Hazed and Confused: The Effect of Air Pollution on Dementia

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ONLINE APPENDIX

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A. Sample Construction and Summary Statistics

We start with all traditional Medicare enrollees who were 65 or older on January 1, 2004 (6.6 million people). Then we make four sample cuts for our main analysis. First, we drop 2.7 million individuals for whom we could not precisely measure air pollution exposure because they lived in "unclassifiable" counties that lacked $PM_{2.5}$ monitors at the time regulation began or because we were unable to observe their residential location (e.g., because their mailing address was a post office box). This data cut is standard in air pollution studies due to the increased scope for measurement error. While our estimation approach addresses measurement error in general, the error could be greater in unmonitored counties and thus undermine our identification because such counties were treated as de facto in attainment. There are some moderate differences between individuals living in monitored counties and individuals living in unmonitored counties, as shown in Table A1. In particular, we note that those living in unmonitored counties were slightly less likely to receive a new diagnosis of dementia over the decade and slightly more likely to die. Next, we restrict the sample to individuals enrolled in traditional Medicare (TM) in 2004 by dropping 0.8 million who enrolled in Medicare Advantage (MA) that year. This is because CMS lacks data on dementia diagnoses of MA enrollees. However, for some analyses, we expand the sample to include MA enrollees and evaluate the use of ADRD medications as the outcome of interest. These results are described in Appendix J.2.

Our third exclusion is to drop 0.3 million individuals who had already been diagnosed with dementia in 2004 because the disease is currently irreversible, leaving no scope for change.[1] Finally, we drop 0.3 million individuals whose CMS records are missing claims in 2004 or who lived in Census block groups that were missing information on neighborhood demographics, or that we could not assign to a single Census block group in 2004 because they moved during that year. These sample cuts are unlikely to compromise external validity. Table A1 shows that the excluded groups are similar to our main estimation sample in terms of average demographics, longevity, and, when observable, medical conditions, health expenditures, pollution exposure, and Census block-group demographics.

The resulting sample consists of 2,439,904 individuals in 2004. We use this sample to estimate the survival functions in Equations (3) and (8). Figure A1 illustrates how between 2004 and 2013,

[1] In Table II of the main text, we report the results of a validation test using a sample that includes those with dementia in 2004.

some of these individuals move outside of the continental US or to unknown locations, move out of TM into MA and perhaps back again, or die. Our primary sample for estimating Equation (2) and Equation (4) is limited to people who survive through the end of 2013. This sample is comprised of 1,179,094 individuals who were enrolled in traditional Medicare in 2013 and survive through the end of that year (1,112,159 individuals who were continuously enrolled in TM from 2004 to 2013 plus 67,244 who moved from TM to MA and then back to TM, less 309 individuals who are dropped during estimation because there is no variation in dementia outcomes among people in their CBSAs).

For the year-specific version of the model discussed in Section IV.D and summarized in Figure V we follow an analogous approach in which the primary sample for estimating Equation (6) and Equation (9) is limited to people who had not been previously diagnosed with dementia at the beginning of year *t* and who are alive through the end of year *t*. Table I5 reports the year-specific sample sizes and dementia diagnosis rates.

FIGURE A1: SAMPLE SIZES AND TRANSITIONS FROM 2004-2013

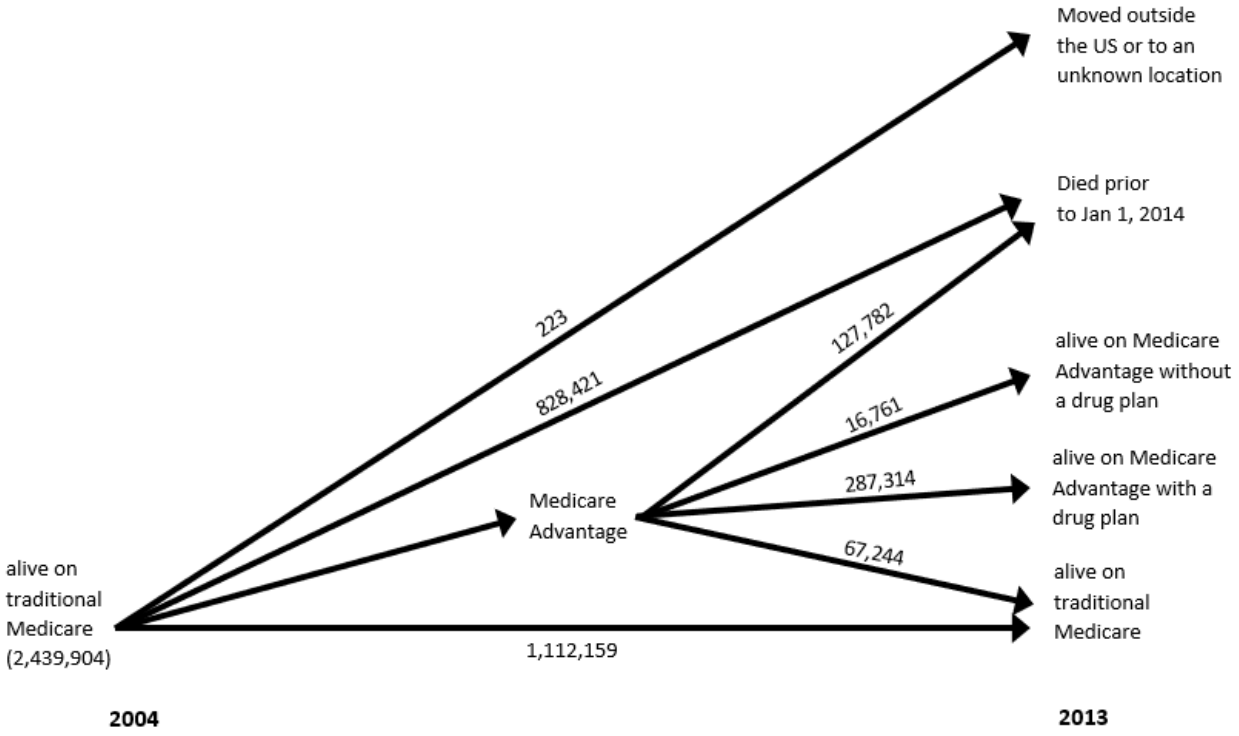


TABLE A1: SUMMARY STATISTICS FOR MEDICARE BENEFICIARY SAMPLES

	(1)	(2)	(3)	(4)	(5)	(6)
	ESTIMATION SAMPLES		EXCLUDED	EXCLUDED	EXCLUDED	EXCLUDED
	survived through 2013	traditional Medicare enrollees in 2004	lived in county without pollution monitors or at unknown location	enrolled in Medicare Advantage in 2004	had dementia in 2004	missing data or moved in 2004
# people	1,179,094	2,439,904	2,683,047	783,911	335,436	328,735
Individual demographics						
mean age at sample entry	69.3	71.1	71.2	71.4	77.3	68.9
mean age in 2013	82.5	84.5	84.7	85.0	91.2	81.7
male (%)	38	41	43	40	32	50
white (%)	84	83	87	75	80	75
black (%)	8	9	6	10	11	11
asian (%)	3	3	1	4	2	4
hispanic (%)	5	5	6	10	6	9
alive at end of 2013 (%)	100	61	56	58	17	69
dementia at end of 2013 (%)	20	25	30	11	100	18
ever moved (%)	32	32	38	36	52	58
ever moved county (%)	15	15	16	17	25	34
ever moved state (%)	8	8	8	8	12	21
2013 gross Medicare expenditures ($)	4,685	6,701	7,131		16,246	
Medical diagnoses as of 2004						
dementia (%)	0	0	10		100	
stroke (%)	7	10	11		34	
congestive heart failure (%)	12	20	21		45	
diabetes (%)	21	25	24		34	
ischemic heart disease (%)	35	42	38		61	
hypertension (%)	66	70	64		84	
Neighborhood characteristics						
$PM_{2.5}$ (hourly $\mu g/m^3$) 2001-2003	13.23	13.28		13.57	13.40	
Nonattainment county (%)	39.91	39.40		42.43	42.37	
household income (median)	65,912	62,026	52,722	60,330	59,800	
income per capita	33,755	31,817	26,808	29,934	31,095	
year built (median)	1970	1969	1973	1967	1968	
house value (median)	267,992	246,628	170,354	278,066	244,766	
house value (average)	138,293	124,492	88,424	131,762	119,107	
gross rent (median)	2,845	2,544	1,722	2,276	2,361	
population over 65 (%)	18	18	19	18	19	
population white not hispanic (%)	69	67	83	58	64	
population black (%)	12	13	7	12	15	
population hispanic (%)	13	13	6	20	14	
education: 9th to 12th (%)	7	8	9	8	8	
education: high school grad (%)	26	27	34	27	27	
education: some college (%)	21	21	21	21	21	
education: associate degree (%)	8	8	8	8	7	
education: bachelor's degree (%)	20	19	15	18	19	
education: graduate degree (%)	13	12	9	11	12	
owner occupied (%)	64	62	64	60	58	
renter occupied (%)	26	28	23	31	32	

Note: Columns (1) and (2) report variable means for our main estimation samples. Column (1) is a balanced panel of individuals who were in traditional Medicare (TM) in 2004 and survived through the end of 2013, at which point they were still enrolled in TM. Column (2) adds individuals who were in TM in 2004 but died or switched to Medicare Advantage (MA) before 2013. All but one of differences in means between the samples in columns (1) and (2) are statistically significant with p-values below 0.01. The exception is "education: associate degree (%)" (p=0.5). Column (3) describes individuals who were in TM in 2004 but not used in estimation because they lived in known locations in counties that were designated by EPA as "unclassifiable" for regulatory purposes due to a lack of pollution monitors (1,523,641) or because their residential location could not be determined (1,159,406) in which case they are excluded when calculating mean values of neighborhood characteristics. Column (4) describes individuals not used in estimation because they were enrolled in MA in 2004, leaving us unable to observe their dementia diagnoses and medical expenditures. Column (5) describes individuals who were in TM in 2004 but not used in estimation (aside from placebo regressions) because they had been diagnosed with dementia by 2004. Column (6) describes individuals who were in TM in 2004 but not used in estimation because they were missing data on medical expenditures, their residential address could not be matched to a Census block group, or they changed addresses in 2004 complicating assignment to a block group and attainment/nonattainment area.

B. Locations of Air Pollution Monitors and Nonattainment counties

Figure B1 shows the attainment status for US counties that had $PM_{2.5}$ monitors in place throughout the 2001-2003 evaluation period. There were 132 nonattainment counties located in 21 states and 528 attainment counties located in 50 states.

FIGURE B1: INITIAL COUNTY (NON)ATTAINMENT DESIGNATIONS FOR $PM_{2.5}$

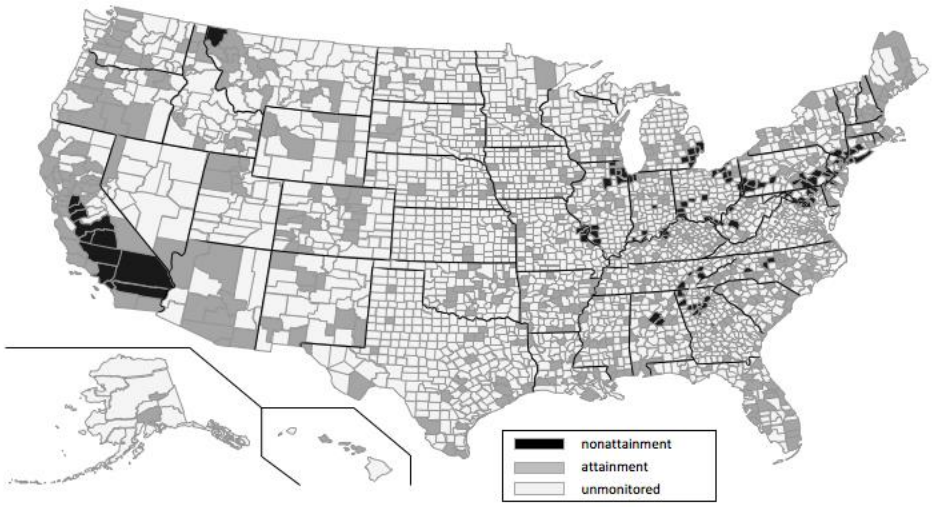


Figure B2 shows the locations of $PM_{2.5}$ monitors. This figure was generated using the Environmental Protection Agency's AirData Air Quality Monitor app: https://www.epa.gov/outdoor-air-quality-data/interactive-map-air-quality-monitors.

