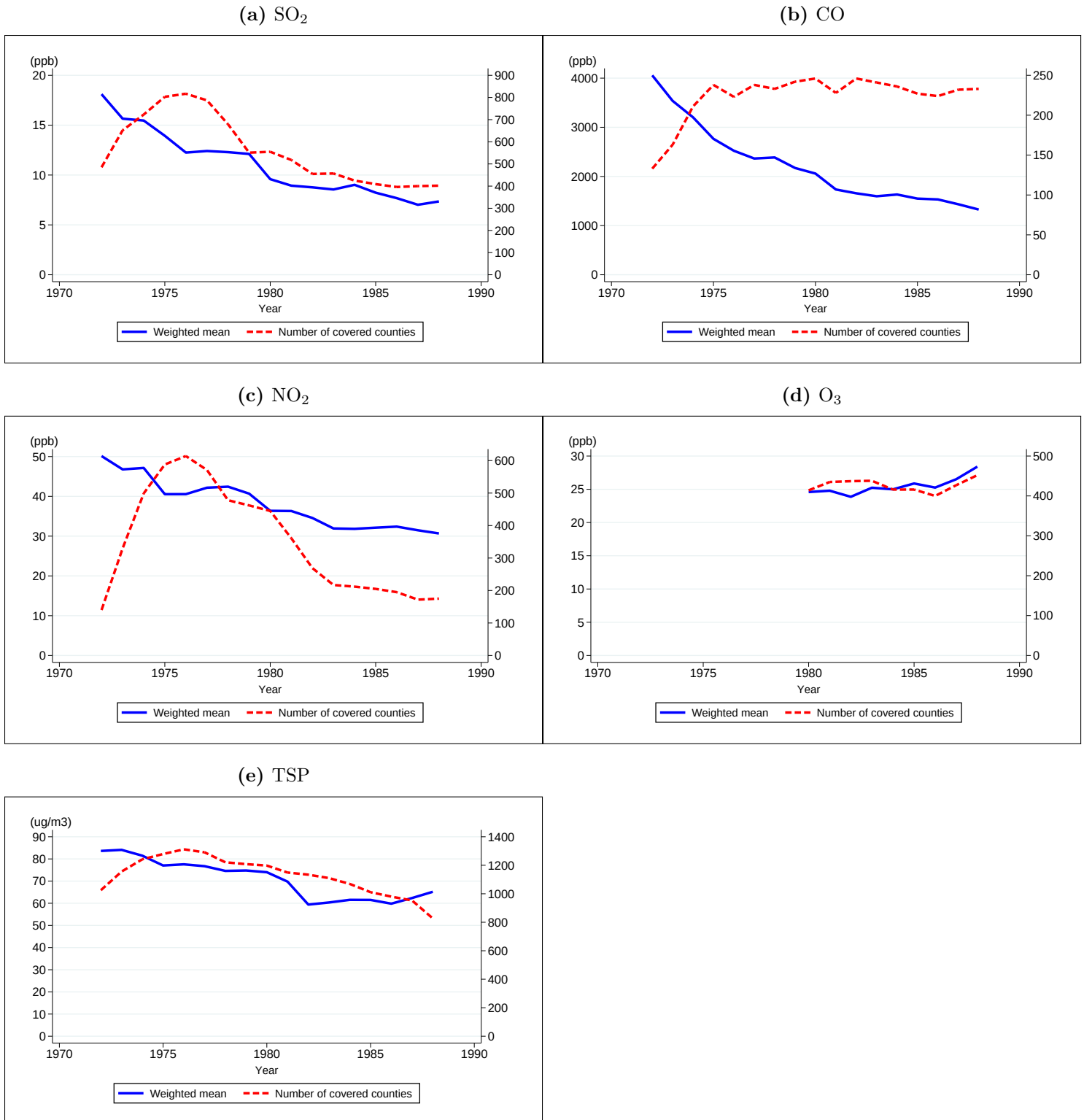
$$\begin{aligned}\tilde{\underline{H}} &= \frac{1}{5} \sum_{a \geq 65} \tilde{\underline{H}}_a \\ \tilde{\delta} &= \frac{1}{5} \sum_{a \geq 65} \tilde{\delta}_a\end{aligned}$$

³Reversing the order—solving for $\tilde{\delta}$ first and then $\tilde{\underline{H}}$ —yields numerically similar, though not identical, results.

⁴For the 85+ age group, we use a midpoint of 90, which is the average age of death in that group during our sample period.

⁵The optimal strategy would employ all $365 \times 5 = 1825$ days in the 5-year age bin, giving more weight to the ages near the midpoint. However, this is computationally burdensome and offers limited additional precision.

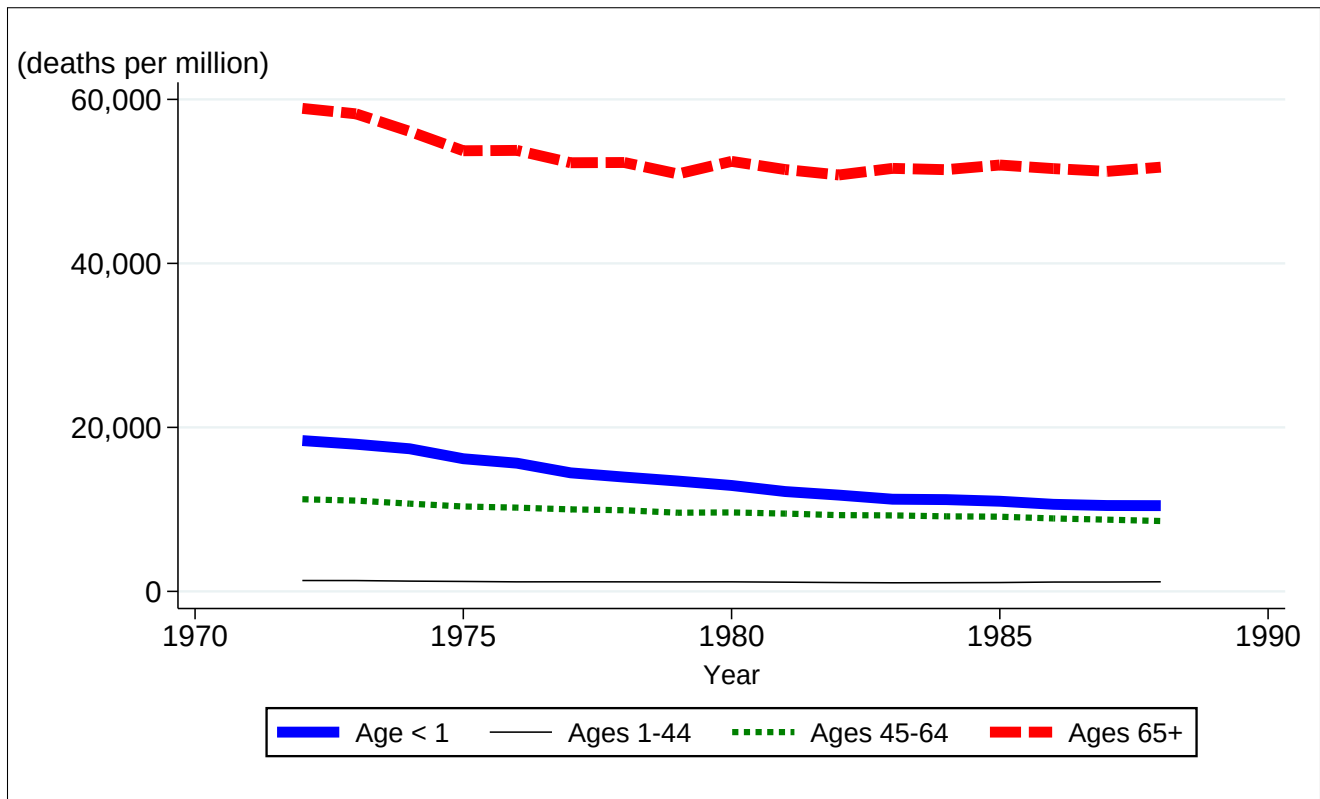
Figure A.1: Air pollution means and population coverage levels, 1972–1988



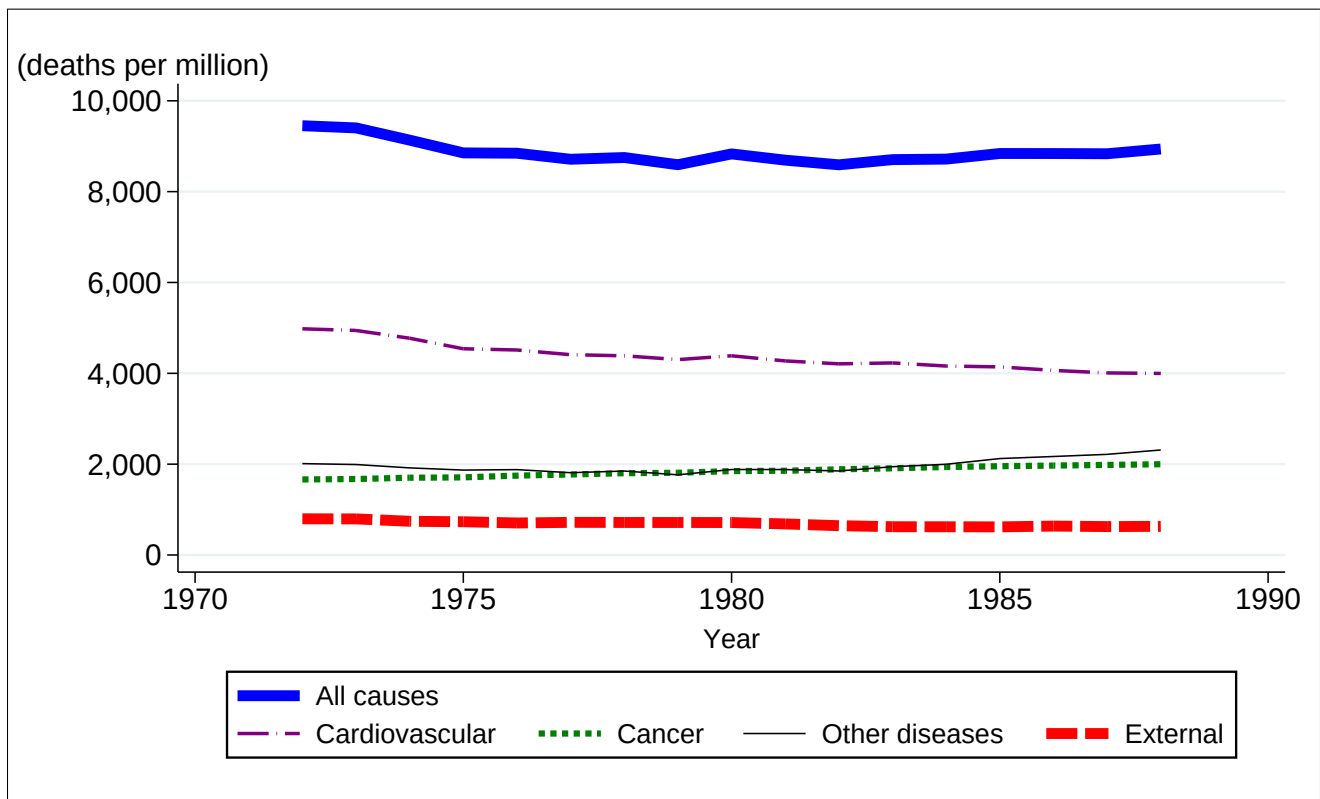
Notes: The solid blue lines report population-weighted pollution levels for all US counties with at least one daily reading for that pollutant. The dashed red line reports the number of counties with at least one operational monitor for the pollutant. Air pollution data are obtained from the EPA Air Quality database. SO₂, CO, NO₂, and O₃ are measured in parts per billion (ppb). Total suspended particulates (TSP) is measured in micrograms per cubic meter ($\mu\text{g}/\text{m}^3$).

Figure A.2: Trends in annual US mortality rates, 1972–1988

(a) By age group

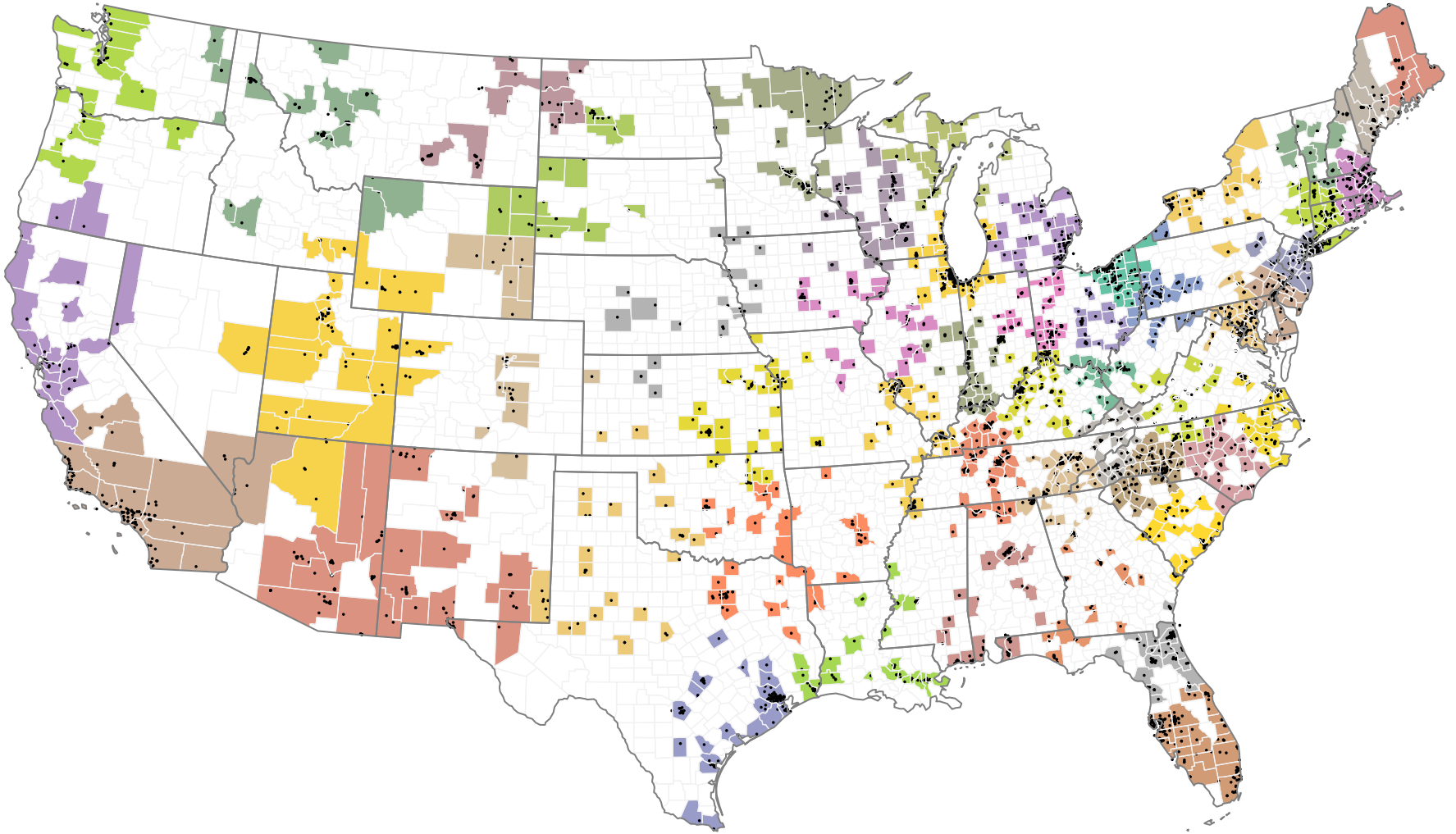


(b) By cause of death



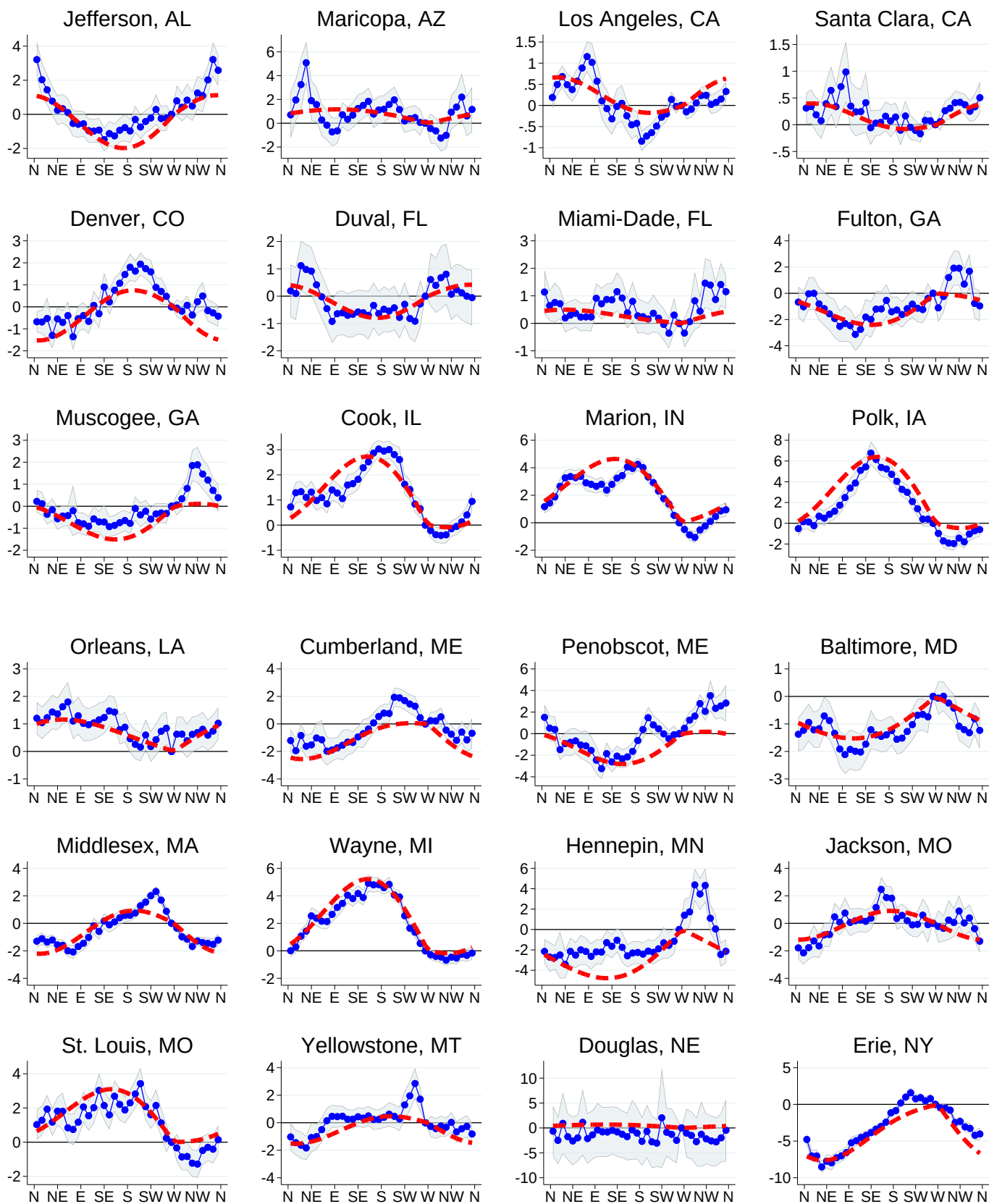
Notes: These two figures report annual mortality rates for the US population. These rates are calculated using mortality data from the National Vital Statistics and population data from SEER. Annual mortality rates are approximately 365 times larger than the daily mortality rates used in the analysis.

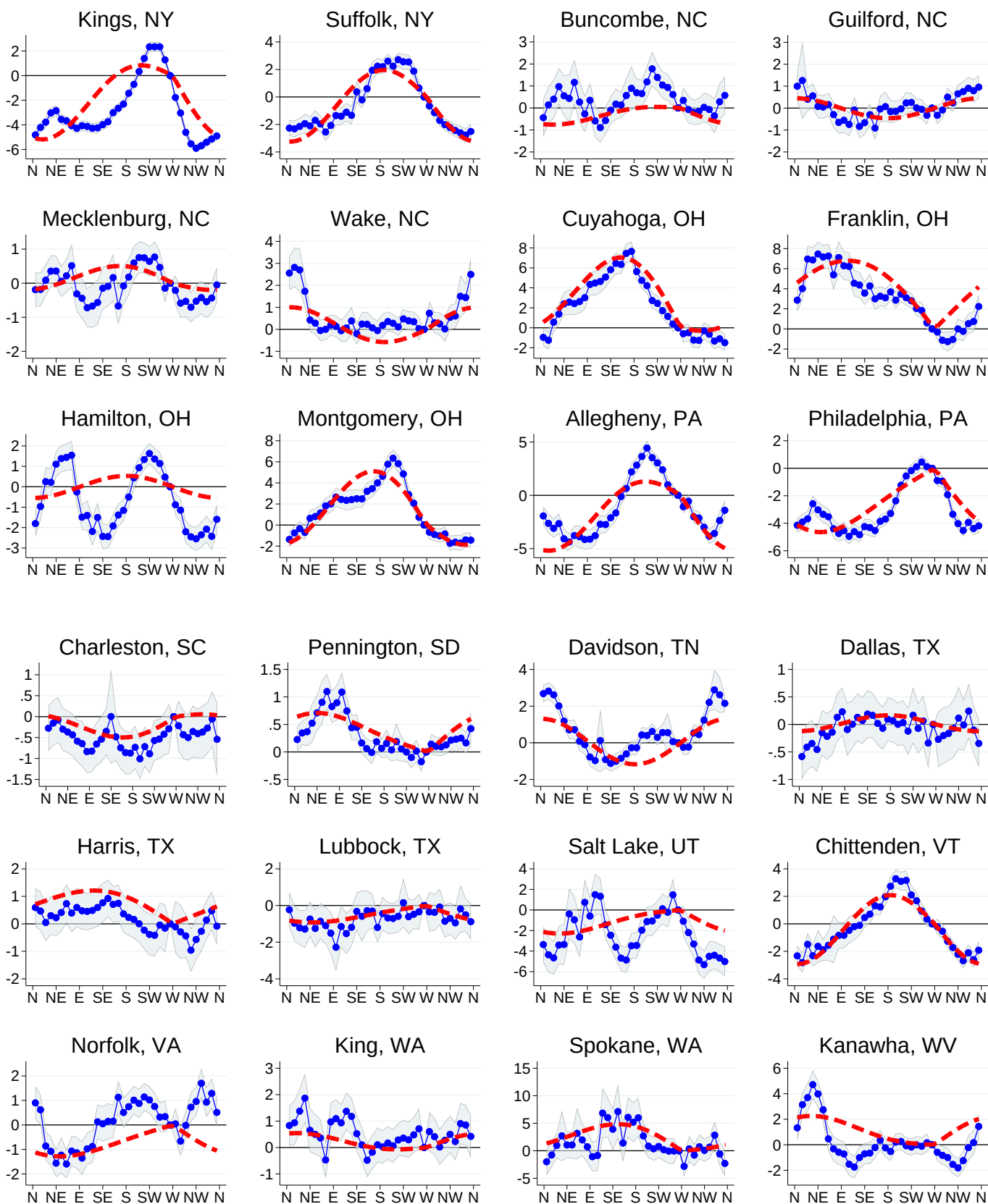
Figure A.3: SO₂ monitor locations and geographic group assignments

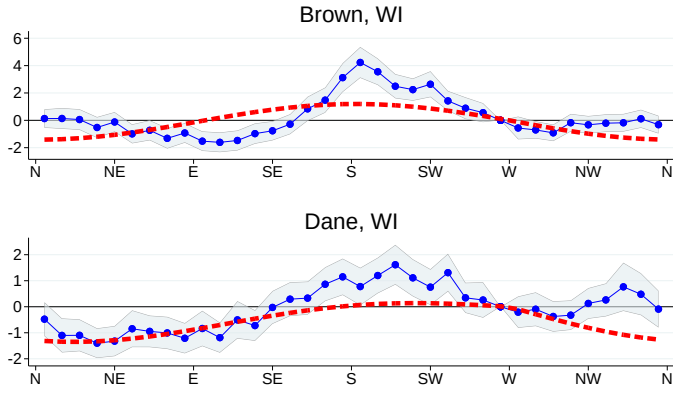


Notes: This map shows the 50 geographic groups included in our main estimation sample, each shaded in a different color. Black dots indicate the locations of SO₂ monitors. Unshaded (white) counties are excluded from the sample. The Southern California and Greater Philadelphia groups are shown in greater detail in Figure 1. As specified in Equation (2), the first-stage effect of wind direction on air pollution levels is allowed to vary across geographic groups.

Figure A.4: The relationship between wind direction and SO₂ levels, by geographic group