FIGURE B2: LOCATIONS OF EPA MONITORING STATIONS FOR $PM_{2.5}$

The US had 1,772 federal regulatory-grade monitors reporting $PM_{2.5}$ at some point during our study period, and 485 reporting data every year from 2001 through 2013. These data represent the best available information on ambient air quality in the United States. They are used by the EPA and other researchers as the benchmark for calibrating and evaluating the accuracy of novel approaches to predicting air quality, such as inexpensive consumer-grade sensors (https://www.epa.gov/air-sensor-toolbox/evaluation-emerging-air-sensor-performance) and satellite images of aerosol optical depth to predict $PM_{2.5}$ concentrations (e.g., Fowlie, Rubin and Walker (2019)). The EPA provides information about air quality monitoring technology and its accuracy here: https://www.epa.gov/amtic/air-monitoring-methods-criteria-pollutants.

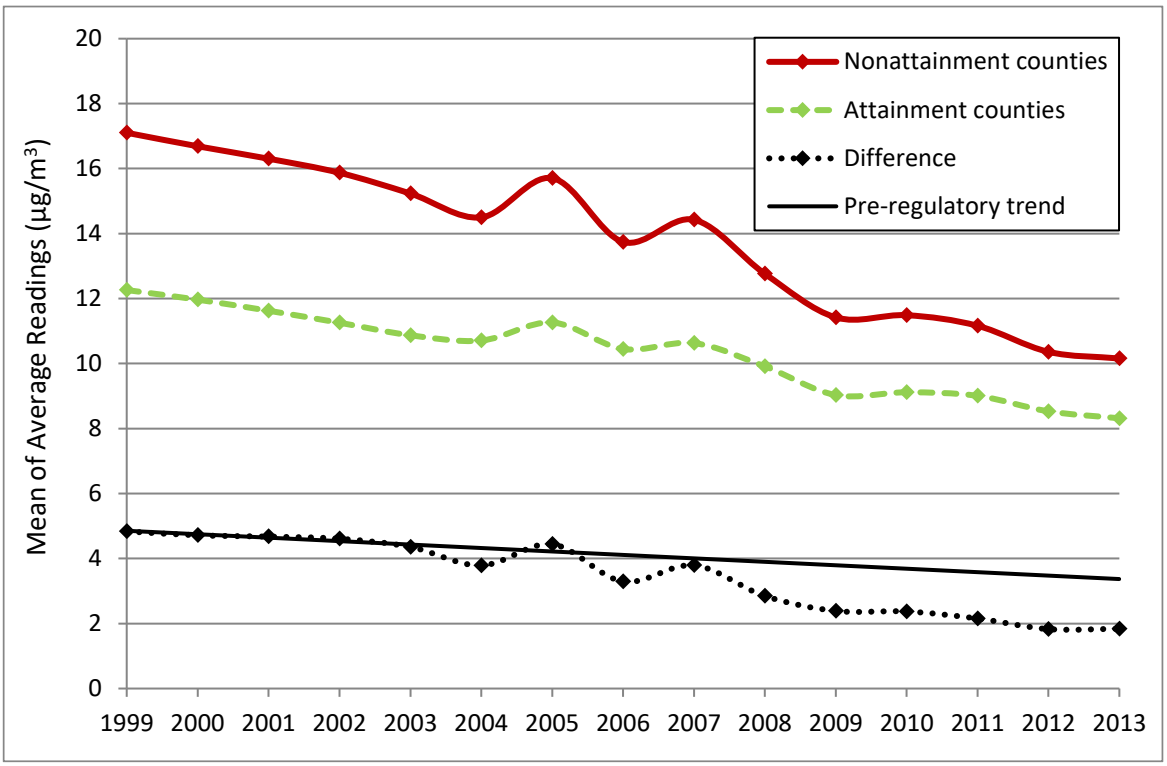
C. Annual $PM_{2.5}$ Concentrations by County Attainment Status

Figure C1 mirrors the analysis that Chay and Greenstone (2005) used to motivate their use of the 1975 nonattainment designations for total suspended particulates (TSP) as instrumental variables to isolate exogenous between-county variation in TSP changes (see Figure 2 in that paper). Here, the nonattainment and attainment lines show annual average concentrations in counties that were designated as nonattainment and attainment for $PM_{2.5}$, respectively.[2] The difference line shows that the difference between the trend lines for attainment and nonattainment counties was fairly stable from 1999 through 2003 with between 4.4 and 4.8 higher $\mu g/m^3$ in nonattainment counties.

Starting in 2004, $PM_{2.5}$ concentrations declined at a noticeably faster rate in nonattainment counties so that by 2013 the gap was only 1.9 $\mu g/m^3$. This differential is 1.5 $\mu g/m^3$ smaller than the gap that would be predicted by projecting the pre-regulatory trend from 1999-2003 forward to 2013 (3.4 $\mu g/m^3$). The cumulative difference between these two trends reveals that the average concentrations from 2004 to 2013 in nonattainment counties was 0.97 $\mu g/m^3$ lower than projected from the pre-regulatory trend.

[2] The figure is based on a balanced panel of 485 $PM_{2.5}$ monitors in operation continuously from 2001-2013. The figure looks virtually identical if we reconstruct it using an unbalanced panel of all monitors ever in operation from 1999-2013.

FIGURE C1: ANNUAL $PM_{2.5}$ CONCENTRATIONS BY COUNTY ATTAINMENT STATUS

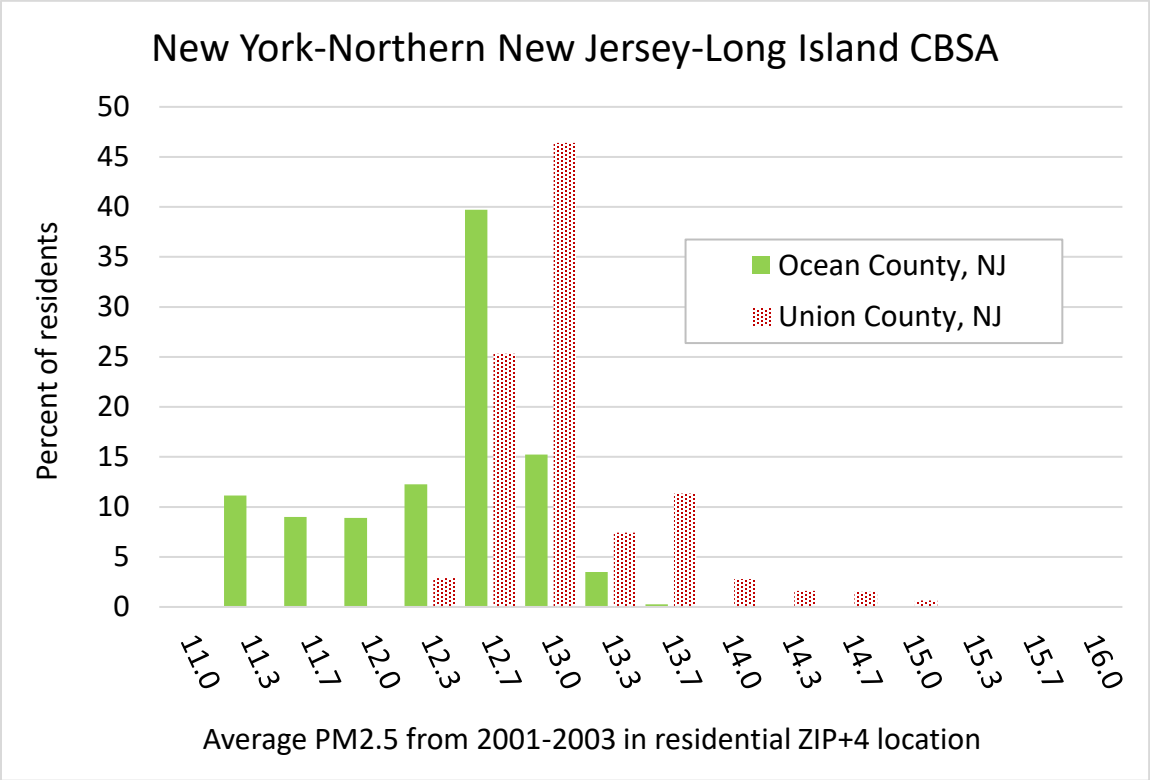


Note: The figure reports annual average concentrations of $PM_{2.5}$. Measurements are taken from air quality monitors in counties designated in 2005 as attainment or nonattainment with the federal standard based on monitor readings from 2001-2003. The nonattainment line is a simple average over monitors in nonattainment counties that were in operation from 2001-2013. The attainment county line is defined similarly. The difference line shows the difference between the nonattainment and attainment lines. The pre-regulatory trend line is a projection of the difference in the pre-regulatory period, as state and local regulators were notified of the impending nonattainment designations in 2004. In 2010 the Census Bureau recorded 41% of the US population age 65 and over as living in attainment counties and 27% as living in nonattainment counties.

D. Within-CBSA Variation in Nonattainment Status by Baseline $PM_{2.5}$

Figure D1 provides an example of the within-county and between-county variation in attainment status, conditional on baseline residential $PM_{2.5}$ concentrations within a CBSA. The horizontal axis describes baseline $PM_{2.5}$ concentrations (in 0.33 microgram per cubic meter bins) for two adjacent counties within the New York-Northern New Jersey-Long Island CBSA. The vertical axis reports the fraction of residents in each bin. The considerable overlap in baseline concentrations between an attainment county (Ocean) and a nonattainment county (Union) may be seen.

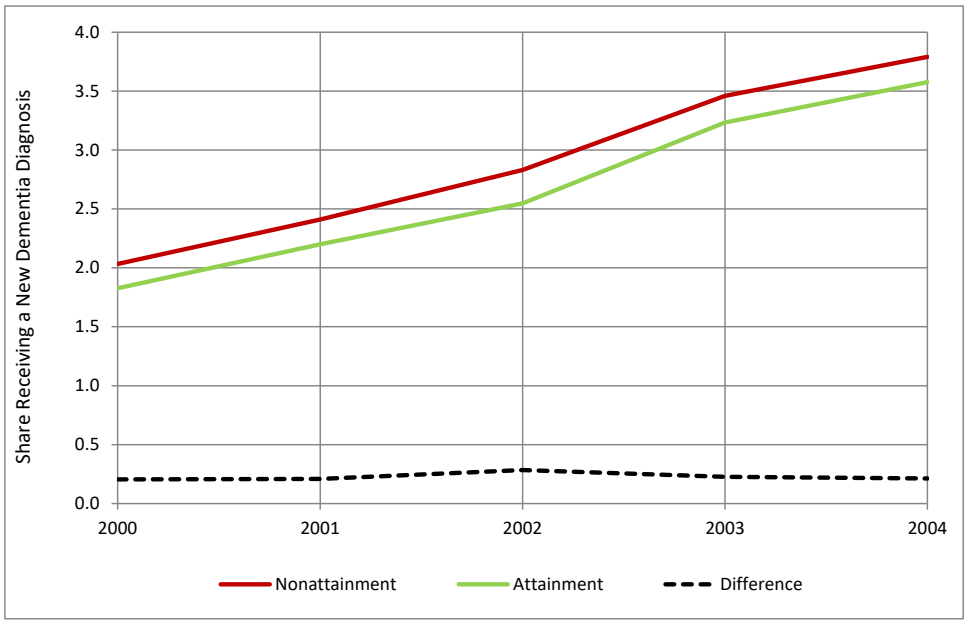
FIGURE D1: WITHIN-CBSA VARIATION IN NONATTAINMENT STATUS BY BASELINE $PM_{2.5}$ LEVELS



E. Trends in New Dementia Diagnoses Prior to 2005

Figure E1 shows that the difference in annual dementia diagnosis rates between attainment and nonattainment counties was stable between 1999 and 2004. While parallel pre-trends is neither necessary nor sufficient for drawing causal inference from our research design, the absence of pre-regulatory differences may assuage concerns that the estimated differences during the subsequent decade are due to other factors such as differential rates of change in doctors' diagnostic and pre-scribing decisions.