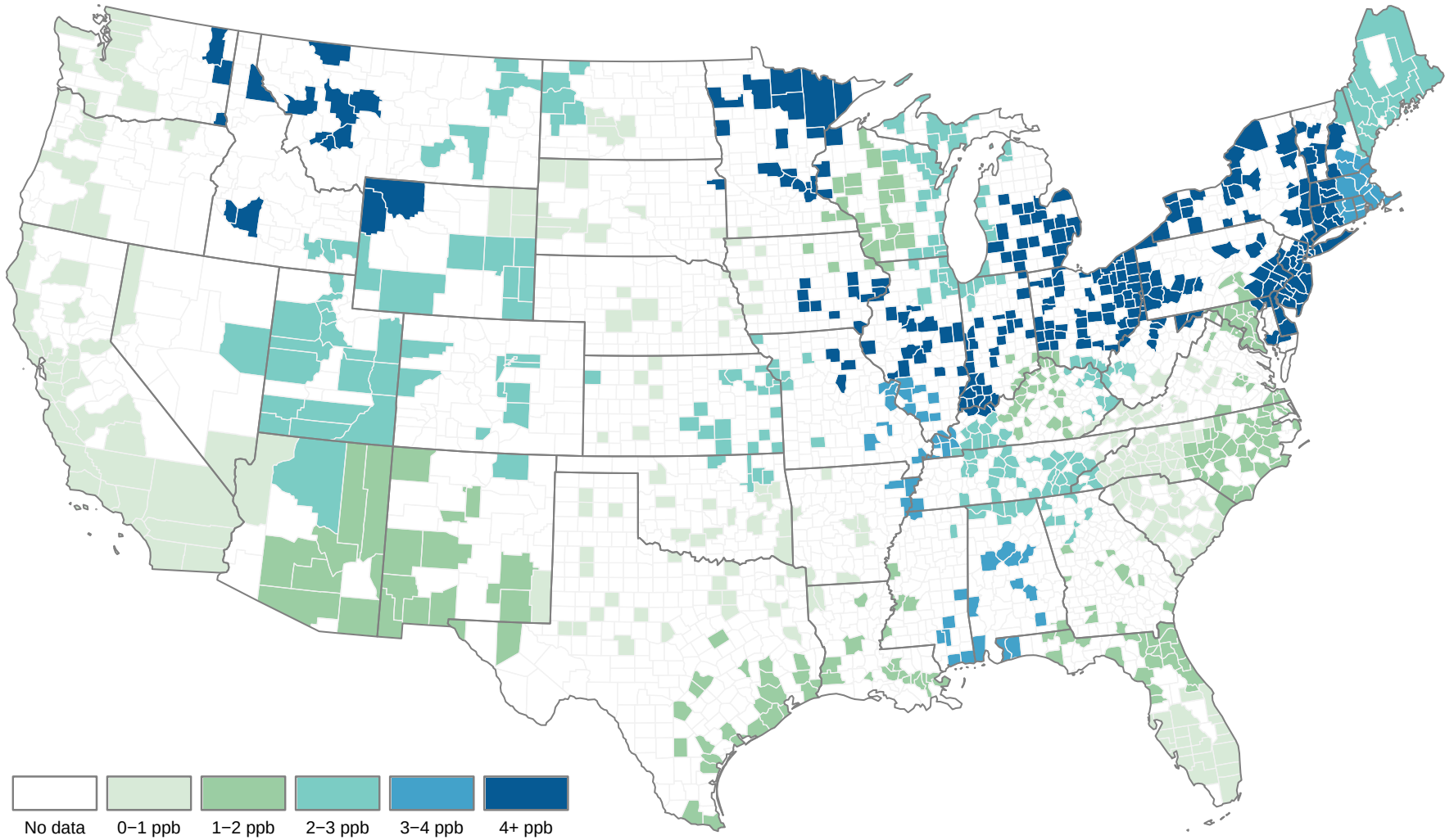






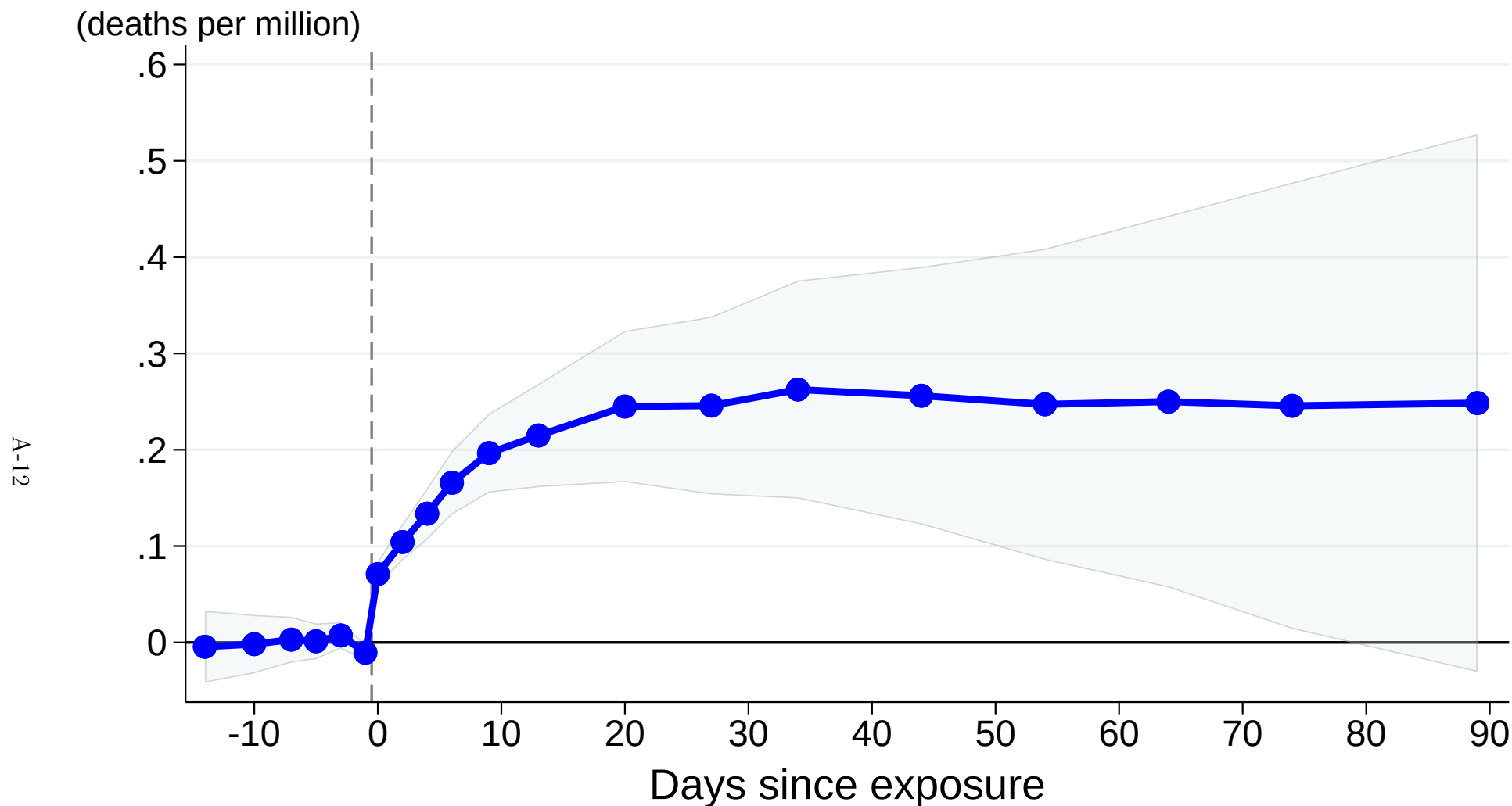
Notes: The graphs plot the relationship between SO₂ levels and windward direction for each of the geographic groups shown in Figure A.3. Windward direction refers to the direction the wind is coming from (e.g., “N” for wind blowing from the North and “NE” for wind blowing from the Northeast). The 36 blue points report coefficient estimates from a non-parametric regression of SO₂ levels on wind direction, grouped into 10-degree angle bins. The shaded areas denote 95% confidence intervals. Red dashed lines report fitted values from the parametric sine-based specification given by $f^g(\theta)$ in Equation (2). All regressions include county-by-month and month-by-year fixed effects, as well as flexible controls for maximum temperature, precipitation, humidity, and wind speed; and two leads and two lags of the instruments. Standard errors are robust to heteroskedasticity. The plots for “Philadelphia, PA” and “Los Angeles, CA” are reproduced in Figure 1 with the labels “Greater Philadelphia area” and “Southern California area.”

Figure A.5: Strength of the first stage, by geographic group



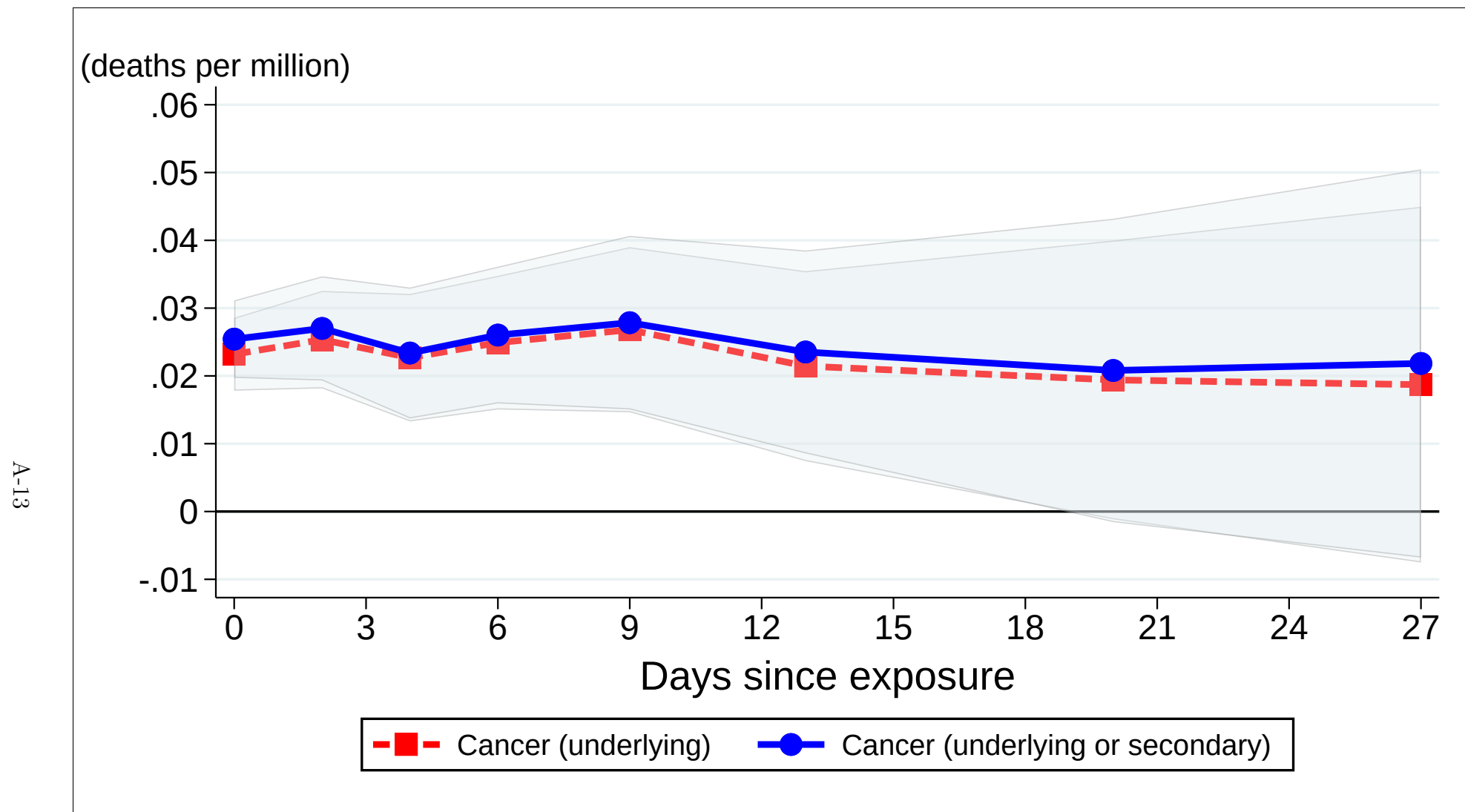
Notes: This map shows the strength of the first-stage relationship from Equation (2) for each of the 50 geographic groups included in our main estimation sample. Strength is measured as the difference in predicted SO_2 levels (in parts per billion) between the most and least polluting wind directions. Predictions are based on the parametric sine specification $\hat{f}^g(\theta) = \hat{\gamma}_g^1 \sin(\theta) + \hat{\gamma}_g^2 \sin(\theta/2)$, for $\theta \in [0, 2\pi)$. Unshaded (white) counties are excluded from the sample.

Figure A.6: IV estimates for the effect of acute (1-day) SO₂ exposure on cumulative mortality up to 90 days following exposure



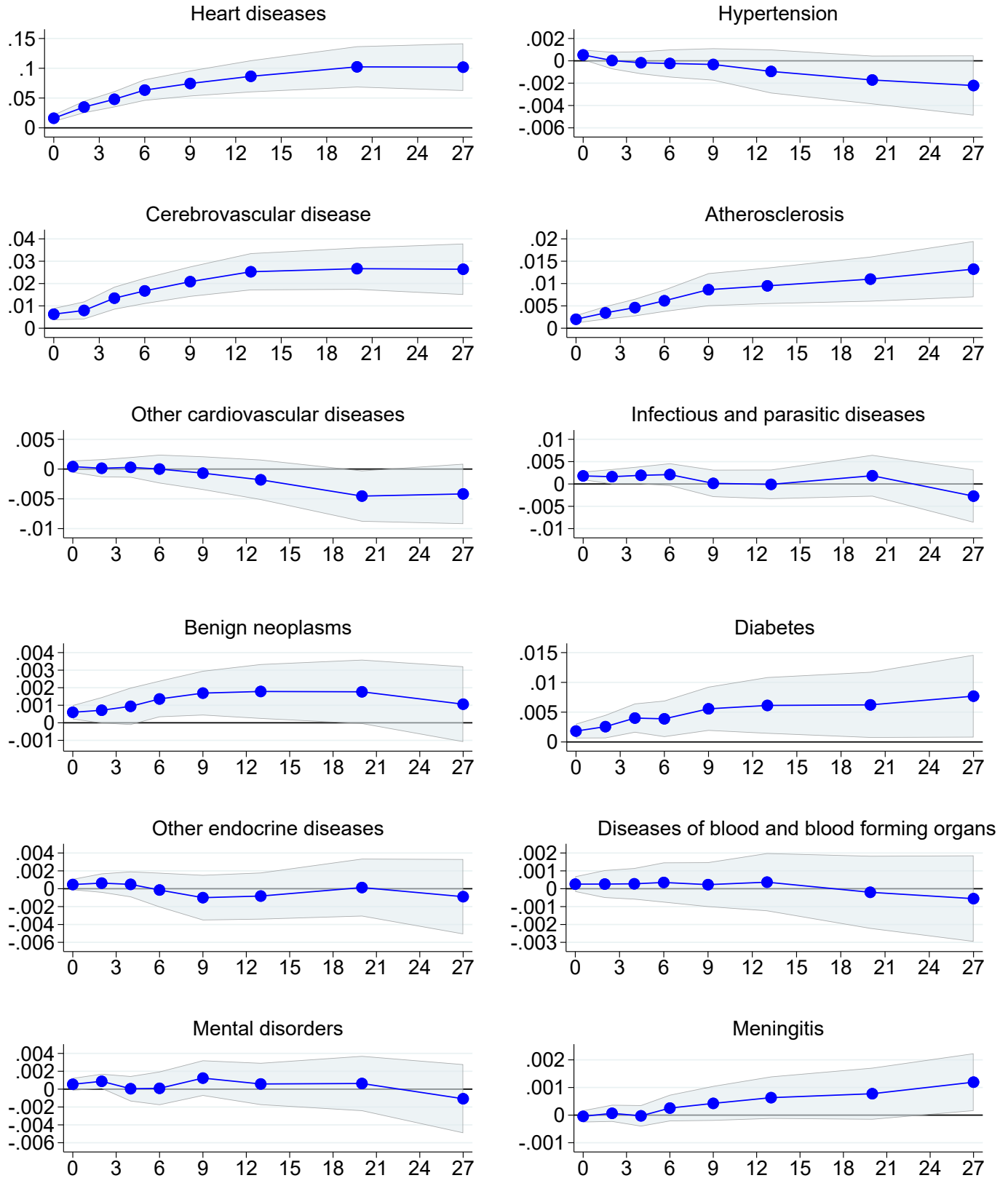
Notes: Each point reports an IV estimate from Equation (1) for the effect of a 1-day, 1 ppb increase in sulfur dioxide (SO₂) exposure on mortality, measured as cumulative deaths per million over time windows ranging from 14 days before to 89 days after exposure (with 0 indicating the day of exposure). The shaded areas denote 95% confidence intervals. Corresponding point estimates for $t = 0$ through $t = 27$ are reported in Column (2) of Table A.5. All regressions include county-by-month and month-by-year fixed effects, as well as flexible controls for maximum temperature, precipitation, humidity, and wind speed; leads of these weather controls (up to 27 leads); and two leads and two lags of the instruments. Estimates are weighted by county population. Standard errors are clustered by county.

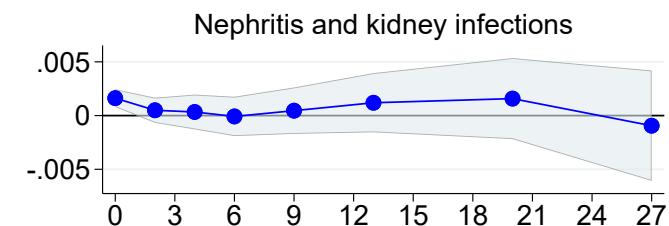
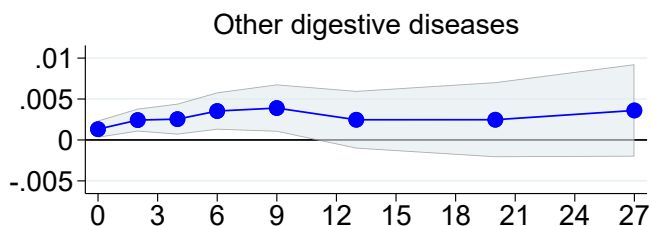
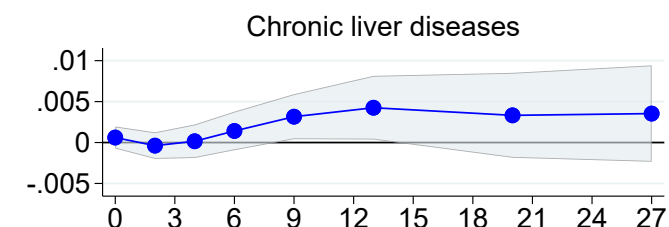
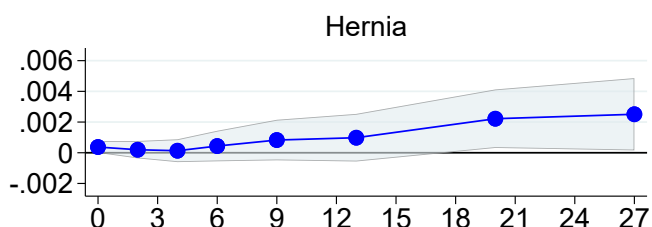
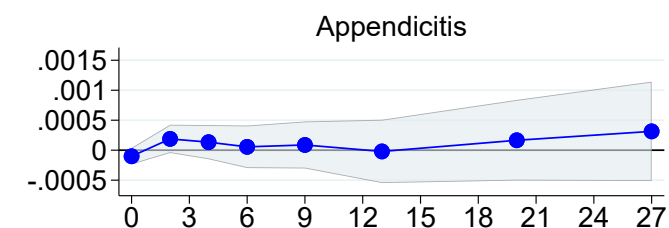
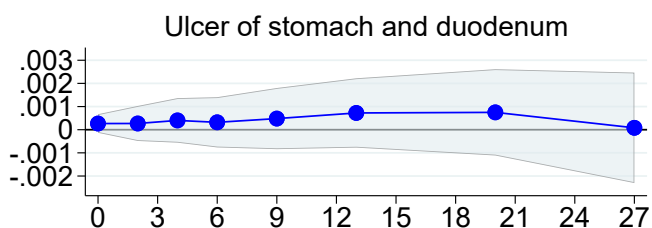
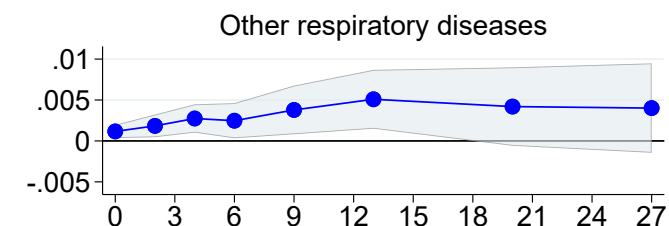
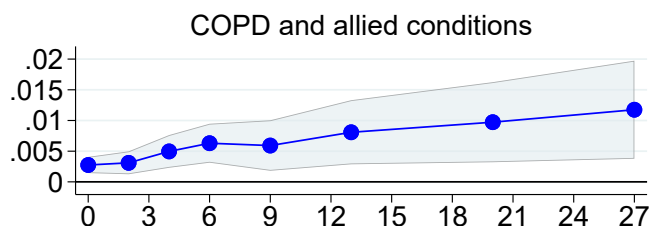
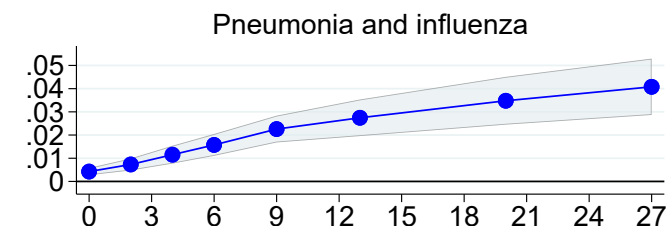
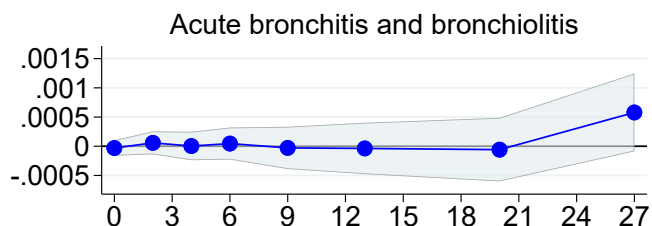
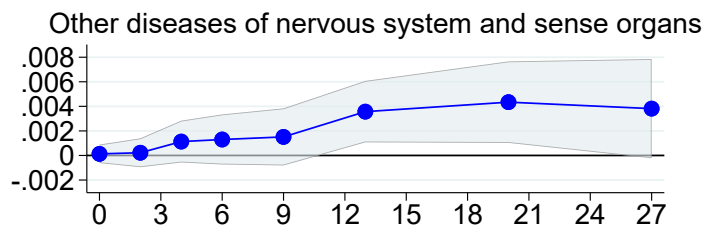
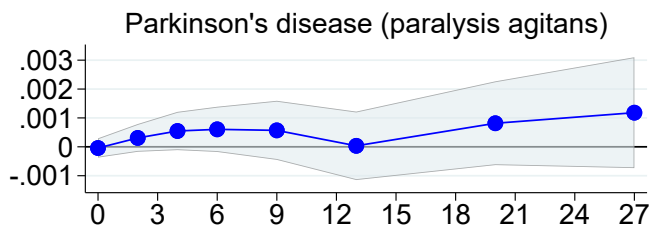
Figure A.7: IV estimates for the effect of acute (1-day) air pollution exposure on cancer-related mortality

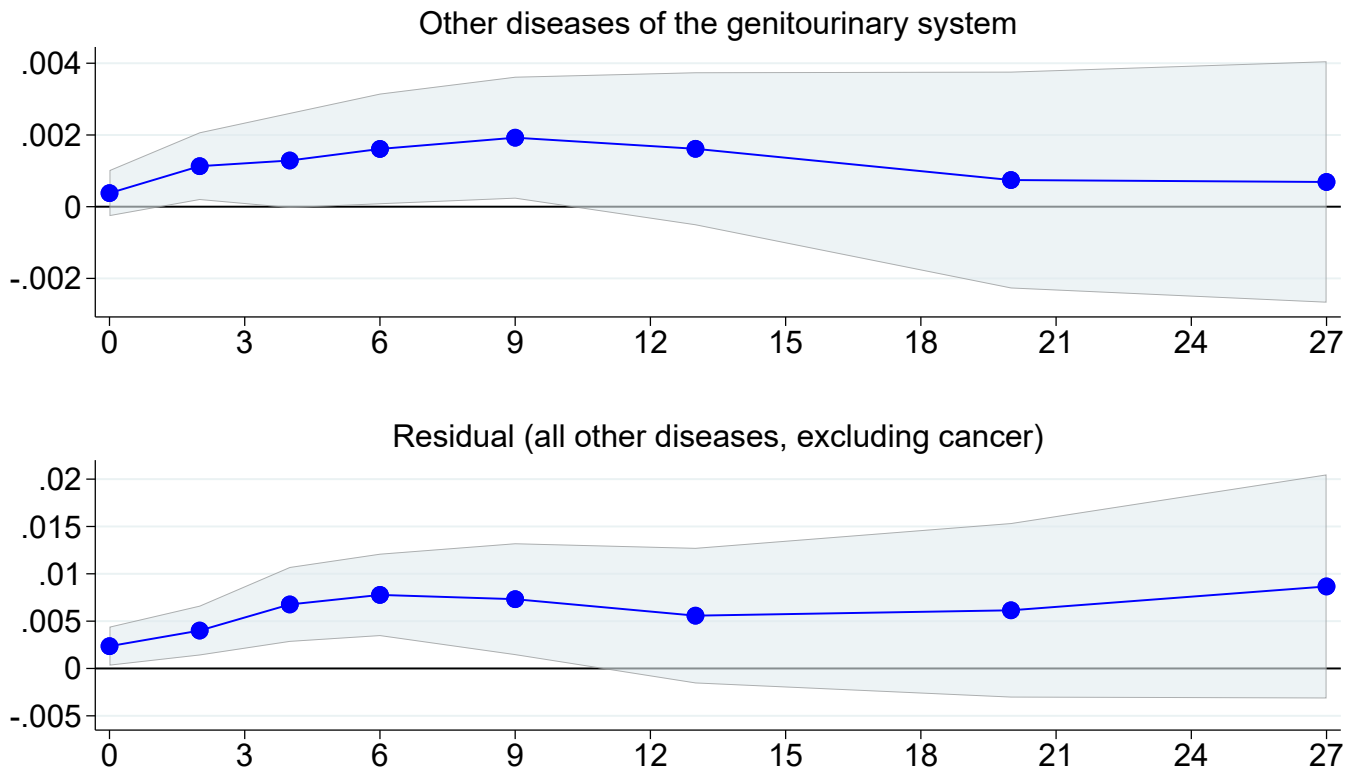


Notes: Each point reports an IV estimate from Equation (1) for the effect of acute (1-day) sulfur dioxide (SO_2) exposure on cancer-related mortality (deaths per million). “Cancer (underlying)”, which replicates the cancer estimates shown in Figure 3, includes only deaths where cancer is listed as the underlying cause of death on the death certificate. “Cancer (underlying or secondary)” expands this definition to include deaths where cancer is listed either as the underlying cause or as a contributing secondary cause. Mortality is measured as cumulative deaths per million over time windows ranging from the day of exposure to 27 days after exposure (with 0 indicating the day of exposure). The shaded areas denote 95% confidence intervals. All regressions include county-by-month and month-by-year fixed effects, as well as flexible controls for maximum temperature, precipitation, humidity, and wind speed; leads of these weather controls; and two leads and two lags of the instruments. Estimates are weighted by county population. Standard errors are clustered by county.