FIGURE E1: NEW DEMENTIA DIAGNOSIS PRIOR TO 2005 BY COUNTY ATTAINMENT STATUS



F. $PM_{2.5}$ Exposure and Migration

We estimate the effects of $PM_{2.5}$ exposure from 2004 through year *t* on the probability of moving to a new address in year *t*. The outcome of interest, $M_{i,t}$, is an indicator for whether person *i*'s residential address on file at CMS has a different ZIP+4 code in year *t+1* relative to year *t*. This effectively measures whether the person moved to a new residential address because ZIP+4 codes are close to street address in terms of spatial precision.

Equations (F1) and (F2) show how we specify the second-stage and first-stage regressions.

$$\text{(F1)} \quad M^*_{i,t} = h\left(durPM_{i,t}; \pi_{i,t}\right) + \theta_{X,t}X_i + \theta_{H,t}H_i + \theta_{W,t}W_i + \theta_{C,t}C_i + f(basePM_i; \theta_{basePM,t}) + \eta_{i,t},$$

$$\text{where} \quad M_{i,t} = 1[M^*_{i,t} > 0].$$

$$\text{(F2)} \quad durPM_{i,t} = \kappa_{Z,t}Z_i + \kappa_{X,t}X_i + \kappa_{H,t}H_i + \kappa_{W,t}W_i + \kappa_{C,t}C_{i,t} + f\left(basePM_i; \kappa_{basePM,t}\right) + u_{i,t}.$$

We instrument for $durPM_{i,t}$ using a vector of instruments, Z_i, that is comprised of an indicator for residing in a nonattainment county in 2004 and interactions between this indicator and a fourth-order polynomial of $basePM_i$. The coefficient of interest, π, measures the effect of $PM_{2.5}$ exposure from 2004 through year *t* on the probability of moving in year *t*.

FIGURE F1: ESTIMATED EFFECTS OF $PM_{2.5}$ EXPOSURE ON THE PROBABILITY OF MOVING

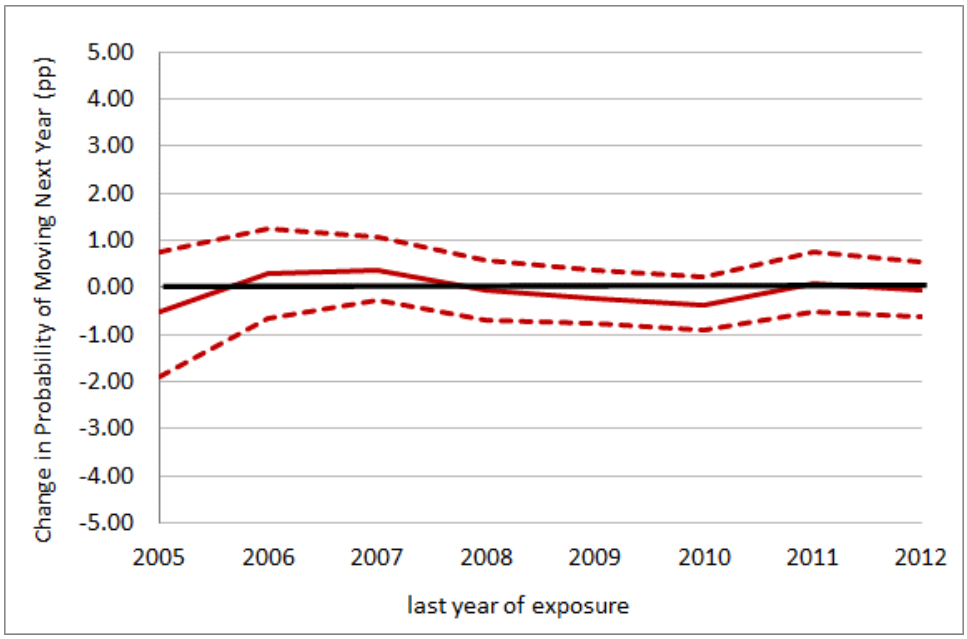


Figure F1 shows our 2SCML estimates for π and 95% confidence bands. The point estimates range from -0.52 pp to 0.38 pp and the confidence bands include zero in all years. For reference, the share of people moving each year ranges from a low of 3.97 pp in 2007 to a high of 5.23 pp in 2005.

FIGURE F2: ANNUAL AVERAGE CHANGES IN $PM_{2.5}$ BY AGE, MIGRATORY STATUS, AND DEMENTIA

Next, we analyze the changes in $PM_{2.5}$ experienced by movers with dementia compared to non-movers of the same age (who may or may not have dementia). Specifically, we regress the year-to-year change in individuals' $PM_{2.5}$ exposures on indicators for their integer age and an interaction term comprised of indicators for (i) integer age, (ii) whether the individual has dementia ($y_{i,t}$), and (iii) whether the year-to-year change in $PM_{2.5}$ exposure straddled a move ($M_{i,t-1}$).

$$\Delta PM2.5 = PM2.5_{i,t} - PM2.5_{i,t-1} = \mu_0 + \mu_1 age_{i,t} + \mu_2 age_{i,t} * M_{i,t-1} * y_{i,t} + \vartheta_{i,t}.$$

All individuals age 100 and over are grouped into a single age bin at 100. Because the model includes 9 observations per individual and the errors may exhibit autocorrelation, the confidence bands are constructed from robust standard errors clustered at the individual level.

Figure F2 plots our estimates for μ_2 and 95% confidence bands. The solid line shows that younger movers with dementia tend to experience relatively larger year-to-year reductions in their $PM_{2.5}$ exposures as a result of moving compared to non-movers of the same age (who may or may not have dementia). The differential diminishes with age and the confidence bands include zero for most ages beyond 80.

G. A Model of a Dementia-Diagnosis Production Function

We illustrate how our decadal model for new dementia diagnoses can be linked to a more primitive "production function" for dementia diagnoses. We start by writing the latent propensity to be diagnosed with dementia by the end of year t as a function of the lifetime history of $PM_{2.5}$ exposure (from an initial year *B* to year *t*), a vector of time-varying determinants, $\zeta_{i,t}$ (which includes both observable factors and unobservable factors), and a vector of time-invariant determinants, ξ_i (which includes both observable factors and unobservable factors). While some factors, such as genetics, are time-invariant, they may have time-varying impacts on the latent propensity to be diagnosed with dementia, and would, in that case, be included in $\zeta_{i,t}$.

$$\text{(G1)}\ y_{i,t}^{*} = k\left(PM_{i,t-9}, PM_{i,t-8}, \dots, PM_{i,t-1}, PM_{i,t}\right) + g\left(PM_{i,B}, PM_{i,B+1}, \dots, PM_{i,t-11}, PM_{i,t-10}\right) + \zeta_{i,t} + \xi_i$$

In Equation (G1), we specify the impact of the most recent decade of $PM_{2.5}$ exposure via the nonparametric function $k(\cdot)$. We allow all previous exposure to enter via the nonparametric function $g(\cdot)$. The $k(\cdot)$ and $g(\cdot)$ functions place no restrictions on the importance of more recent exposures relative to more distant exposures, or on how they differ from individual to individual. The reason we distinguish between the most recent decade and earlier exposures in (G1) is because the most recent decade is the longest period over which we can observe exogenous variation in $PM_{2.5}$ exposures given the existing data and the institutional features of our application (summarized in Section I of the main text). Differencing yields:

$$\text{(G2)}\ \Delta y_{i,2013}^{*} \equiv\ y_{i,2013}^{*} - y_{i,2004}^{*}$$

$$= k\left(PM_{i,2004}, \dots, PM_{i,2013}\right) - k\left(PM_{i,1995}, \dots, PM_{i,2004}\right) + \Delta g_{i,2013}\left(PM_{i,B}, \dots, PM_{i,2003}\right) +\ \Delta\zeta_{i,2013},$$

where $\Delta g_{i,2013}\left(PM_{i,B}, \dots, PM_{i,2003}\right) = g\left(PM_{i,B}, \dots, PM_{i,2003}\right) - g\left(PM_{i,B}, \dots, PM_{i,1994}\right)$

and $\Delta\zeta_{i,2013} = \zeta_{i,2013} - \zeta_{i,2004}$. Note that ξ_i has dropped out.

To illustrate how this specification relates to our decadal model of new dementia diagnoses in the text, consider the following rewrite of Equation (G2),

$$\text{(G3)}\ \Delta y_{i,2013}^{*} = h\left(durPM_{i,2013}; \alpha_i\right) + \eta_i,$$

where $\eta_i =\ k\left(PM_{i,2004}, \dots, PM_{i,2013}\right) - h\left(durPM_{i,2013}; \alpha_i\right) - k\left(PM_{i,1995}, \dots, PM_{i,2004}\right) +$
$\Delta g_{i,2013}\left(PM_{i,B}, \dots, PM_{i,2003}\right) +\ \Delta\zeta_{i,2013}.$

In the text we follow Angrist and Pischke (2009) in decomposing η_i into a linear function of observable controls, $X_i, H_i, W_i, C_i,\ basePM_i$, and an error term, e_i:

(G4) $\eta_i = \beta_x X_i + \beta_H H_i + \beta_W W_i + \beta_C C_{i,2013} + f(basePM_i; \beta_{basePM}) + e_i.$

The controls, $X_i, H_i, W_i, C_i,\ basePM_i$, are natural choices to include in Equation (G4) as they contain risk factors (i.e., demographic, health, and location characteristics) that are likely to be associated with new dementia diagnoses as discussed in Section II. By directly conditioning on these controls in Equation (G4), we effectively remove them from η_i. However, we do not assume that $durPM_{i,2013}$ is exogenous with respect to the residual unobserved factors, e_i, and we address the endogeneity of $durPM_{i,2013}$ using the instrument, Z_i.

Combining equations (G2), (G3), and (G4) yields the following equation, which is identical to Equation (1) in the text:

(G5) $\Delta y^*_{i,2013} = h\big(durPM_{i,2013}; \alpha_i\big) + \beta_X X_i + \beta_H H_i + \beta_W W_i + \beta_C C_{i,2013} + f(basePM_i; \beta_{basePM}) + e_i$

The parameter of interest in Equation (G5) is α, which describes the causal effect of the most recent decade's $PM_{2.5}$ exposure on a new dementia diagnosis, holding all else constant. A necessary condition to identify α is that Z_i is conditionally independent of η_i, where Z_i are the instruments for $PM_{2.5}$ described in Section IV.A. We assume that the model controls for potential confounding factors that determine a new dementia diagnosis, such that e_i is independent of the non-attainment instruments, Z_i. Thus, we treat $durPM_{i,2013}$ as endogenous with respect to e_i and we assume that Z_i and the controls $X_i, H_i, W_i, C_{i,2013}$, and $f(basePM_i)$, are exogenous with respect to e_i.[3] Analogs to our conditional independence assumption on Z_i and our exogeneity assumption on the controls are ubiquitous in the economic literature linking pollution to health outcomes (e.g., Schlenker and Walker 2016, Isen, Rossin-Slater, and Walker 2017, and Deryugina et al. 2019).

This instrumental-variables based approach will address measurement error in $PM_{2.5}$. This measurement error may arise from unobserved variation in indoor air, daily mobility, and activities that create differences between observable measures of ambient pollution and what individuals

[3] As discussed in the main text, exogeneity of the controls is established by a mean-independence assumption that is equivalent to the assumption that the functional form specified in the population regression Equation (G4) is sufficiently flexible to capture the relationship between the controls and η_i (Angrist and Pischke, 2009). Then, exogeneity of Z is established by making the additional assumption that Z_i is independent of η_i conditional on the controls. Three features of our research design support the credibility of the functional-form assumption in (G4). First, as discussed in Section II, our controls are extensive. Second, our model is saturated within some control vectors (e.g., integer-age-by-gender dummies and the full-factorial of baseline health conditions) and flexible in other control vectors (e.g., fourth order polynomial functions of medical spending and baseline pollution). Third, the estimated AMEs are relatively insensitive to adding additional interactions and additional flexibility in unsaturated control vectors. See, for example, the discussions in Sections IV.C, IV.D, VI.B, and Appendix J.

actually breathe or from our interpolation between geography-based measures of ambient pollution (i.e., monitors) required to develop individual-level measures of exposure.