Figure A.8: IV estimates for the effect of acute (1-day) air pollution exposure on mortality, by detailed cause of death



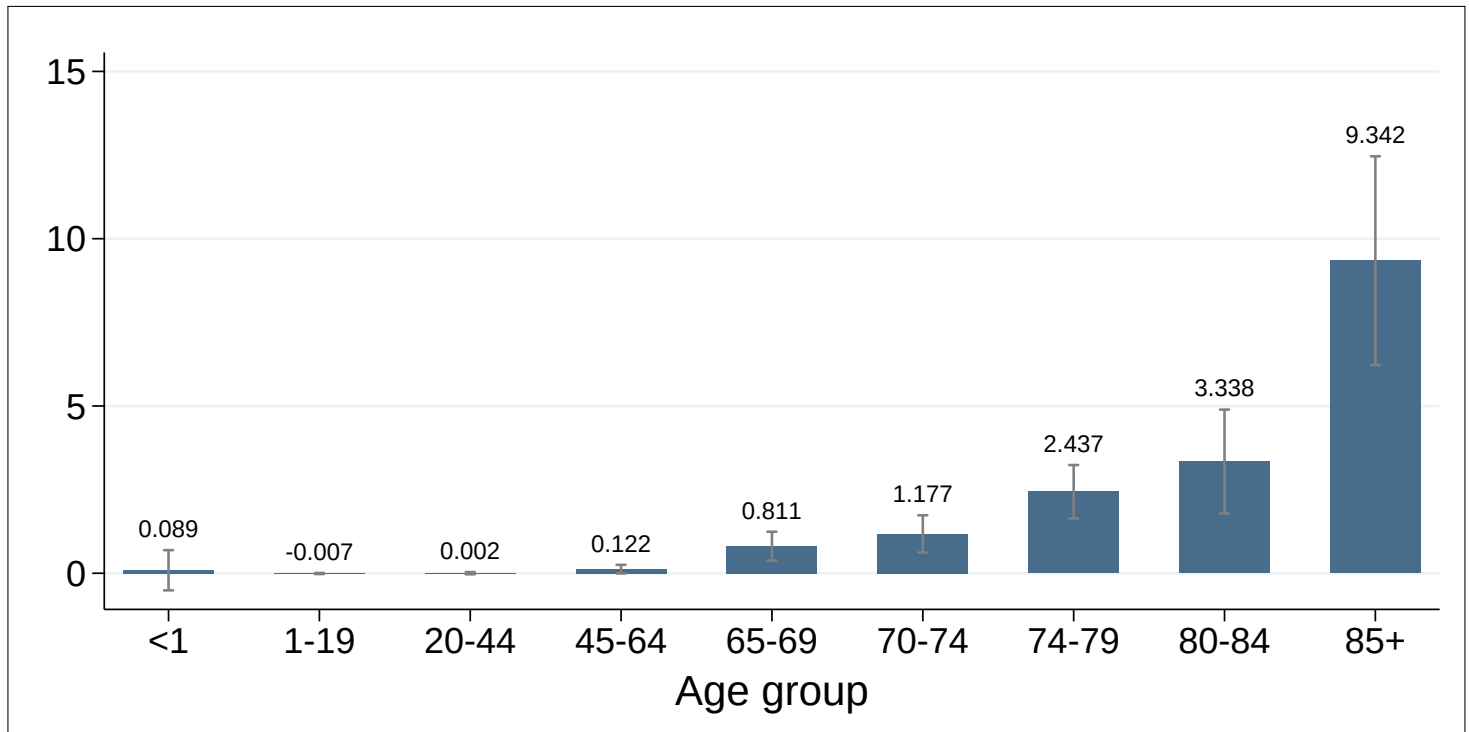




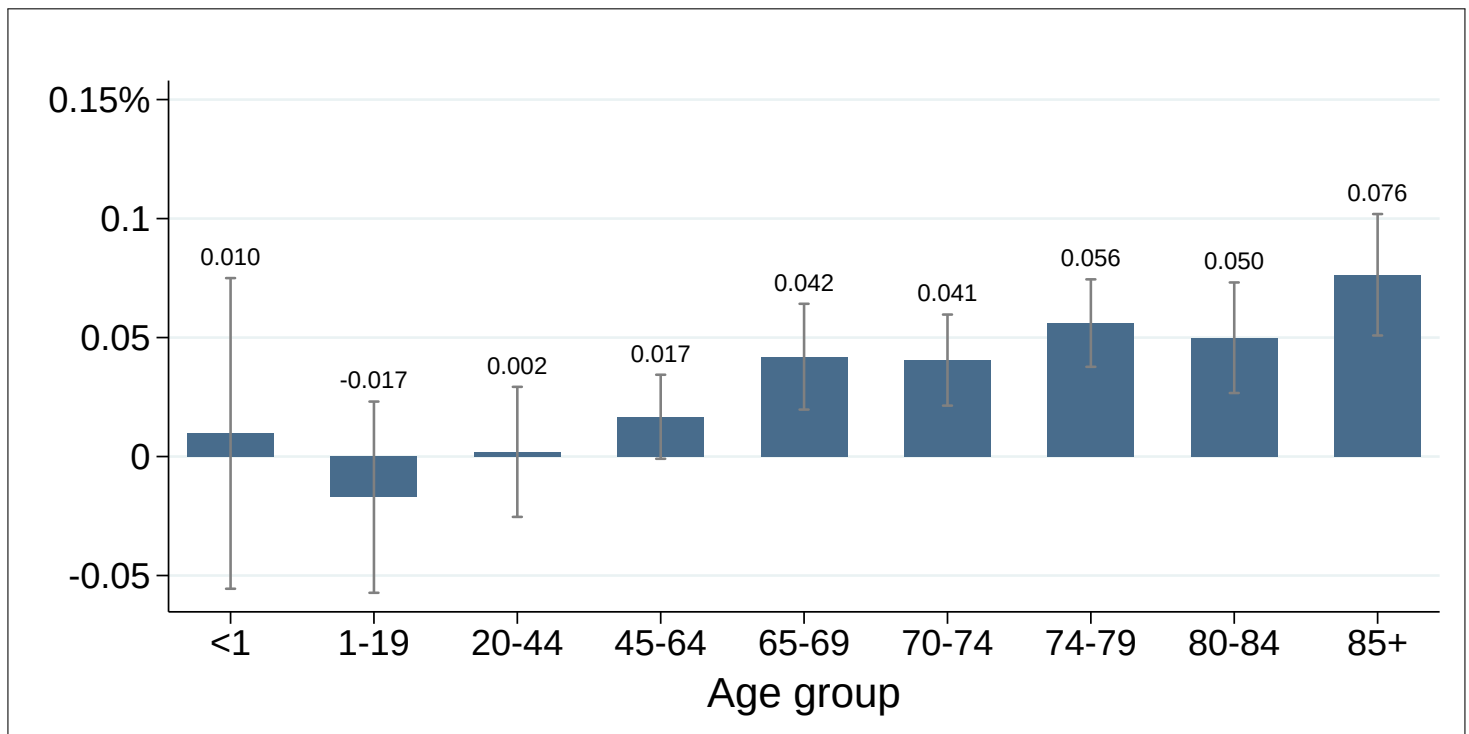
Notes: Each point reports an IV estimate from Equation (1) for the effect of a 1-day, 1 ppb increase in sulfur dioxide (SO_2) exposure on mortality from twenty-six different causes of death. Cause-of-death definitions are available in Table A.15. Mortality is measured as cumulative deaths per million over time windows ranging from the day of exposure to 27 days after exposure. The shaded areas denote 95% confidence intervals. All regressions include county-by-month and month-by-year fixed effects, as well as flexible controls for maximum temperature, precipitation, humidity, and wind speed; leads of these weather controls; and two leads and two lags of the instruments. Estimates are weighted by county population. Standard errors are clustered by county.

Figure A.9: IV estimates for the effect of acute (1-day) air pollution exposure on 28-day mortality, by age group

(a) Absolute increase (deaths per million)

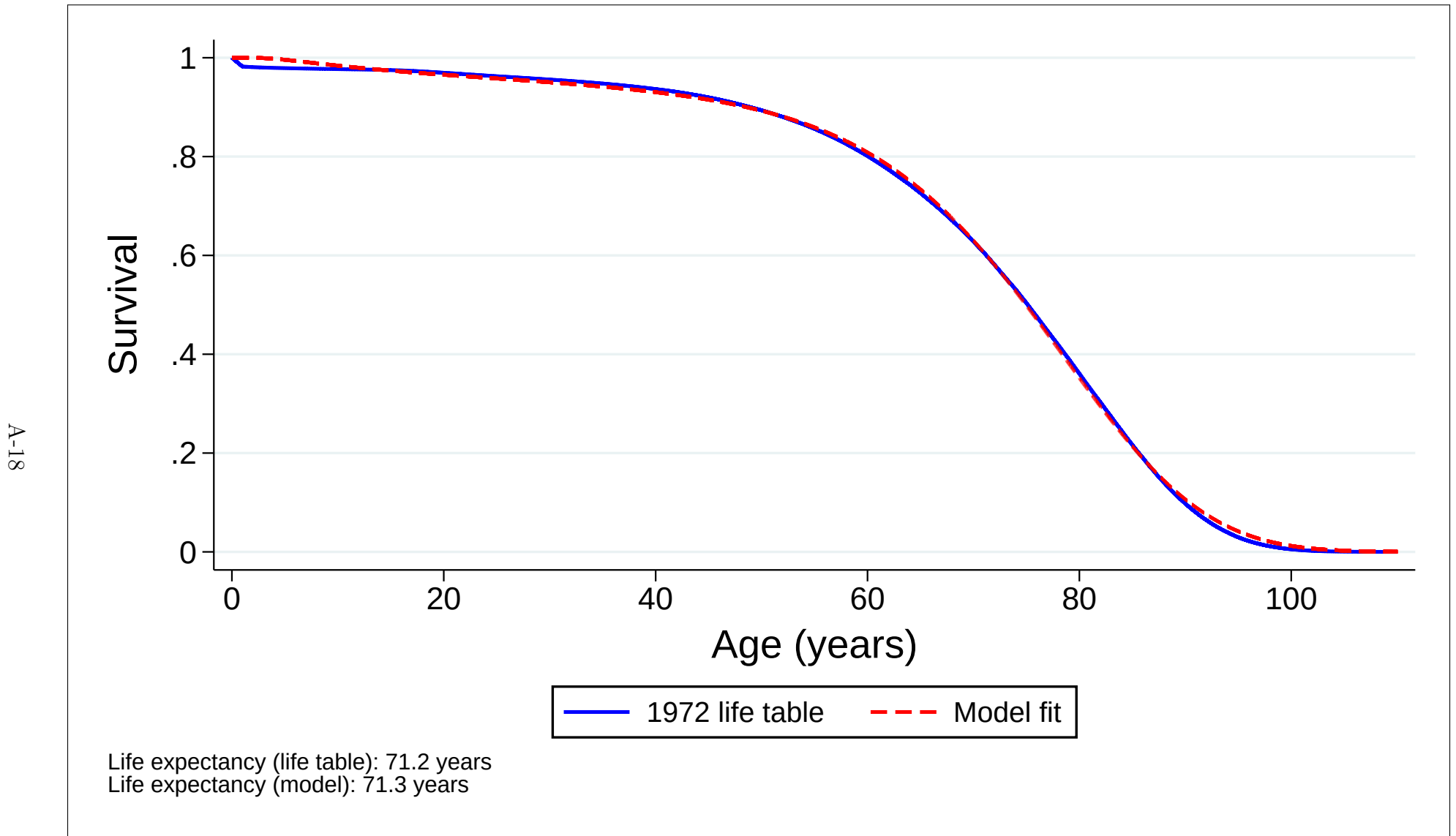


(b) Relative increase (percent of 28-day mortality)



Notes: Each bar represents an IV estimate from Equation (1) for the effect of a 1-day, 1 ppb increase in sulfur dioxide (SO_2) exposure on 28-day mortality for a particular age group. Error bars represent 95% confidence intervals. Estimates are also reported in Table A.7. All regressions include county-by-month and month-by-year fixed effects, as well as flexible controls for maximum temperature, precipitation, humidity, and wind speed; leads of these weather controls; and two leads and two lags of the instruments. Estimates are weighted by county population. Standard errors are clustered by county.

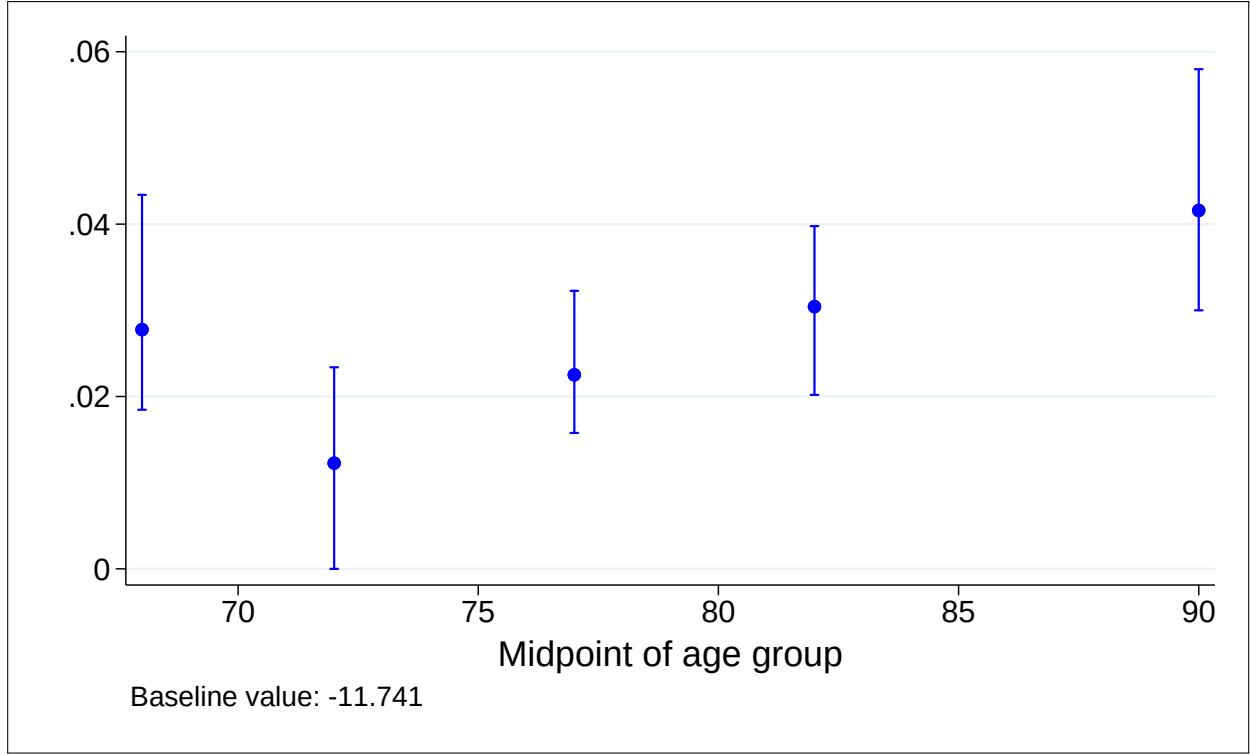
Figure A.10: Baseline calibration of the dynamic production model of health



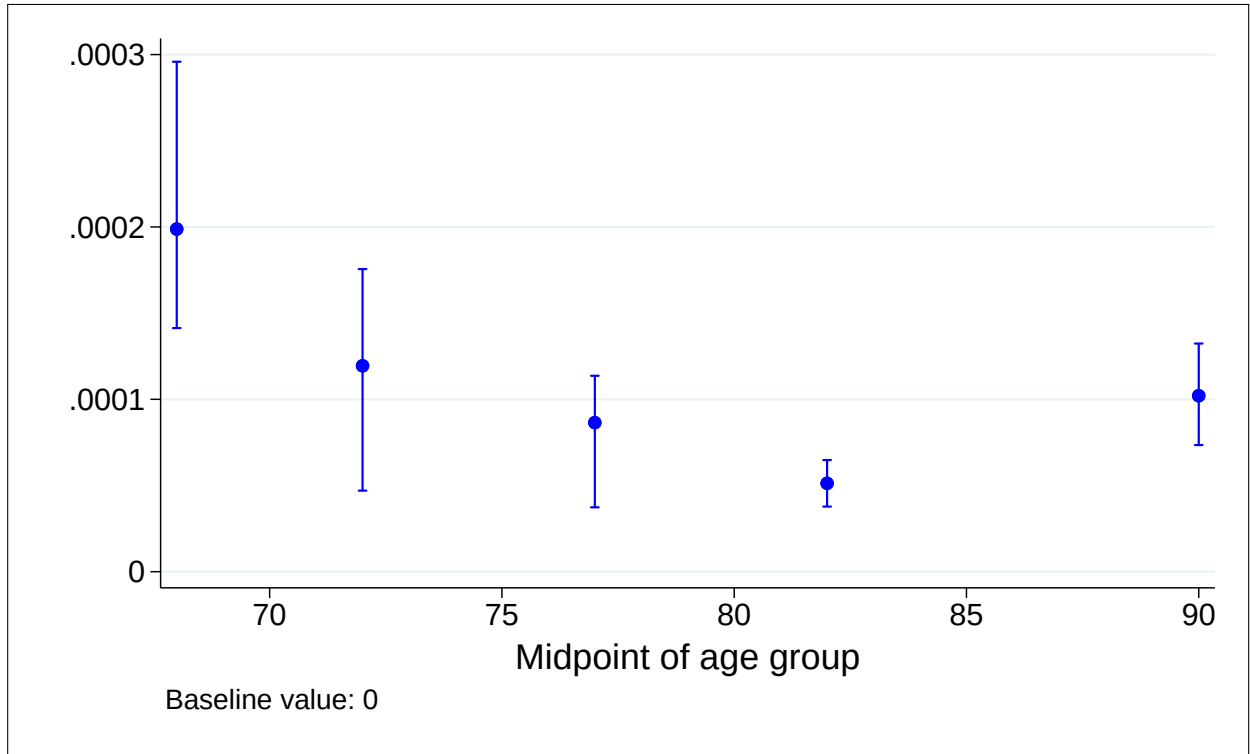
Notes: The solid blue line depicts the survival curve derived from the 1972 period life table for the United States. The dashed red line reports the predicted survival curve produced by our dynamic production model of health (3), which was calibrated using the 1972 life table data. The corresponding calibrated model parameters are reported in Column (2) of Table A.16.

Figure A.11: The effect of acute (1-day) air pollution exposure on model parameters

(a) Increase in $\ln(\delta)$ ($\ln(\tilde{\delta}_a) - \ln(\delta^*)$)



(b) Increase in $\underline{H}$ ($\underline{\tilde{H}}_a - \underline{H}^*$)

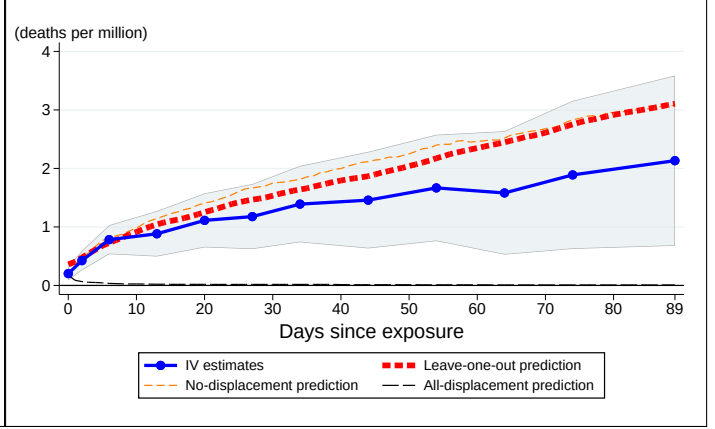
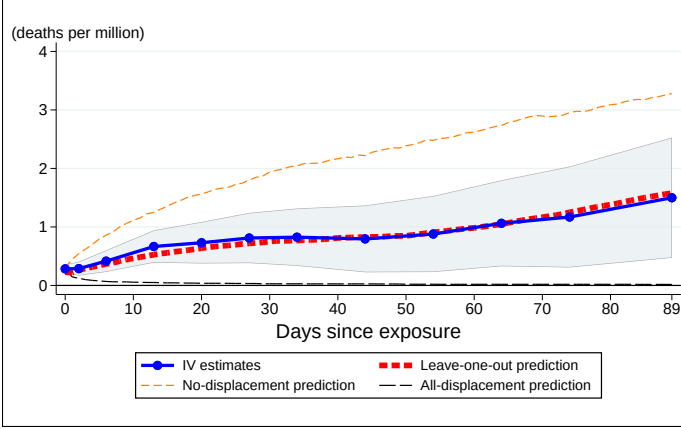


Notes: Panel (a) reports the effect of a 1-day, 1 ppb increase in SO_2 exposure on the change in the parameter δ from the health model (3), assuming all non-cancer deaths reflect changes in δ while all cancer deaths reflect changes in $\underline{H}$. The baseline value is denoted by δ^* , and the calibrated post-exposure value for age group a is denoted by $\tilde{\delta}_a$. Panel (b) reports the corresponding effect on the parameter $\underline{H}$, which governs mortality displacement and is calibrated using cancer-related deaths. The error bars report the 5th and 95th percentiles from 100 bootstrap replications. Baseline parameter values are reported in Column (2) of Table A.16.

Figure A.12: Comparison of model predictions to IV estimates, by age group

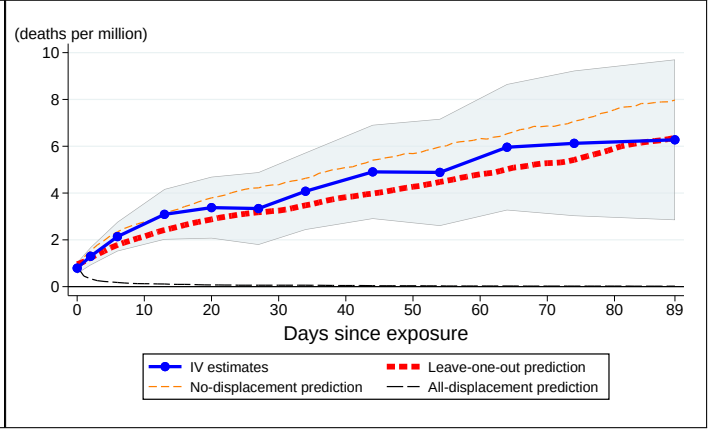
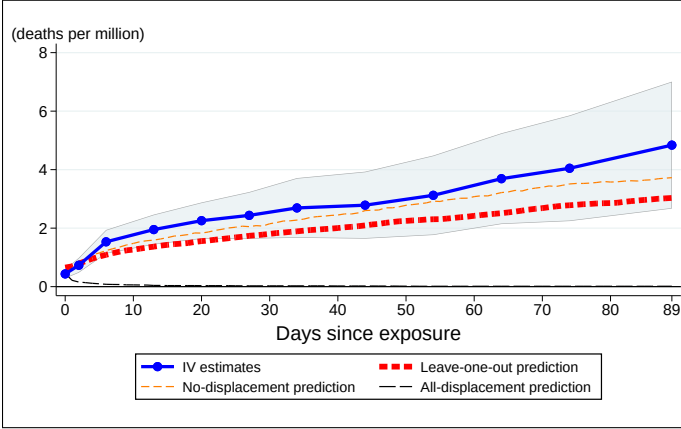
(a) Ages 65–69