To address any threats to identification from mis-specification, we rely on a series of robustness checks. While our preferred simple average of $PM_{2.5}$ exposure is consistent with the medical literature's accumulation hypothesis, there could be relevant forms of depreciation that cause more distant $PM_{2.5}$ exposures to have smaller or larger effects than more recent exposures, for example, unknown biological mechanisms, temporal variation in the chemical composition of $PM_{2.5}$, and zero marginal effects of further exposures after an individual is diagnosed with dementia. Several sensitivity checks indicate robustness of our results. First, Tables J1 and J4 show that our results persist across several alternative specifications, i.e., specifications where we modify the spatial scale of the nonattainment instruments, increase size of the geographic area where exposure is assumed to occur, reduce minimum allowable distance from the nearest monitor, increase the degree of flexibility in our controls for baseline pollution exposure during 2001-2003, and add controls for other measures of air pollution. Second, the results of our featured specification, the year-by-year model underlying the results shown in Column (6), Table I, shows little impact on the AME of interest when we reduce the scope for specification error due to temporal aggregation of both the treatment and outcome measures (relative to the decadal model). As a final sensitivity check, we repeat the estimation of the decadal model using a depreciation parameter, δ, where we re-define exposure as $\widetilde{durPM_{\iota,2013}} = (\sum_{s=2004}^{2013} PM_{i,s}\,(1-\delta)^{2013-s})/(\sum_{s=2004}^{2013}(1-\delta)^{2013-s})$. We estimate the model for both $\delta = 0.05$ and $\delta = -0.05$ and note that our corresponding specification that has no depreciation parameter is equivalent to setting $\delta = 0$. The resulting estimates for α are not directly comparable with estimates from our corresponding specification (i.e., setting $\delta = 0$) because $\widetilde{durPM_{\iota,2013}}$ and $durPM_{i,2013}$ weight annual exposures differently; however, the AMEs are again similar (2.12 pp for $\delta = 0.05$ and 2.63 pp for $\delta = -0.05$ versus 2.38 for $\delta = 0$).

Finally, we consider the threat to identification posed by omitted variables. After instrumenting for $PM_{2.5}$, the key identifying assumption required for consistent estimation is that individuals living in 2004 in counties that differ in their likelihood of being designated nonattainment do not systematically differ in their likelihood of a new dementia diagnosis post-attainment due to omitted variables after conditioning on an extensive set of covariates. These covariates are given by (1) the CBSAs where individuals live in 2013; (2) their observed individual demographics; (3) their observed measures of individual health in 2004; (4) the observed measures of socioeconomic status

among the individuals living in their neighborhood in 2004; and (5) the baseline $PM_{2.5}$ concentration in their 2004 location (average concentrations from 2001-2003). We view this last covariate as essential for the plausibility of the identifying assumption, because the EPA solely relied on average concentrations from 2001-2003 to make nonattainment designations. As shown in Figure II.i, these designations led to lower levels of $PM_{2.5}$ over 2004-2013 for people living in nonattainment counties relative to people in attainment counties, conditional on the measure of baseline $PM_{2.5}$ and all other covariates.

Several robustness checks and placebo tests support our assumption that $PM_{2.5}$ exposures prior to 2001 and other omitted variables are uncorrelated with our instruments. First, there is no meaningful change in our estimate of α when we add controls for $PM_{2.5}$ concentrations in 1999 and 2000 (2.35 pp versus 2.38 pp).[4] Second, Table II also shows that our decadal model is unable to reject the hypothesis of no relationship between dementia rates in 2004 and $PM_{2.5}$ exposure over the subsequent decade. This suggests that, conditional on the covariates, people at a lower unobserved risk for dementia were not more or less likely to live in areas that were subsequently designated as nonattainment. Finally, Table J5 shows that when we estimate our decadal model for several other placebo outcomes the estimated average marginal effects are relatively small and statistically indistinguishable from zero at the 10% level.

[4] Column (2) of Table II in the main text shows that results from our featured year-by-year model are also robust to this addition.

H. Using Partial Identification to Address Selection on Mortality

In the case of sample selection based on survival, the total effect of $PM_{2.5}$ on the dementia rate would combine both the causal effect of $PM_{2.5}$ on dementia (our object of interest) plus a compositional effect. In other words, if individuals were exposed to a change in $PM_{2.5}$, the dementia rate would change for two reasons. First, the change in $PM_{2.5}$ would have a causal effect on dementia. Second, the change in $PM_{2.5}$ would have a causal effect on survival and, if the underlying propensity to be diagnosed with dementia for the marginal individuals (i.e., those individuals who are induced to die by the change in $PM_{2.5}$) differs from the propensity for the inframarginal individuals, the estimated effect of $PM_{2.5}$ on dementia would incorporate the effects of this compositional change.[5]

As outlined in Honoré and Lleras-Muney (2006), the prior literature has developed several approaches to addressing the role of selection-driven compositional change. We apply them to our decadal model, taking a bottom-up approach. First, we use a partial-identification approach to estimate bounds without making assumptions about the relationship between the propensity to be diagnosed with dementia and the propensity to survive (e.g., Manski 1990, Horowitz and Manski 2000, Lee 2009). Next, we sharpen the bounds by adding plausible assumptions about the relationship between the propensity to be diagnosed with dementia and the propensity to survive (e.g., Manski and Pepper 2000, Honoré and Lleras-Muney 2006, and Bhattacharya, Shaikh, and Vytlacil 2012). Finally, we return to the decadal specification shown in Column (5) of Table I, which secures point identification by adding an additional set of instruments and additional distributional assumptions (e.g., Heckman 1979).

The bounds approach follows Lee (2009) and is modified for our application. For notational simplicity, we suppress right-hand side variables other than $durPM_{2013}$, including the residuals from our first stage, Equation (2), $\hat{\varepsilon}$.[6] S is the binary variable denoting decadal survival. We consider changes in the expected value of ΔY for a marginal increase in $durPM_{2013}$, denoted h.

We are interested in the causal, marginal effect of $durPM_{2013}$ on ΔY, holding selection on survival constant. We denote this causal effect α_{PM}. For any given $durPM_{2013}$, α_{PM} is defined as the change in the expected value of ΔY among those who would survive under both $durPM_{2013}$ and $durPM_{2013} + h$ exposures, i.e., inframarginal individuals:

[5] See also Blundell, Gosling, Ichimura, and Meghir (2007), which analyzes changes in wage distributions in the presence of compositional changes.
[6] That is, absent selection, we are treating $durPM_{2013}$ as exogenous.

$$\alpha_{PM} = \lim_{h \to 0} \frac{E[\Delta Y | durPM_{2013} + h, S(durPM_{2013} + h) = 1] - E[\Delta Y | durPM_{2013}, S(durPM_{2013} + h) = 1]}{h}.$$

We note that the conditioning statement in the second term in the numerator describes a counter-factual set not directly observed in the data. That is, for any given $durPM_{2013}$, we observe the dementia status of survivors who were exposed to $durPM_{2013}$, but do not observe whether they would have survived had they been exposed to h more units of $durPM_{2013}$.

We denote the total marginal effect of $durPM_{2013}$ on ΔY as A_{PM}.

(H1) $$\mathrm{A}_{PM} = \lim_{h \to 0} \frac{E[\Delta Y | durPM_{2013} + h, S(durPM_{2013} + h) = 1] - E[\Delta Y | durPM_{2013}, S(durPM_{2013}) = 1]}{h}.$$

This term captures the fact that individuals exposed to $durPM_{2013} + h$ will have a different survival rate compared with individuals exposed to only $durPM_{2013}$. In contrast to α_{PM}, we note that the conditioning statements in the conditional expectations in (H1) describe sets directly observed in the data. Denoting the share of marginal individuals as $\rho_{PM,h}$ allows us to rewrite the second term in the numerator of Equation (H1) as:

(H2) $$E[\Delta Y | durPM_{2013}, S(durPM_{2013}) = 1] =$$
$$\rho_{PM,h} E[\Delta Y | durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013} + h) = 0]$$
$$+ (1 - \rho_{PM,h}) E[\Delta Y | durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013} + h) = 1].$$

After gathering like terms, this allows us to write the numerator in Equation (H1) as:

$$E[\Delta Y | durPM_{2013} + h, S(durPM_{2013} + h) = 1]$$
$$- E[\Delta Y | durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013} + h) = 1]$$
$$- \rho_{PM,h} (E[\Delta Y | durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013} + h) = 0]$$
$$- E[\Delta Y | durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013} + h) = 1]).$$

We assume monotonicity, such that S is weakly decreasing in $durPM_{2013}$, implying that $S(durPM_{2013} + h) = 1 \rightarrow S(durPM_{2013}) = 1$, and that:

$$E[\Delta Y | durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013} + h) = 1] = E[\Delta Y | durPM_{2013}, S(durPM_{2013} + h) = 1].$$

This allows us to write:

(H3) $$\mathrm{A}_{PM} = \alpha_{PM} + \lim_{h \to 0} \frac{\rho_{PM,h}}{h} (E[\Delta Y | durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013} + h) = 1] -$$
$$E[\Delta Y | durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013} + h) = 0]).$$

This equation shows that the total difference in the expected value of ΔY under the marginal change in $durPM_{2013}$ is comprised of two terms. The first term reflects the effect of the marginal

increase in $durPM_{2013}$ on the expected value of ΔY for inframarginal individuals. The second term reflects the underlying difference in the expected value of ΔY between inframarginal and marginal individuals, scaled by ρ_{Xh}. In other words, the total marginal effect is comprised of a causal marginal effect of $durPM_{2013}$ on expected ΔY and a compositional effect. As α_{PM} is the object of interest, we rearrange Equation (H3) to get:

$$\text{(H4)} \quad \alpha_{PM} = \mathrm{A}_{PM} + \lim_{h\to 0}\frac{\rho_{PM,h}}{h}\big(E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013}+h) = 0] - E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013}+h) = 1]\big).$$

Given the assumptions maintained in the main text regarding Equations (1) and (2), A_{PM} and $\rho_{PM,h}$ are estimable using the data. However, the remaining two conditional expectations in Equation (H4) rely on conditions not observed in the data. In contrast, the conditioning statement in the conditional expectation, $E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1]$, describes a set observed in the data. If A_{PM}, $\rho_{PM,h}$, and $E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1]$ were known, we could construct lower and upper bounds for the difference in the two unknown conditional expectations in (H4), using the fact that $E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013}+h) = 0]$ is naturally bounded between 0 and 100 in our application. This allows us to construct bounds for α_{PM}. Specifically, for the lower bound, we set $E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013}+h) = 0]$ to 0 and solve for $E[\Delta Y|decPM, S(decPM) = 1, S(decPM+h) = 1]$ using:

$$E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013}+h) = 1]$$
$$= \left(\frac{E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1] - \rho_{PM,h}E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013}+h) = 0]}{1-\rho_{PM,h}}\right)$$

which follows from Equation (H2). We then use these values, along with A_{PM} and $\rho_{PM,h}$, in Equation (H4) to recover the lower bound for α_{PM}. The upper bound is constructed analogously by setting $E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013}+h) = 0]$ to 100.

The construction of these bounds is quite intuitive; while we do not know which specific individuals are marginal and which individuals are inframarginal, we can estimate the share of marginal individuals, $\rho_{PM,h}$, and use this, along with the restriction that probabilities must lie between 0 and 1, to inform the bounds.

Thus, the inputs to calculating bounds are A_{PM}, $\rho_{PM,h}$, and $E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1]$, which are the total marginal effect of $durPM_{2013}$ on ΔY,

the share of marginal individuals, and the mean ΔY among survivors, respectively. Each of these terms is allowed to vary with $durPM_{2013}$ (and the other suppressed right-hand side variables). Therefore, we can calculate individual-specific values of α_{PM} using individual-specific values of A_{PM}, $\rho_{PM,h}$, and $E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1]$. Bounds for the average value of α_{PM}, i.e., our AME of interest, are constructed as the average lower bound and average upper bound of α_{PM}.

Because A_{PM} is the total marginal effect of $durPM_{2013}$ on ΔY, we estimate it using the marginal effects from a specification that uses the attainment-based control function, $\hat{\varepsilon}$, but not the survival-based control function, $\hat{v}$. In particular, we estimate a version of the specification shown in Column (5) of Table 1 where we omit any terms that include $\hat{v}$. The individual-specific marginal effects yield estimates of A_{PM} at different values of $durPM_{2013}$. From this same estimation, which only uses data describing survivors, the individual-specific fitted values of ΔY given $durPM_{2013}$ provide estimates of $E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1]$. We note that estimating A_{PM} and $E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1]$ relies on the functional form and distributional assumptions maintained in Equations (1) and (2), but does not require us to estimate Equation (3) or employ the additional survival instruments, M_i.