(b) Ages 70–74

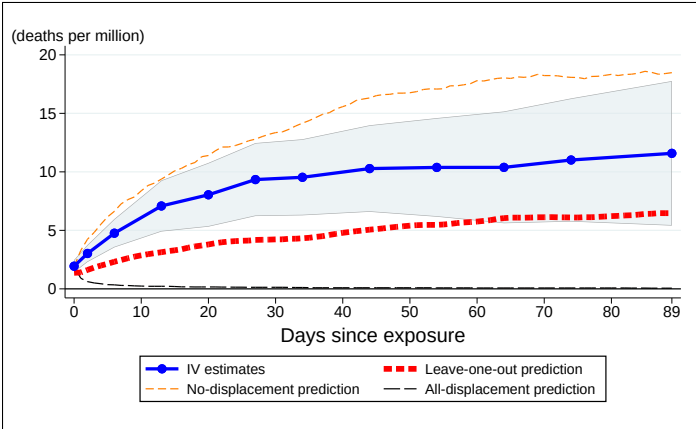


(c) Ages 75–79

(d) Ages 80–84

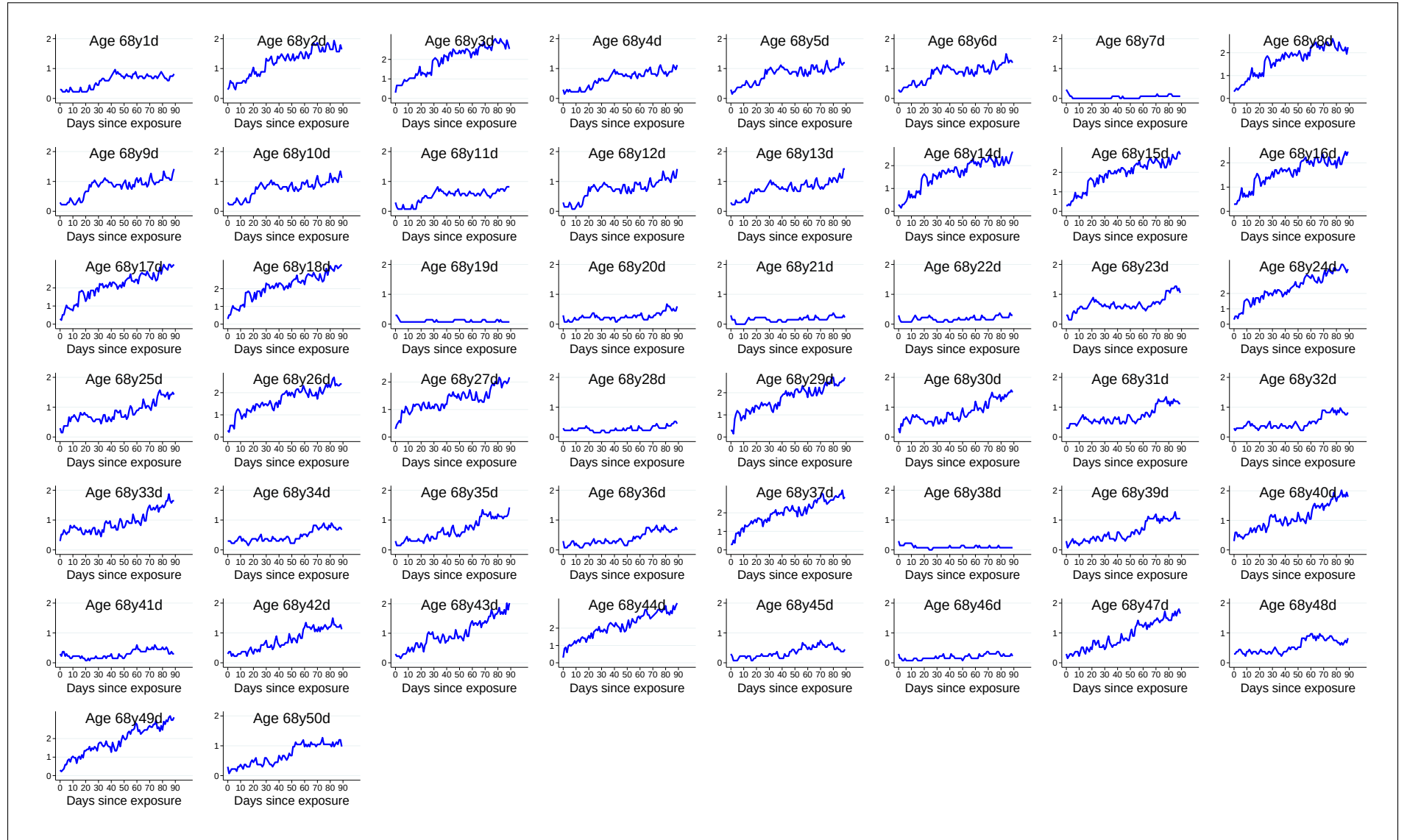


(e) Ages 85+



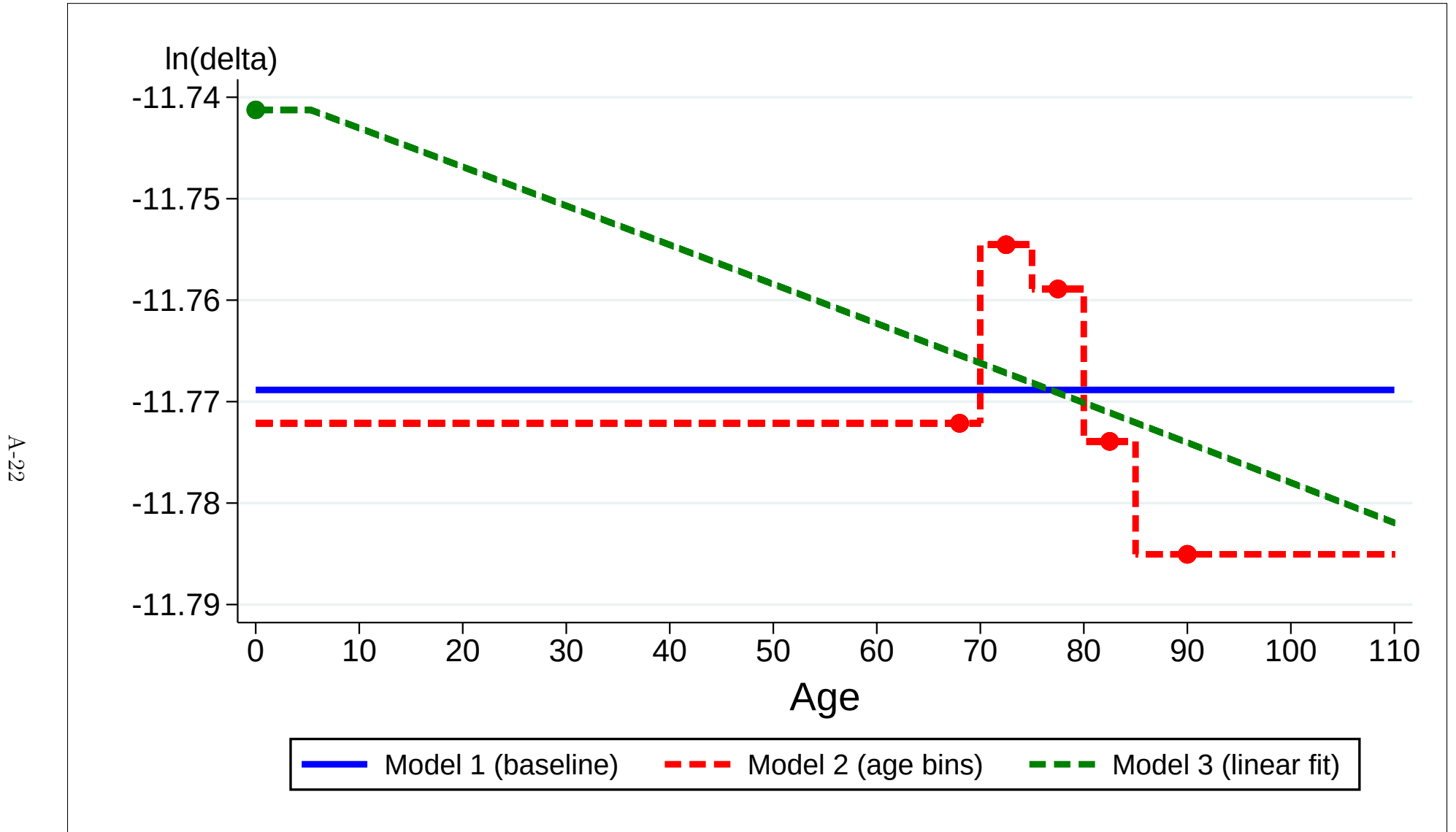
Notes: The solid blue line reports IV estimates from Equation (1) for the effect of a 1-day, 1 ppb increase in SO_2 exposure on cumulative mortality, with 95% confidence intervals given by the blue shaded area. The thick red dashed line (“leave-one-out”) reports model predictions using the average of the calibrated values from other older age groups (70–74, 75–79, 80–84, and 85+). The orange dashed line (“no-displacement”) at the top of the plot reports model predictions under the assumption that none of the 1-day mortality effect reflects mortality displacement, while the black dashed line (“all-displacement”) at the bottom of the plot assumes that the entire 1-day effect reflects mortality displacement.

Figure A.13: Predicted effects of acute (1-day) air pollution exposure on cumulative mortality, for selected ages



Notes: Each plot corresponds to a different age (68 years and 1 day, 68 years and 2 days, etc.) from the dynamic production model of health (3). The plots show the predicted effect of a 1-day, 1 ppb increase in SO₂ exposure on mortality, measured as cumulative deaths per million over time windows ranging up to 89 days after exposure (with 0 indicating the day of exposure). The average across these 50 plots corresponds to the green dot-dashed line (“own-age prediction”) in Figure 5.

Figure A.14: Values of $\tilde{\delta}$ used across models for long-run survival projections



Notes: This figure shows the counterfactual values $\tilde{\delta}$ assumed by the different models producing the long-run survival projections shown in Figure 7. The five red markers report the log of $\tilde{\delta}_a$ from the age-specific calibrations. The “baseline” specification uses the average of these five values, $\{\frac{1}{5} \sum_{a \geq 65} \tilde{\delta}_a\}$, represented by the solid blue line. The “age bins” model uses separate parameter values for each older age group (65–69, 70–74, 75–79, 80–84, and 85+) and assigns the 65–69 values to younger ages. The “linear fit” model assumes the effect is zero at birth (age 0) and fits a line through the five calibrated values and the age-zero point, subject to the constraint that exposure cannot improve health. The same approach is used to assign counterfactual values for $\tilde{H}$.

Table A.1: Complier county characteristics for the wind direction instruments

	Population	Percent 65+	Percent Black	Per- capita income (dollars)	Per- capita transfers (dollars)	Employment rate	Mean SO ₂ (ppb)
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
First-stage strength, ppb	−152,394 (142,214)	0.19* (0.088)	−0.24 (0.33)	62 (76)	55** (12)	−0.67 (0.78)	1.6** (0.16)
Mean outcome	1,303,561	11	13	10,987	1,290	52	9.5
Sample size	9,497	9,497	9,365	9,356	9,356	9,356	9,497
R-squared	0.042	0.074	0.0055	0.79	0.74	0.042	0.41

Notes: Complier characteristics are estimated by regressing the county-level variable reported at the top of each column on the strength of the first stage. First-stage strength is measured as the difference in predicted SO₂ levels between the most and least polluting wind directions. All regressions include year fixed effects and are weighted by county population. County characteristics are obtained from the Regional Economic Information System dataset published by the Bureau of Economic Analysis. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level.

Table A.2: Autocorrelation in wind-driven SO₂ exposure, for different numbers of instrument leads and lags

	(1)	(2)	(3)	(4)	(5)
SO ₂ , ppb	0.27** (0.020)	0.033* (0.015)	0.017 (0.013)	0.018 (0.012)	0.021 (0.012)
Number of instrument leads	0	1	1	2	3
Number of instrument lags	0	0	1	2	3
First-stage F -statistic	751	654	602	569	570
Sample size	1,865,352	1,865,352	1,865,352	1,865,352	1,864,462

Notes: The dependent variable is tomorrow's SO₂ levels. We instrument for today's SO₂ levels using today's wind direction instruments. All regressions include county-by-month and month-by-year fixed effects, along with flexible controls for both today's and tomorrow's maximum temperature, precipitation, humidity, and wind speed. Columns (2)–(5) include leads and lags of the instruments as controls. All regressions are weighted by county population. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level.

Table A.3: IV estimates for the effect of acute SO₂ exposure on 1-day mortality, using different numbers of instrument leads and lags

	(1)	(2)	(3)	(4)	(5)	(6)
SO ₂ , ppb	0.071** (0.0067)	0.075** (0.0071)	0.075** (0.0068)	0.071** (0.0067)	0.071** (0.0067)	0.070** (0.0066)
Number of instrument leads	2	0	1	3	4	6
Number of instrument lags	2	0	1	3	4	6
First-stage <i>F</i> -statistic	623	845	661	625	621	621
Mean outcome	24	24	24	24	24	24
Sample size	2,043,563	2,043,563	2,043,563	2,042,619	2,041,682	2,039,854

Notes: The dependent variable is number of deaths per million people on the day of exposure. All regressions include county-by-month and month-by-year fixed effects, along with flexible controls for maximum temperature, precipitation, humidity, and wind speed. Columns (1)–(6) include differing numbers of leads and lags of the instruments as controls. All regressions are weighted by county population. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level.

Table A.4: IV estimates for the effect of acute SO₂ exposure on 1-day mortality, using different numbers of weather leads and lags

	(1)	(2)	(3)	(4)	(5)
SO ₂ , ppb	0.071** (0.0067)	0.040** (0.0060)	0.044** (0.0061)	0.063** (0.0065)	0.057** (0.0064)
Number of weather leads	0	0	0	1	2
Number of weather lags	0	1	2	0	0
First-stage <i>F</i> -statistic	623	522	529	595	576
Mean outcome	24	24	24	24	24
Sample size	2,043,563	2,043,371	2,043,171	2,043,563	2,043,563
Number of weather controls	1,550	3,105	4,648	3,098	4,643

Notes: The dependent variable is number of deaths per million people on the day of exposure. All regressions include county-by-month and month-by-year fixed effects, along with flexible controls for maximum temperature, precipitation, humidity, and wind speed; and two leads and two lags of the instruments. All regressions are weighted by county population. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level.

Table A.5: OLS and IV estimates for the effect of acute SO₂ exposure on cumulative mortality, for different outcome windows

	(1)	(2)
Outcome window	OLS	IV
1 day	0.0084** (0.0028)	0.071** (0.0067)
3 days	0.021* (0.0084)	0.10** (0.0096)
5 days	0.030* (0.014)	0.13** (0.014)
7 days	0.034 (0.019)	0.17** (0.017)
10 days	0.040 (0.027)	0.20** (0.021)
14 days	0.047 (0.037)	0.21** (0.027)
21 days	0.054 (0.053)	0.24** (0.040)
28 days	0.054 (0.069)	0.25** (0.047)

Notes: The dependent variable is cumulative number of deaths per million people in the days following acute (1-day) exposure. Each estimate comes from a separate regression. IV estimates in Column (2) correspond to those shown in Figure 2. All regressions include county-by-month and month-by-year fixed effects, along with flexible controls for maximum temperature, precipitation, humidity, and wind speed; leads of these weather controls; and two leads and two lags of the instruments. All regressions are weighted by county population. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level.

Table A.6: IV estimates for the effect of acute SO₂ exposure on 1-day mortality, for different age groups

	(1)	(2)
Age group	Absolute effect, deaths per million	Relative effect, percent
0–1	0.014 (0.043)	0.041 (0.13)
1–19	0.00047 (0.0015)	0.031 (0.10)
20–44	0.0090** (0.0033)	0.21** (0.075)
45–64	0.047** (0.010)	0.18** (0.039)
65–69	0.28** (0.038)	0.41** (0.055)
70–74	0.20** (0.060)	0.20** (0.058)
75–79	0.44** (0.078)	0.28** (0.050)
80–84	0.79** (0.12)	0.33** (0.052)
85+	1.9** (0.27)	0.44** (0.062)

Notes: The dependent variable is number of deaths per million people on the day of exposure. Each estimate comes from a separate regression. The relative effect is expressed as a percentage of the age group's mean one-day mortality rate. Estimates are also shown in panels (a) and (b) of Figure 4. All regressions include county-by-month and month-by-year fixed effects, along with flexible controls for maximum temperature, precipitation, humidity, and wind speed; and two leads and two lags of the instruments. All regressions are weighted by county population. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level.

Table A.7: IV estimates for the effect of acute SO₂ exposure on cumulative mortality, for different age groups and outcome windows

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Outcome window	0–1	1–19	20–44	45–64	65–69	70–74	75–79	80–84	85+
1 day	0.014 (0.043)	0.00047 (0.0015)	0.0090** (0.0033)	0.047** (0.010)	0.28** (0.038)	0.20** (0.060)	0.44** (0.078)	0.79** (0.12)	1.9** (0.27)
3 days	0.16** (0.057)	0.00064 (0.0029)	0.012** (0.0045)	0.061** (0.015)	0.29** (0.061)	0.42** (0.085)	0.73** (0.13)	1.3** (0.20)	3.0** (0.37)
7 days	0.18 (0.11)	−0.0017 (0.0052)	0.027** (0.0072)	0.067** (0.022)	0.42** (0.094)	0.78** (0.13)	1.5** (0.21)	2.1** (0.32)	4.8** (0.62)
14 days	0.013 (0.19)	−0.0071 (0.0059)	0.021* (0.0091)	0.088* (0.038)	0.67** (0.14)	0.88** (0.20)	2.0** (0.26)	3.1** (0.55)	7.1** (1.1)
28 days	0.089 (0.31)	−0.0071 (0.0086)	0.0024 (0.017)	0.12 (0.066)	0.81** (0.22)	1.2** (0.28)	2.4** (0.41)	3.3** (0.79)	9.3** (1.6)

Notes: The dependent variable is cumulative number of deaths per million people in the days following acute (1-day) exposure. Each estimate comes from a separate regression. Age groups are indicated at the top of each column. All regressions include county-by-month and month-by-year fixed effects, along with flexible controls for maximum temperature, precipitation, humidity, and wind speed; leads of these weather controls; and two leads and two lags of the instruments. All regressions are weighted by county population. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level.

Table A.8: IV estimates for the effect of acute SO₂ exposure on cumulative mortality, for different causes of death and outcome windows

	(1)	(2)	(3)	(4)
Outcome window	Cardiovascular	Cancer	Other	External
1 day	0.025** (0.0033)	0.023** (0.0027)	0.021** (0.0027)	0.0018 (0.0011)
3 days	0.047** (0.0056)	0.025** (0.0037)	0.028** (0.0034)	0.0044* (0.0018)
5 days	0.066** (0.0076)	0.023** (0.0048)	0.040** (0.0056)	0.0045 (0.0026)
7 days	0.086** (0.010)	0.025** (0.0050)	0.049** (0.0070)	0.0054 (0.0028)
10 days	0.10** (0.012)	0.027** (0.0062)	0.061** (0.0089)	0.0060* (0.0031)
14 days	0.12** (0.016)	0.021** (0.0071)	0.070** (0.011)	0.0053 (0.0037)
21 days	0.13** (0.021)	0.019 (0.010)	0.082** (0.016)	0.0093 (0.0057)
28 days	0.14** (0.023)	0.019 (0.013)	0.085** (0.020)	0.0067 (0.0072)

Notes: The dependent variable is cumulative number of deaths per million people in the days following acute (1-day) exposure. Each estimate comes from a separate regression. The underlying cause of death is indicated at the top of each column. Estimates are also shown in Figure 3. All regressions include county-by-month and month-by-year fixed effects, along with flexible controls for maximum temperature, precipitation, humidity, and wind speed; leads of these weather controls; and two leads and two lags of the instruments. All regressions are weighted by county population. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level.