Finally, to estimate the share of marginal individuals, $\rho_{PM,h}$, we estimate a specification that replaces ΔY with *S* as the dependent variable in the specification shown in Column (5) of Table I. We continue to use the attainment-based control function, $\hat{\varepsilon}$. The survival control function, $\hat{v}$, is not relevant because we use all the data and do not condition on survival. We then calculate $\rho_{PM,h}$ as the ratio of two terms. The numerator is the individual-specific marginal effect of $durPM_{2013}$ on *S*, and the denominator is the individual-specific fitted value of *S* given $durPM_{2013}$. Analagous to the estimation of A_{PM} and $E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1]$, the estimation of $\rho_{PM,h}$ relies on the functional form and distributional assumptions maintained in Equations (1) and (2) (as applied when Equation (1) replaces ΔY with *S* as the dependent variable) but does not require us to estimate Equation (3) or employ the additional survival instruments, M_i.

We sharpen these bounds by assuming a plausible form of monotone treatment selection (Manski and Pepper 2000) in which individuals who would be induced to die if they were exposed to $durPM_{2013}+h$ versus $durPM_{2013}$ were, on average, no less likely to develop dementia than those whose survival was unaffected by the increase in exposure. In other words, we assume that $E[\Delta Y|durPM_{2013}, S(durPM_{2013}) = 1, S(durPM_{2013} + h) = 0] \geq E[\Delta Y|durPM_{2013}, S(durPM_{2013}) =$

$1, S(durPM_{2013} + h) = 1]$. Intuitively, this assumes that the latent health driving mortality is weakly positively correlated with the latent health driving dementia. Under this assumption, the construction of the upper bound remains the same as before, and the lower bound is set to A_{PM}.

We note that the specification shown in Column (5) of Table I is point identified and that point identification relies on the additional instruments, M_i, and additional model parameterizations. Specifically, we rely on the functional form specification in Equation (3), the assumption of joint normality between the errors that determine the latent propensities for survival and dementia, and the existence of instruments, M_i, that affect survival but not dementia, conditional on the controls.

Results

TABLE H1—AVERAGE MARGINAL EFFECTS ALLOWING FOR SELECTION ON SURVIVAL

	(1)	(2)	(3)
1 μg/m^3 increase in decadal $PM_{2.5}$	[0.693, 4.839] (-0.118, 6.222)	[1.707, 4.839] (0.836, 6.222)	2.384*** (0.568)
number of individuals: survival outcome	2,439,904	2,439,904	2,439,904
number of individuals: dementia function	1,179,094	1,179,094	1,179,094
share with dementia in 2013	20	20	20
share who survive through 2013	61	61	61

Note: The dependent variable equals 100 if an individual was diagnosed with dementia prior to the end of 2013 and 0 otherwise. Column (1) reports the identification region in brackets from a simple, worst-case bounding approach. Column (2) reports the identification region in brackets after imposing an additional assumption of positive correlation between the latent health of survival and the latent health of cognition to sharpen the bounds. Finally, Column (3) reports results from the decadal specification shown in Column (5) of Table I that uses exclusion restrictions to arrive at a point estimate. Underneath the identification regions in Columns (1)-(2), we report in parentheses the 95% confidence intervals using the method described in Imbens and Manski (2004). Underneath the point estimate in Column (3), we report a standard error. The standard errors underlying the confidence intervals in Columns (1)-(2) and the standard error in Column (3) are calculated using a bootstrap with 1,000 repetitions, clustered at initial Census block group. Asterisks indicate statistical significance for Column (3) at the 10% (*), 5% (**), and 1% (***) levels.

We first calculate bounds that do not rely on assumptions regarding the correlation between the unobserved factors that determine the propensity to be diagnosed with dementia and the propensity to survive. The estimated identification region for the average causal marginal effect of decadal $PM_{2.5}$ on new dementia diagnoses is shown in brackets in the Column (1) of Table H1. The lower bound of the identification region (shown in brackets) is 0.69, despite embedding the extreme assumption that individuals who are induced to die by an increase in $PM_{2.5}$ would have a zero probability of being diagnosed with dementia had they survived. When we compare this with our estimate of the average total marginal effect, $\bar{A}_{pm}$ =1.71, we conclude that even in this worst-case scenario, only 59% of the total effect would be attributed to a compositional effect. A 95%

confidence interval for the AME, is shown below the identification region (in parentheses) and is calculated following Imbens and Manski (2004). Overall, our lower-bound results show that a zero causal effect of $PM_{2.5}$ on dementia is unlikely, and our upper-bound results do not rule out causal effects much larger than what we find in Table I, Column (5).

We then sharpen the bounds by assuming a plausible form of monotone treatment selection (Manski and Pepper 2000) in which individuals who are induced to die when exposed to an increase in $PM_{2.5}$ are no less likely to be diagnosed with dementia than those whose survival was unaffected by an increase in exposure. Table H1, Column (2) shows that under this assumption the lower bound of the identification region increases to our estimate of the average total marginal effect, 1.71. For completeness, Column (3) of Table H1 repeats the point estimate from our main decadal specification (Column (5) of Table I), which relies on estimating the correlation between the propensity to develop dementia and the propensity to survive.

Because our model allows us to calculate heterogeneity in the average causal marginal effects of $PM_{2.5}$, we are able to calculate bounds separately for three subsamples with average decadal $PM_{2.5}$ within 1 $\mu g/m^3$ windows centered around 10, 11, and 12 $\mu g/m^3$. The identification regions of the average marginal effects for these three subsamples are [1.47, 4.91], [0.51, 4.88], and [-0.29, 4.82], respectively. Intuitively, the lower bounds are decreasing in $PM_{2.5}$ because the magnitude of the estimated total effect is decreasing across the three bins. In addition, the width of the identification regions are increasing in $PM_{2.5}$ as a result of mortality increasing in $PM_{2.5}$. The lower bounds reflect the extreme assumption that those who suffered from $PM_{2.5}$-driven mortality would have been immune to dementia diagnoses had they survived. Applying the assumption that these individuals were merely no less sensitive than the survivors in terms of dementia sharpens the bounds. Under this assumption, the identification regions become [2.30, 4.91], [1.59, 4.88], and [0.99, 4.82], respectively.

I. Additional results referenced in Sections III and IV

1. Effect of $PM_{2.5}$ Exposure on Mortality

TABLE I1—AVERAGE MARGINAL EFFECTS OF DECADAL EXPOSURE TO $PM_{2.5}$ ON MORTALITY

	(1)	(2)	(3) or (4)	(5)
1 μg/m^3 increase in decadal $PM_{2.5}$	0.542***	0.390***	2.430***	2.475***
	(0.056)	(0.083)	(0.485)	(0.476)
ind. & neigh. covariates		x	x	x
$PM_{2.5}$ control function			x	x
polynomial functions of covariates				x
first-stage F statistic			611	611
number of individuals	2,439,904	2,439,904	2,439,904	2,439,904
share who die before Jan 1, 2014	39	39	39	39

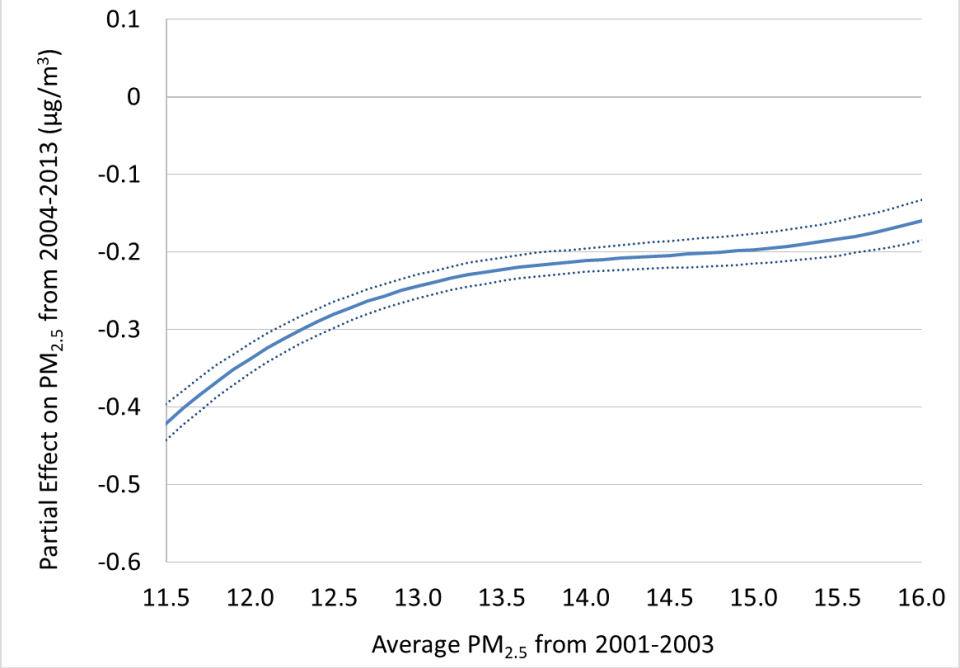
Note: The dependent variable equals 100 if an individual died on or before December 31, 2013 and 0 otherwise. The columns are numbered to correspond to the specifications in the corresponding columns of Table I in the main text. Col (1) is a probit model with CBSA-specific intercepts. Col (2) adds all covariates for baseline health in 2004, individual demographics, demographics for the individual's Census block group, and pre-regulatory $PM_{2.5}$ levels at their residence from 2001-2003. Col (3) or (4) is the 2SCML analogue to Col (2) and Col (5) adds polynomial functions of covariates as shown in Equation (5) of the main text. The first row presents the average marginal effect of decadal $PM_{2.5}$ on mortality. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered at the block group. Standard errors in Columns (3) or (4) and (5) are bootstrapped using 1,000 repetitions.

Table I1 shows results from repeating estimation of the decadal models shown in Columns (1) through (5) of Table I using mortality as the outcome. The models and covariates are otherwise the same as in Table I, except that Columns (3) or (4), and (5) of Table I1 exclude the mortality control function. The Column "(3) or (4)" label highlights that the results in that column can be viewed as an analog to the results in either Column (3) or Column (4) of Table I because the only difference between those two columns is the mortality control function that we exclude here. The flexible 2SCML specification in Column (5) implies that a 1 μg/m^3 increase in average $PM_{2.5}$ exposure from 2004 through 2013 increased the probability of a death by the end of 2013 by 2.48 percentage points. This AME is six times larger than the AME shown in Column (2). Column (3) or (4) shows that the six-fold increase in the AME is almost entirely due to using the nonattainment instruments for $PM_{2.5}$ exposure. Comparing Columns (3) or (4) and (5) shows that adding polynomial functions of covariates has very little effect on the resulting AME.