Table A.9: IV estimates for the effect of acute SO₂ exposure on 1-day mortality, controlling for all pollutants except TSP

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
SO ₂ , ppb	0.082** (0.0099)	0.059** (0.014)	0.087** (0.012)	0.074** (0.011)	0.060** (0.013)	0.078** (0.012)	0.064** (0.015)
NO ₂ , ppb		0.029* (0.012)			0.026 (0.014)		0.028 (0.015)
O ₃ , ppb			-0.022 (0.017)			-0.016 (0.017)	-0.019 (0.018)
CO, ppm				0.27 (0.16)	0.050 (0.20)	0.25 (0.16)	0.0038 (0.22)
First-stage <i>F</i> -statistic	250	65	41	93	44	40	39
Mean outcome	26	26	26	26	26	26	26
Sample size	277,481	277,481	277,481	277,481	277,481	277,481	277,481

Notes: The dependent variable is number of deaths per million people on the day of exposure. All regressions include county-by-month and month-by-year fixed effects, along with flexible controls for maximum temperature, precipitation, humidity, and wind speed; and two leads and two lags of the instruments. All regressions are weighted by county population. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level. National means for all air pollutants are available in Table 1.

Table A.10: IV estimates for the effect of acute SO₂ exposure on 1-day mortality, using different fixed effects

	(1)	(2)	(3)	(4)	(5)	(6)
SO ₂ , ppb	0.071** (0.0067)	0.065** (0.0068)	0.071** (0.0069)	0.072** (0.0071)	0.057** (0.0062)	0.061** (0.0064)
Fixed effects	county-month, month-year	county, year, month	county, state-year-month	county, year, state-month	county-month, state-year-week	county-month, month-year, state-day-of-year
First-stage <i>F</i> -statistic	623	593	663	597	625	571
Mean outcome	24	24	24	24	24	24
Sample size	2,043,563	2,043,563	2,043,541	2,043,563	2,043,216	2,043,558

Notes: The dependent variable is number of deaths per million people on the day of exposure. All regressions include flexible controls for maximum temperature, precipitation, humidity, and wind speed; and two leads and two lags of the instruments. All regressions are weighted by county population. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level.

Table A.11: IV estimates for the effect of acute SO₂ exposure on 1-day mortality, clustering standard errors at different levels

	(1)	(2)	(3)	(4)
SO ₂ , ppb	0.071** (0.0067)	0.071** (0.0067)	0.071** (0.0067)	0.071** (0.0061)
Clustering level(s)	County	County, geographic group by year	Geographic group	State
First-stage F -statistic	623	623	623	623
Mean outcome	24	24	24	24
Sample size	2,043,563	2,043,563	2,043,563	2,043,563

Notes: The dependent variable is number of deaths per million people on the day of exposure. All regressions include county-by-month and month-by-year fixed effects, along with flexible controls for maximum temperature, precipitation, humidity, and wind speed; and two leads and two lags of the instruments. All regressions are weighted by county population. Standard errors, clustered at the level(s) indicated in each column, are reported in parentheses. Geographic groups are shown in Figure A.3. A */** indicates significance at the 5%/1% level.

Table A.12: IV estimates for the effect of acute SO₂ exposure on 1-day mortality, using different first-stage specifications

	(1)	(2)	(3)	(4)	(5)	(6)
SO ₂ , ppb	0.071** (0.0067)	0.083** (0.0077)	0.061** (0.0056)	0.060** (0.0051)	0.062** (0.0056)	0.066** (0.0061)
Number of geographic groups	50	25	100	50	50	50
Wind angle parameterization	Sines	Sines	Sines	40-degree bins	60-degree bins	90-degree bins
Number of instruments	100	50	200	400	250	150
First-stage F -statistic	623	1,090	364	179	248	337
Mean outcome	24	24	24	24	24	24
Sample size	2,043,563	2,043,563	2,043,563	2,043,563	2,043,563	2,043,563

Notes: The dependent variable is number of deaths per million people on the day of exposure. Column (1) reports our main specification, which allows the effect of wind direction to vary across 50 geographic groups and assumes that its effect on pollution, $f^g(\cdot)$, follows the sine parameterization in Equation (2). Columns (2)–(3) vary the number of geographic groups employed in the first stage. Columns (4)–(6) use alternative non-parametric parameterizations for $f^g(\cdot)$. All regressions include county-by-month and month-by-year fixed effects, along with flexible controls for maximum temperature, precipitation, humidity, and wind speed. All regressions are weighted by county population. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level.

Table A.13: 2SLS and LIML estimates for the effect of acute SO₂ exposure on cumulative mortality, for different outcome windows

	(1)	(2)	(3)	(4)	(5)	(6)
SO ₂ , ppb	0.071** (0.0067)	0.071** (0.0068)	0.17** (0.017)	0.17** (0.017)	0.25** (0.047)	0.25** (0.047)
IV method	2SLS	LIML	2SLS	LIML	2SLS	LIML
Outcome window (days)	1	1	7	7	28	28
First-stage <i>F</i> -statistic	623	623	567	567	509	509
Mean outcome	24	24	170	170	681	681
Sample size	2,043,563	2,043,563	2,043,563	2,043,563	2,043,563	2,043,563

Notes: The dependent variable is cumulative number of deaths per million people in the days following acute (1-day) exposure. All regressions include county-by-month and month-by-year fixed effects, along with flexible controls for maximum temperature, precipitation, humidity, and wind speed; leads of these weather controls; and two leads and two lags of the instruments. All regressions are weighted by county population. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level.

Table A.14: Placebo tests for the effect of acute SO₂ exposure on mortality

	(1)	(2)	(3)
SO ₂ , ppb	0.030 (0.060)	-0.33 (0.23)	-0.46 (0.49)
Outcome window, days	1	7	28
First-stage <i>F</i> -statistic	3.3	3.4	3.4
Mean outcome	24	170	681
Sample size	2,043,563	2,043,563	2,043,563

Notes: This table reports the results of placebo regressions where the instruments are based on wind direction that is a randomly generated variable. The dependent variable is cumulative number of deaths per million people in the days following acute (1-day) exposure. All regressions include county-by-month and month-by-year fixed effects, along with flexible controls for maximum temperature, precipitation, humidity, and wind speed; leads of these weather controls; and two leads and two lags of the instruments. All regressions are weighted by county population. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level.

Table A.15: ICD-8 and ICD-9 codes for subcategories of cardiovascular and other diseases

ID	Disease	ICD-8 codes (1968–1978)	ICD-9 codes (1979–1998)	Prevalence (%)
Cardiovascular diseases				
1	Heart diseases	390–398.9, 402–402.9, 404–404.9, 410–429.9	390–398.9, 402–402.9, 404–429.9	37.58
2	Hypertension	400–400.9, 401–401.9, 403–403.9	401–401.9, 403–403.9	0.37
3	Cerebrovascular disease	Same as ICD-9	430–438.9	8.69
4	Atherosclerosis	Same as ICD-9	440–440.9	1.38
5	Other cardiovascular diseases	Same as ICD-9	441–448.9	1.17
Other diseases				
6	Infectious and parasitic diseases	Same as ICD-9	001–139.9	1.13
7	Benign neoplasms	Same as ICD-9	210–239.9	0.29
Endocrine, nutritional and metabolic diseases, and immunity disorders				
8	Diabetes	Same as ICD-9	250–250.9	1.81
9	Other endocrine, nutritional and metabolic diseases, and immunity disorders	Same as ICD-9	240–249.0, 260–279.9	0.57
10	Diseases of blood and blood forming organs	Same as ICD-9	280–289.9	0.32
11	Mental disorders	Same as ICD-9	290–319	0.71
Diseases of the nervous system and sense organs				
12	Meningitis	Same as ICD-9	320–322.9	0.08
13	Parkinson's disease (paralysis agitans)	342	332–332.1	0.21
14	Other diseases of nervous system and sense organs	320–341.9, 343–389.9	323–331.9, 332.2–389.9	0.89
Diseases of the respiratory system				
15	Acute bronchitis and bronchiolitis	Same as ICD-9	466–466.9	0.03
16	Pneumonia and influenza	470–474.9, 480–486.9	480–487.9	2.97
17	COPD and allied conditions	490–493.9	490–496.9	2.52
18	Other respiratory diseases	460–469.9, 475–479.9, 487–489.9, 494–519.9	460–465.9, 467–479.9, 488–489.9, 497–519.9	1.25
Diseases of the digestive system				
19	Ulcer of stomach and duodenum	Same as ICD-9	531–533.9	0.33
20	Appendicitis	Same as ICD-9	540–543.9	0.03
21	Hernia	Same as ICD-9	550–553.9, 560–560.9	0.29
22	Chronic liver diseases	Same as ICD-9	571–571.9	1.48
23	Other digestive diseases	Same as ICD-9	520–530.9, 534–539.9, 544–549.9, 554–559.9, 561–570.9, 572–579.9	1.54
Diseases of the genitourinary system				
24	Nephritis and kidney infections	Same as ICD-9	580–590.9	0.91
25	Other diseases of the genitourinary system	Same as ICD-9	591–629.9	0.64
26	Residual: Complications of pregnancy, childbirth, and the puerperium; diseases of veins and lymphatics, and other diseases of circulatory system; diseases of the skin and subcutaneous tissue; diseases of the musculoskeletal system and connective tissue; congenital anomalies; certain conditions originating in the perinatal period; and ill-defined conditions	Same as ICD-9	450–459.9, 630–799.9	4.28

Notes: This table lists the ICD-8 and ICD-9 codes used to define the causes of death shown in Figure A.8. Death certificates used ICD-8 codes during the years 1968–1978 and ICD-9 codes during the years 1979–1998. ICD-8 codes are shown only when they differ from the corresponding ICD-9 codes. Prevalence reports the number of deaths from each cause during 1972–1988 as a percentage of total deaths.

Table A.16: Baseline parameter values for the dynamic production model of health

	(1)	(2)
Parameter	Annual data	Daily data
I	0.74773	0.0020521
α	1.53762	1.537619
$\ln \delta$	-5.83878	-11.74124
μ_H	10.39737	11.43803
σ_e	2.25247	0.1178985
N	1,000,000	100,000
SSE	57.80479	20880.82

Notes: This table reports baseline parameter values for the dynamic production model of health given by Equation (3). Column (1) presents values calibrated to annual survival data from a 1972 period life table, while Column (2) reports corresponding values based on daily data. The parameters $\underline{H}$ and σ_H (not shown) are normalized to 0 and 1, respectively. N denotes the number of individuals in the simulation, and SSE is the sum of squared errors. The model fit corresponding to Column (2) is shown in Figure A.10.

Table A.17: IV estimates for the effect of acute SO₂ exposure on all-cause and cancer-related 1-day mortality, ages 65 and over

	(1)	(2)
Age group	All causes	Cancer-related causes
65–69	0.28** (0.038)	0.13** (0.021)
70–74	0.20** (0.060)	0.11** (0.026)
75–79	0.44** (0.078)	0.14** (0.036)
80–84	0.79** (0.12)	0.13* (0.055)
85+	1.9** (0.27)	0.17** (0.061)

Notes: These estimates are used to calibrate the effect of air pollution exposure on mortality in the dynamic production model of health given by Equation (3). The dependent variable is number of deaths per million people on the day of exposure. Each estimate comes from a separate regression. All regressions include county-by-month and month-by-year fixed effects, along with flexible controls for maximum temperature, precipitation, humidity, and wind speed; and two leads and two lags of the instruments. All regressions are weighted by county population. Standard errors, clustered by county, are reported in parentheses. A */** indicates significance at the 5%/1% level.

Table A.18: Projected effect of permanent change in SO₂ on survival gains (years)

	(1)	(2)	(3)	(4)
	IV extrapolation	Model 1 (baseline)	Model 2 (age bins)	Model 3 (linear fit)
1-ppb decrease	0.12 [0.06, 0.25]	1.18 [0.42, 2.01]	1.25 [0.76, 1.97]	0.80 [0.04, 1.63]
2-ppb decrease	0.24 [0.12, 0.50]	2.37 [0.72, 4.22]	2.29 [1.58, 3.05]	1.61 [−0.04, 3.45]
3-ppb decrease	0.37 [0.18, 0.76]	3.53 [1.00, 6.44]	3.43 [2.42, 4.79]	2.42 [−0.09, 5.33]
1-ppb increase	−0.12 [−0.24, −0.06]	−1.10 [−1.97, −0.40]	−1.05 [−1.72, −0.71]	−0.72 [−1.60, −0.03]
2-ppb increase	−0.23 [−0.48, −0.12]	−2.24 [−3.81, −0.72]	−2.23 [−2.97, −1.34]	−1.47 [−3.06, 0.05]
3-ppb increase	−0.35 [−0.72, −0.17]	−3.17 [−5.05, −0.93]	−3.26 [−3.96, −2.43]	−2.11 [−4.01, 0.15]

Notes: Each value in this table reports the projected change in life expectancy (in years) resulting from a permanent change in SO₂ exposure of up to 3 part per billion (ppb) for the cohort of US individuals born in 1972. In the absence of any change in exposure, predicted life expectancy is 71.32 years. 90% bootstrap confidence intervals, based on the 5th to 95th percentiles of the bootstrapped distribution, are reported in brackets. Values in Column (1) are calculated by extrapolating our age-specific 28-day IV estimates to the whole life-cycle. Values in Column (2) come from the dynamic production model of health described by Equation (3), under the assumption that the effect of pollution exposure on model parameters is constant across ages. Values in Column (3) come from a model that applies separate, piecewise-constant parameter shifts for each age bin. Values in Column (4) come from a model that assumes a linear age trend. Figure 7 shows how the survival gains reported in the first row are distributed across the life cycle.