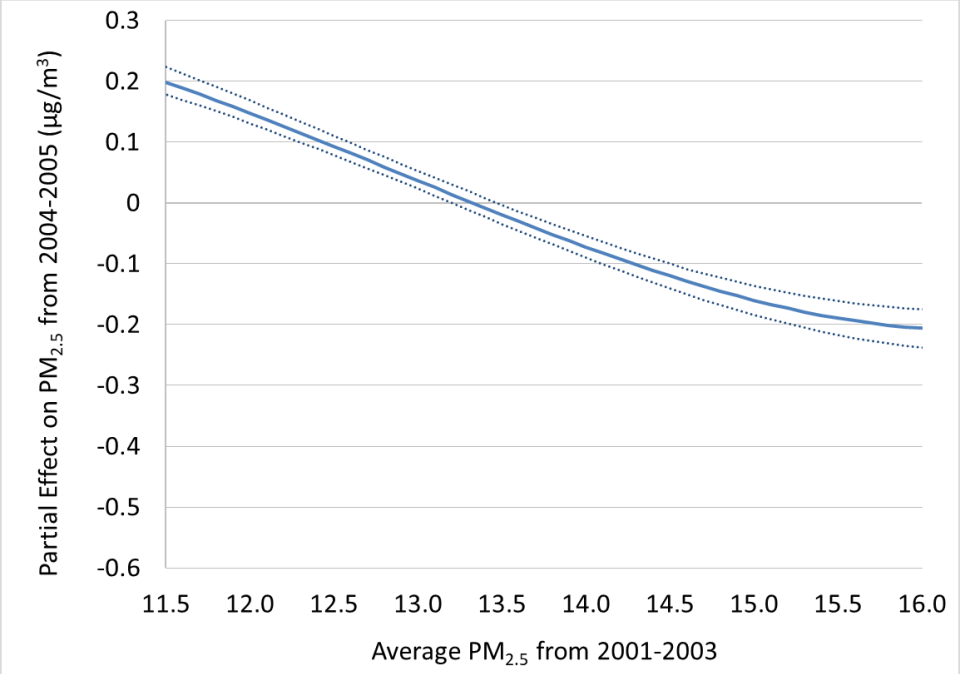
2. *Duration-Specific Partial Effects of Nonattainment on $PM_{2.5}$ Exposure*

The EPA's nonattainment designations created quasi-random differences in pollution exposure conditional on $basePM_i$ and the additional controls in Equation (2) and Equation (9). Figure I1 shows this identifying variation for the decade using Equation (2) in Panel (i) and for each year $t = [2005,2012]$ using Equation (9) in Panels (ii)-(ix). Each panel plots the coefficients on the instruments from the relevant regression equation to show the estimated partial effect of nonattainment across different levels of $basePM_i$. Intuitively, the partial effects are negative (with the exception of low baseline concentrations in the first year of the policy), showing that nonattainment status reduced pollution.

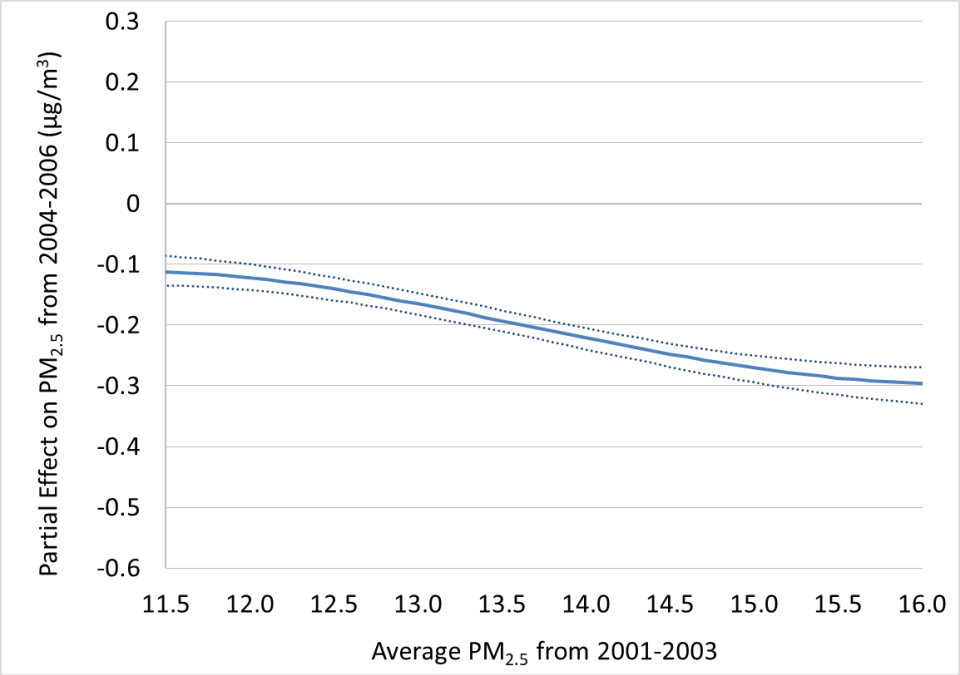
FIGURE I1— ESTIMATED PARTIAL EFFECTS OF NONATTAINMENT (NA) ON $PM_{2.5}$ EXPOSURE



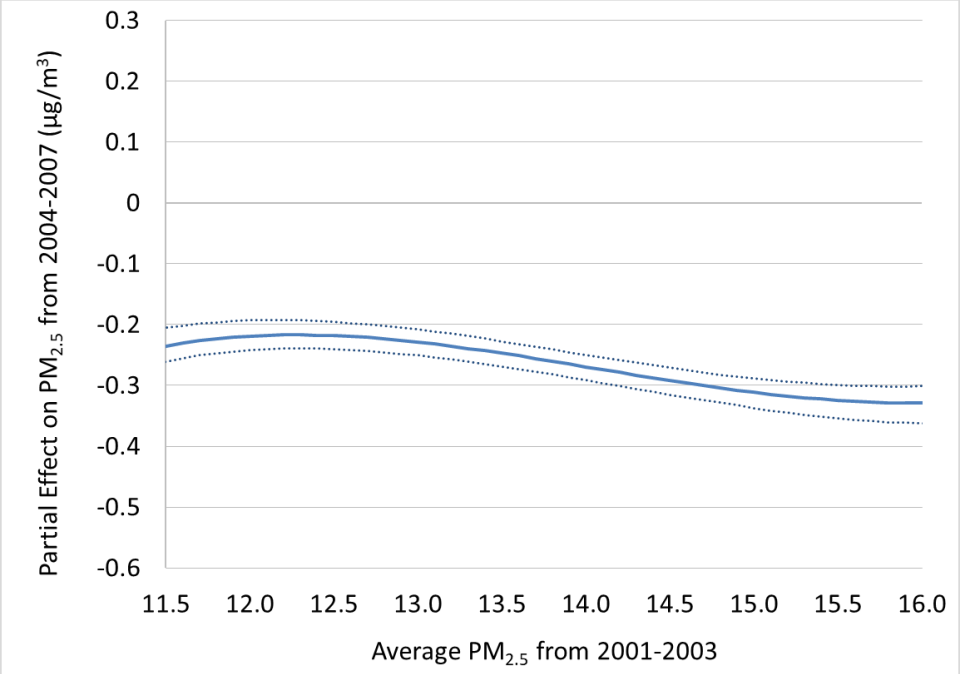
Panel i: Estimated Partial Effect of NA on Decadal $PM_{2.5}$ Exposure, 2004-2013



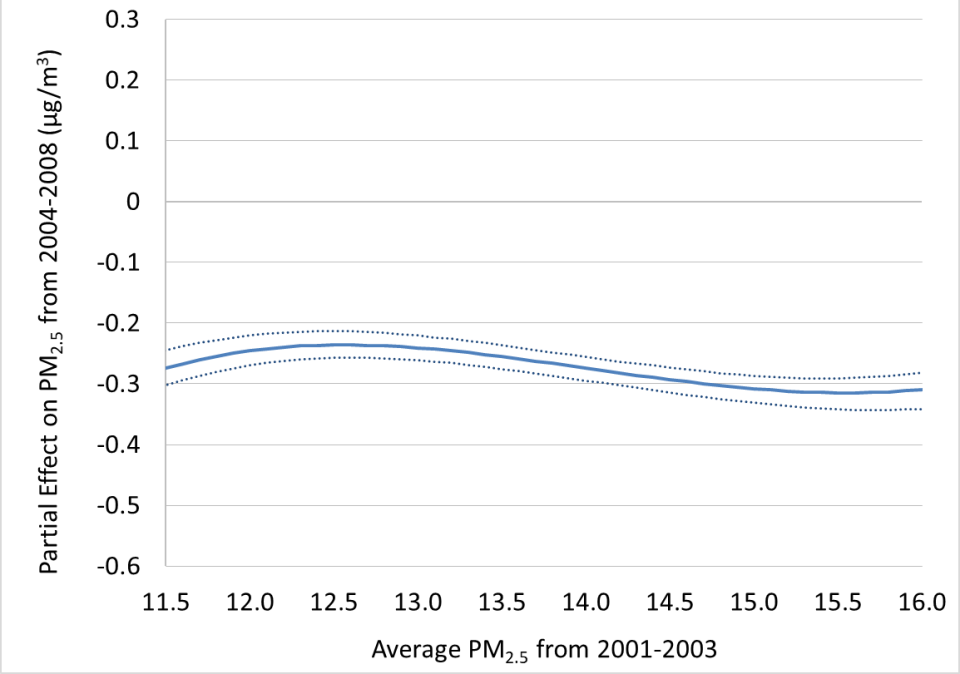
Panel ii: Estimated Partial Effect of NA on $PM_{2.5}$ Exposure, 2004-2005



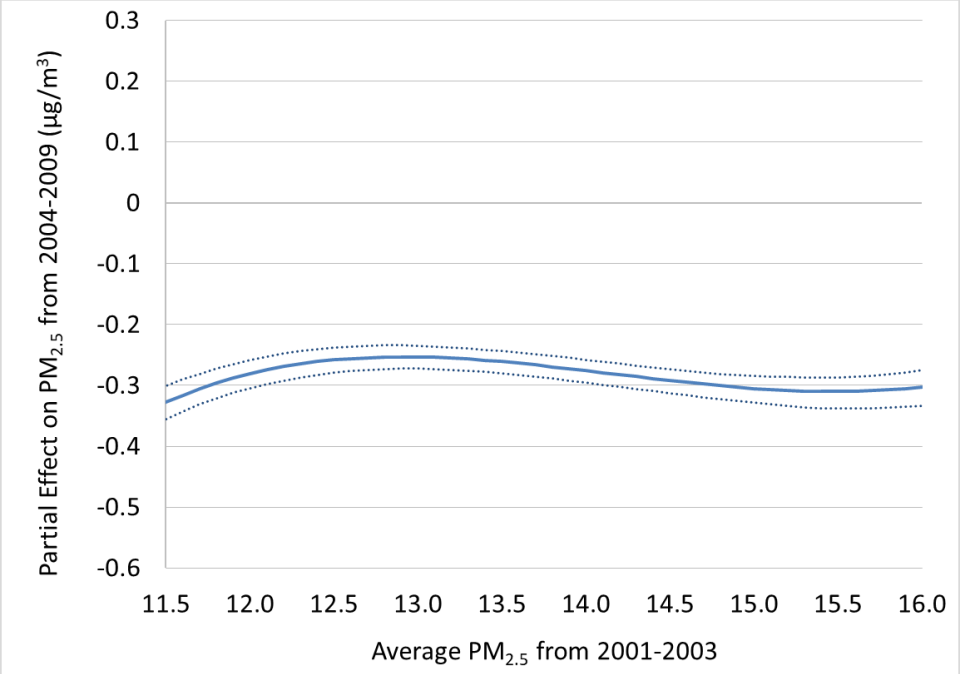
Panel iii: Estimated Partial Effect of NA on $PM_{2.5}$ Exposure, 2004-2006



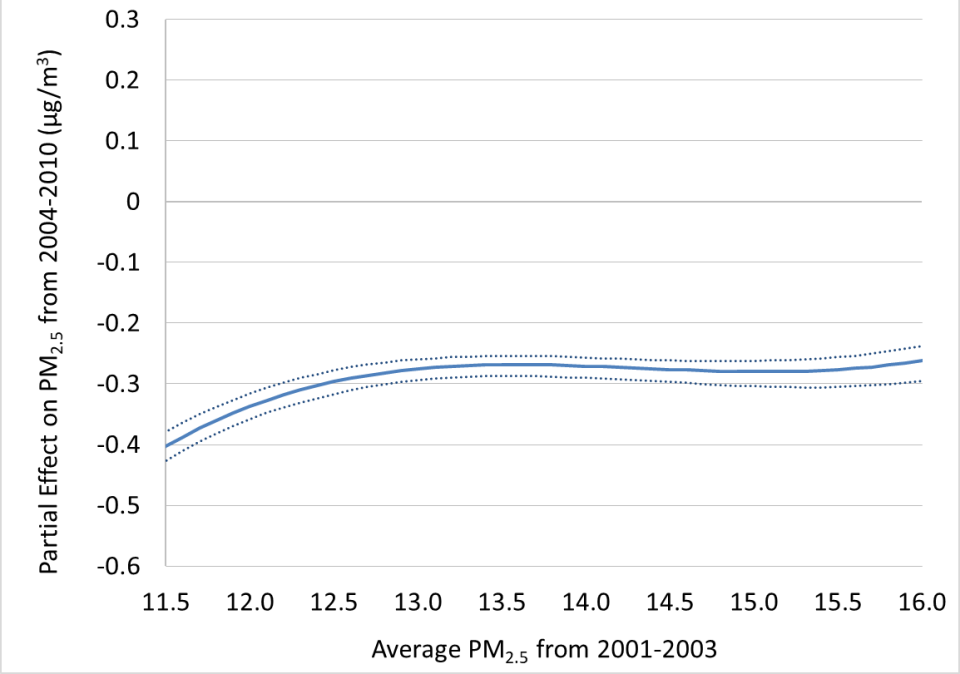
Panel iv: Estimated Partial Effect of NA on $PM_{2.5}$ Exposure, 2004-2007



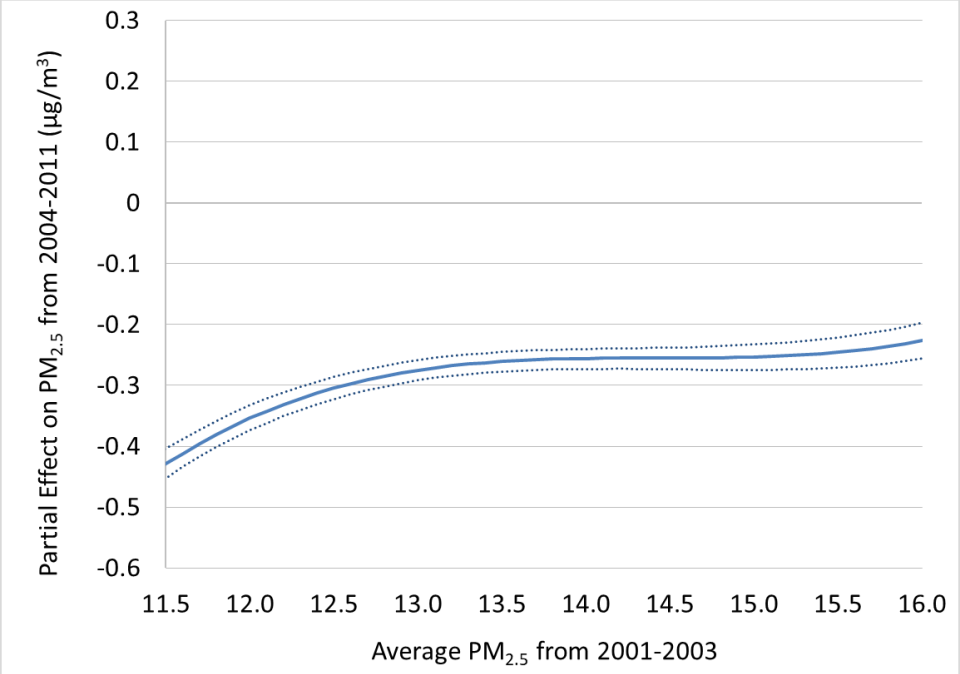
Panel v: Estimated Partial Effect of NA on $PM_{2.5}$ Exposure, 2004-2008



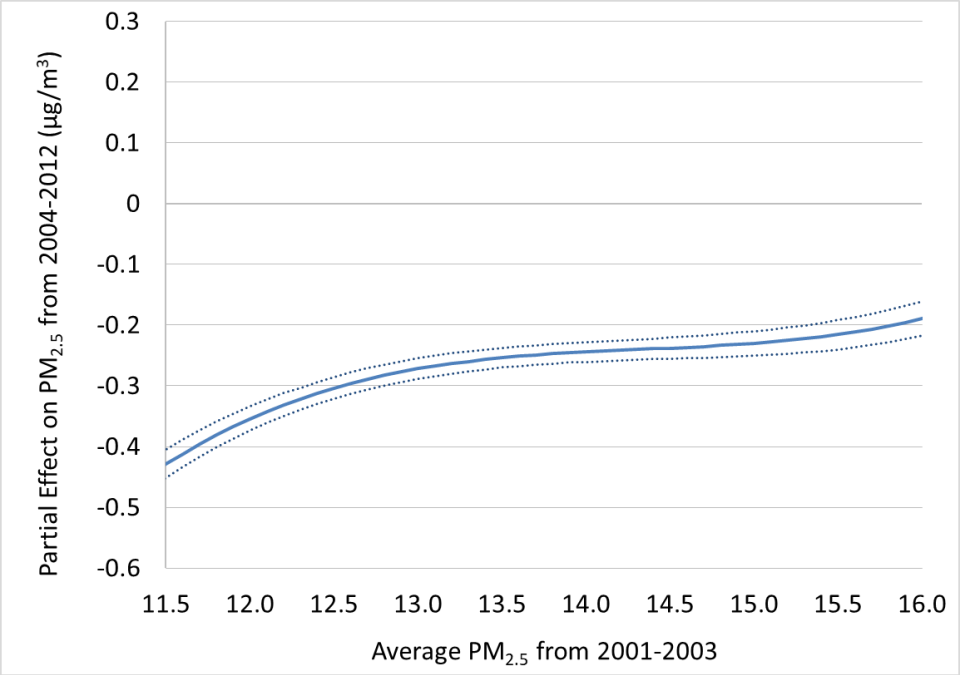
Panel vi: Estimated Partial Effect of NA on $PM_{2.5}$ Exposure, 2004-2009



Panel vii: Estimated Partial Effect of NA on $PM_{2.5}$ Exposure, 2004-2010



Panel viii: Estimated Partial Effect of NA on $PM_{2.5}$ Exposure, 2004-2011



Panel ix: Estimated Partial Effect of NA on $PM_{2.5}$ Exposure, 2004-2012

3. Average Marginal Effects of Cancer Instruments in Equation (3)

TABLE I2—AVERAGE MARGINAL EFFECTS FOR CANCER INSTRUMENTS IN THE SURVIVAL FUNCTION

Breast cancer in 2004	-3.87***
	(0.13)
Prostate cancer in 2004	-0.47***
	(0.13)
Colorectal cancer in 2004	-3.57***
	(0.17)
Endometrial cancer in 2004	-4.81***
	(0.35)
Leukemia/Lymphoma in 2004	-11.73***
	(0.25)
number of individuals	2,439,904
share who survive through 2013	61

Note: The dependent variable equals 100 if an individual survived through the end of 2013. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered at the Census block group.

Table I2 shows average marginal effects of the cancer instruments from the survival function. The AMEs are reported as percentage point changes in the probability of survival. For example, a pre-existing diagnosis of colorectal cancer in 2004 reduced the probability of survival through the end of 2013 by 3.57pp.

4. Coefficient Estimates for Equations (4) and (5)

Table I3 shows estimates for the decadal specification of the outcome equation reported in Column (5) of Table I, followed by the estimates of the first-stage $PM_{2.5}$ function and survival function. In the interest of brevity, we report results from the decadal model and suppress results for the 900+ CBSA indicators. Additional coefficients from the models with varying durations of exposure are available upon request (as are the CBSA coefficients for this model). In the table of results, we use the following abbreviations for chronic conditions in 2004: hypertension (H), stroke (S), diabetes (D), ischemic heart disease (I), and congestive heart failure (C). We use "cf_pm2.5" and "cf_survival" to represent the control functions for $PM_{2.5}$ and survival. The excluded reference categories are: age (75); CMS race code ("other"), Census block group education attainment (%

with 8th grade or less); Census block group housing stock (% vacant). Confidence intervals are based on 1,000 bootstrap repetitions, clustered at the Census block group level.

TABLE I3: RESULTS FROM THE DECADAL 2SCML SPECIFICATION
OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
$PM_{2.5}$ (1 µg/m^3) (Decadal, 2004-2013)	37.495	25.849	-12.319	90.670
$(PM_{2.5})^2$	4.999	2.932	-0.485	10.526
$(PM_{2.5})^3$	-0.463	0.169	-0.785	-0.149
$(PM_{2.5})^4$	0.012	0.004	0.005	0.019
$PM_{2.5}$ (1 µg/m^3) interacted with:				
2004 Gross Medicare Expenditures ($10,000)				
expenditures	1.371	0.968	-0.460	3.307
expenditures^2	-0.806	0.375	-1.569	-0.116
expenditures^3	0.108	0.048	0.018	0.204
expenditures^4	-0.004	0.002	-0.008	0.000
2004 Census Block Group Demographics				
median household income / 1000	-0.003	0.007	-0.015	0.011
per capita income / 1000	0.005	0.011	-0.016	0.026
median year built	-0.010	0.006	-0.023	0.002
median house value / 1000	-0.001	0.001	-0.003	0.000
average house value / 1000	0.000	0.000	-0.001	0.001
median gross rent / 1000	-0.006	0.019	-0.044	0.031
% over 65	-2.130	0.932	-3.948	-0.289
% white	1.034	0.933	-0.715	2.931
% black	-0.156	1.146	-2.374	2.016
% hispanic	-4.627	1.030	-6.661	-2.544
% 9th through 12th	-3.066	2.514	-7.542	1.940
% high school graduate	-4.993	1.863	-8.708	-1.250
% some college	-4.784	1.841	-8.414	-1.409
% associate degree	-2.587	2.374	-7.167	2.445
% bachelor's degree	-7.199	1.907	-11.229	-3.296
% graduate degree	-7.026	2.114	-11.090	-2.992
% owner occupied	-2.583	1.011	-4.495	-0.543
% renter occupied	-1.314	1.083	-3.457	0.763

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
$PM_{2.5}$ (1 μg/m^3) interacted with:				
<u>Chronic conditions in 2004</u>				
H	0.234	0.261	-0.279	0.745
S	0.706	1.201	-1.591	3.203
S, H	0.566	0.734	-0.853	2.064
D	1.452	0.691	0.156	2.837
D, H	1.320	0.481	0.336	2.231
D, S	1.194	3.301	-5.412	7.599
D, S, H	4.264	1.318	1.788	6.811
I	0.550	0.459	-0.365	1.411
I, H	1.183	0.338	0.543	1.785
I, S	2.000	1.540	-0.894	5.132
I, S, H	1.633	0.786	0.155	3.128
I, D	2.775	1.086	0.673	4.798
I, D, H	1.953	0.544	0.840	3.033
I, D, S	7.369	3.878	-0.828	15.051
I, D, S, H	2.730	1.137	0.495	5.005
C	4.571	1.520	1.770	7.546
C, H	2.433	0.923	0.619	4.157
C, S	0.145	5.631	-10.370	11.557
C, S, H	1.391	1.959	-2.669	5.234
C, D	5.493	3.179	-0.322	11.770
C, D, H	3.709	1.290	1.139	6.253
C, D, S	-13.811	14.365	-39.912	17.850
C, D, S, H	4.332	2.823	-1.022	10.229
C, I	3.318	1.333	0.687	5.861
C, I, H	3.310	0.858	1.617	5.004
C, I, S	2.771	3.168	-3.593	9.318
C, I, S, H	3.391	1.266	0.937	5.960
C, I, D	4.865	2.609	-0.188	9.968
C, I, D, H	4.804	1.161	2.355	7.138
C, I, D, S	2.567	6.684	-10.026	15.451
C, I, D, S, H	4.473	1.584	1.203	7.558

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
$PM_{2.5}$ (1 µg/m^3) interacted with:				
Age				
76	-0.254	0.700	-1.641	1.182
77	0.482	0.663	-0.844	1.803
78	0.331	0.672	-1.008	1.645
79	0.942	0.685	-0.455	2.197
80	1.074	0.688	-0.236	2.382
81	0.583	0.707	-0.898	1.949
82	0.600	0.753	-0.890	2.106
83	0.301	0.770	-1.333	1.779
84	0.592	0.828	-1.070	2.173
85	1.661	0.875	-0.144	3.389
86	1.787	0.925	-0.157	3.613
87	2.362	0.998	0.348	4.318
88	2.789	1.115	0.569	4.847
89	1.759	1.224	-0.755	4.044
90	3.542	1.361	0.828	6.265
91	4.751	1.500	1.740	7.719
92	3.843	1.626	0.391	6.940
93	5.172	1.808	1.449	8.681
94	6.574	1.982	2.513	10.393
95	4.481	2.132	0.227	8.305
96	5.643	2.350	0.650	10.188
97	7.890	2.579	2.766	12.553
98	4.756	2.831	-1.017	10.233
99	7.518	3.038	1.315	13.517
100 and over	8.793	3.274	1.805	15.021
control functions				
cf_pm2.5	1.242	0.367	0.534	1.962
$(cf\_pm2.5)^2$	-0.165	0.093	-0.334	0.030
cf_survival	-11.082	3.412	-17.540	-3.984
$(cf\_survival)^2$	2.429	0.878	0.540	4.042

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
$PM_{2.5}$ (1 μg/m^3) interacted with:				
Age x male				
76	-0.081	1.110	-2.218	2.034
77	-0.915	1.033	-2.901	1.112
78	-0.484	1.039	-2.510	1.492
79	-1.989	1.049	-3.995	0.207
80	-2.501	1.044	-4.611	-0.440
81	-0.356	1.028	-2.385	1.768
82	-1.247	1.065	-3.293	0.757
83	0.366	1.043	-1.700	2.494
84	-0.744	1.045	-2.848	1.282
85	-1.026	1.041	-2.975	1.019
86	0.368	1.073	-1.688	2.435
87	-0.050	1.080	-2.239	2.177
88	-0.734	1.100	-2.972	1.499
89	0.395	1.168	-2.013	2.639
90	-0.231	1.198	-2.585	2.051
91	-0.488	1.279	-2.963	2.065
92	-1.124	1.325	-3.798	1.404
93	-0.585	1.465	-3.533	2.147
94	-0.743	1.678	-3.937	2.478
95	2.426	1.822	-1.191	5.974
96	-0.510	2.200	-4.909	3.764
97	-1.867	2.615	-6.992	3.343
98	2.266	3.329	-4.198	8.830
99	2.361	4.055	-5.530	10.321
100 and over	-2.329	3.005	-8.430	3.353
individual demographics				
male	1.625	0.808	-0.058	3.128
White	-1.114	0.737	-2.510	0.338
Black or African American	-1.033	0.866	-2.697	0.677
Asian	-1.029	0.787	-2.563	0.513
Hispanic	-2.882	0.782	-4.502	-1.330

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
cf_survival	75.020	38.070	-6.040	146.613
$(cf\_survival)^2$	-32.390	9.822	-50.539	-11.592
cf_pm2.5	-22.278	4.816	-32.406	-13.454
$(cf\_pm2.5)^2$	3.923	1.129	1.623	6.061
Chronic conditions in 2004				
H	1.114	2.872	-4.556	6.641
S	24.993	13.155	-2.052	49.909
S, H	30.579	8.086	14.282	46.116
D	0.742	7.647	-14.363	15.759
D, H	3.059	5.291	-7.071	14.309
D, S	46.719	36.678	-24.743	119.243
D, S, H	6.478	14.671	-21.305	34.500
I	2.313	5.075	-7.195	12.233
I, H	-1.932	3.761	-8.770	5.195
I, S	17.399	17.179	-16.960	49.854
I, S, H	23.951	8.773	6.994	41.027
I, D	-8.540	12.063	-31.620	14.825
I, D, H	3.580	6.048	-8.165	16.141
I, D, S	-43.365	43.638	-132.063	46.768
I, D, S, H	27.807	12.761	2.537	52.483
C	-21.021	16.852	-54.720	11.104
C, H	0.148	10.286	-19.045	20.810
C, S	46.470	63.352	-84.456	163.363
C, S, H	41.605	21.784	-1.952	86.917
C, D	-11.695	35.429	-81.698	52.420
C, D, H	4.904	14.389	-23.253	33.639
C, D, S	230.404	162.408	-124.224	527.102
C, D, S, H	31.873	32.111	-36.656	93.936
C, I	-5.788	14.881	-34.375	23.194
C, I, H	-4.123	9.580	-22.052	14.570
C, I, S	20.891	34.838	-47.472	89.692
C, I, S, H	25.112	14.238	-2.820	52.601
C, I, D	-4.752	28.891	-59.831	52.782
C, I, D, H	-1.396	12.918	-26.061	25.325
C, I, D, S	63.500	73.935	-79.146	202.917
C, I, D, S, H	40.215	17.723	5.197	76.292

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
2004 Gross Medicare Expenditures ($10,000)				
expenditures	-7.819	10.659	-29.167	12.259
expenditures^2	5.296	4.131	-2.187	13.784
expenditures^3	-0.726	0.523	-1.803	0.232
expenditures^4	0.032	0.022	-0.009	0.075
Age				
76	9.204	7.770	-6.168	24.592
77	7.389	7.326	-7.101	22.315
78	15.354	7.483	0.466	29.949
79	15.956	7.597	1.726	30.990
80	23.105	7.598	9.035	37.284
81	33.874	7.874	18.559	50.206
82	41.560	8.319	24.868	57.412
83	53.714	8.542	37.905	71.564
84	56.599	9.212	38.384	75.077
85	54.996	9.699	36.112	75.018
86	62.767	10.215	43.225	83.703
87	64.327	11.032	42.569	86.685
88	68.824	12.357	45.467	92.894
89	89.696	13.592	64.143	117.638
90	78.423	15.127	47.727	108.520
91	73.593	16.613	42.018	108.464
92	90.994	18.024	56.684	128.590
93	85.063	20.067	47.948	126.895
94	81.527	21.952	39.800	127.324
95	113.801	23.698	70.727	161.665
96	112.704	26.117	62.279	167.530
97	96.006	28.771	42.532	155.848
98	140.668	31.415	82.014	203.274
99	121.001	34.031	55.085	187.834
100 and over	120.070	36.441	50.700	194.638

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
Age x male				
76	-0.231	12.290	-24.162	22.733
77	9.149	11.410	-13.508	31.248
78	4.628	11.630	-18.078	27.070
79	19.920	11.707	-3.474	42.810
80	23.816	11.628	1.334	47.623
81	-0.833	11.492	-24.485	21.816
82	11.221	11.755	-10.489	34.176
83	-7.765	11.617	-31.609	14.877
84	5.532	11.691	-16.046	29.412
85	7.645	11.560	-14.559	30.072
86	-7.925	11.886	-31.957	14.554
87	-4.806	12.058	-29.330	19.385
88	2.392	12.239	-21.791	26.867
89	-9.709	13.018	-34.658	17.078
90	-3.295	13.295	-29.142	23.396
91	-3.963	14.169	-32.538	23.925
92	6.991	14.648	-22.047	36.095
93	-0.554	16.258	-32.113	32.857
94	-1.182	18.393	-35.859	33.604
95	-36.316	20.098	-75.501	2.717
96	-3.478	24.304	-49.600	46.066
97	16.694	29.169	-41.674	72.660
98	-38.612	36.434	-110.867	32.419
99	-39.495	44.344	-126.756	46.923
100 and over	8.068	33.451	-53.474	75.826

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
individual demographics				
male	-18.971	8.986	-35.928	-0.288
White	16.074	7.915	0.607	31.268
Black or African American	24.738	9.475	5.794	43.434
Asian	9.510	8.418	-7.172	26.376
Hispanic	41.351	8.409	25.348	58.641
2004 Census Block Group Demographics				
median household income / 1000	0.003	0.074	-0.148	0.148
per capita income / 1000	-0.073	0.120	-0.297	0.164
median year built	0.092	0.070	-0.050	0.222
median house value / 1000	0.001	0.010	-0.018	0.022
average house value / 1000	0.001	0.004	-0.008	0.010
median gross rent / 1000	0.133	0.213	-0.260	0.555
% over 65	15.884	10.136	-3.654	36.214
% white	-4.454	10.315	-24.675	15.135
% black	12.282	12.939	-13.480	37.991
% hispanic	53.817	11.384	29.783	76.231
% 9th through 12th	37.372	28.489	-20.266	89.031
% high school graduate	39.389	20.932	-2.056	80.630
% some college	28.407	20.694	-9.611	68.211
% associate degree	-4.222	26.527	-61.196	47.685
% bachelor's degree	49.971	21.488	5.327	93.467
% graduate degree	49.453	23.686	2.762	94.380
% owner occupied	16.508	11.045	-4.658	37.178
% renter occupied	21.308	11.842	-1.127	45.274
$PM_{2.5}$ (1 µg/m3) baseline (2001-2003)	-35.936	8.987	-54.736	-18.638
$(PM_{2.5})^2$	2.481	0.912	0.681	4.275
$(PM_{2.5})^3$	-0.090	0.040	-0.168	-0.012
$(PM_{2.5})^4$	0.001	0.001	0.000	0.003

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: DECADAL $PM_{2.5}$ (2004-2013)

	Coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
nonattainment	-2324.333	135.876	-2601.440	-2050.451
nonattainment x $PM_{2.5}$ (1 µg/m3) (2001-2003)	490.520	34.381	422.867	560.052
nonattainment x $(PM_{2.5})^2$	-35.879	3.390	-42.638	-29.210
nonattainment x $(PM_{2.5})^3$	0.967	0.156	0.657	1.273
nonattainment x $(PM_{2.5})^4$	-0.005	0.003	-0.010	0.001
$PM_{2.5}$ (1 µg/m3) (2001-2003)	84.926	15.027	54.034	114.135
$(PM_{2.5})^2$	-6.773	2.008	-10.648	-2.600
$(PM_{2.5})^3$	0.556	0.115	0.315	0.773
$(PM_{2.5})^4$	-0.016	0.002	-0.020	-0.011
cf_survival	-2.481	0.811	-4.077	-0.804
2004 Gross Medicare Expenditures ($10,000)				
expenditures	0.161	0.359	-0.540	0.877
expenditures^2	-0.039	0.139	-0.312	0.228
expenditures^3	-0.004	0.018	-0.039	0.033
expenditures^4	0.001	0.001	-0.001	0.002
individual demographics				
male	0.463	0.246	-0.061	0.903
White	-1.102	0.379	-1.892	-0.359
Black or African American	-0.396	0.416	-1.247	0.436
Asian	0.738	0.511	-0.246	1.729
Hispanic	1.414	0.440	0.564	2.247
Chronic conditions in 2004				
H	-0.133	0.096	-0.330	0.058
S	0.153	0.517	-0.860	1.147
S, H	0.670	0.296	0.067	1.265
D	-0.422	0.267	-0.953	0.097
D, H	-0.162	0.158	-0.475	0.152
D, S	-0.044	1.324	-2.492	2.704
D, S, H	0.402	0.517	-0.600	1.361
I	-0.169	0.183	-0.532	0.176

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: DECADAL $PM_{2.5}$ (2004-2013)

	Coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
I, H	-0.105	0.123	-0.348	0.128
I, S	-0.067	0.705	-1.530	1.291
I, S, H	0.397	0.299	-0.210	0.969
I, D	-0.238	0.420	-1.045	0.581
I, D, H	0.005	0.193	-0.381	0.382
I, D, S	-3.051	1.829	-6.544	0.720
I, D, S, H	0.287	0.447	-0.563	1.141
C	0.466	0.584	-0.764	1.584
C, H	0.425	0.329	-0.210	1.069
C, S	-1.697	2.060	-5.654	2.347
C, S, H	0.053	0.904	-1.677	1.850
C, D	0.813	1.416	-2.094	3.451
C, D, H	0.944	0.499	-0.061	1.945
C, D, S	-3.764	3.370	-10.680	2.823
C, D, S, H	0.838	1.153	-1.388	3.080
C, I	1.335	0.530	0.298	2.307
C, I, H	0.861	0.310	0.209	1.474
C, I, S	0.290	1.593	-2.925	3.237
C, I, S, H	1.148	0.545	0.054	2.178
C, I, D	1.394	0.955	-0.501	3.162
C, I, D, H	1.431	0.425	0.594	2.281
C, I, D, S	4.375	3.808	-3.142	11.797
C, I, D, S, H	1.576	0.655	0.331	2.779
Age				
76	0.390	0.215	-0.029	0.796
77	0.223	0.215	-0.191	0.653
78	0.330	0.216	-0.092	0.748
79	0.125	0.221	-0.301	0.560
80	0.577	0.221	0.113	0.993
81	0.439	0.222	0.017	0.869
82	0.933	0.245	0.445	1.391
83	0.785	0.247	0.243	1.258

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: DECADAL $PM_{2.5}$ (2004-2013)

	Coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
84	0.818	0.268	0.252	1.333
85	1.031	0.289	0.436	1.554
86	1.126	0.303	0.496	1.681
87	1.252	0.340	0.559	1.893
88	1.337	0.371	0.585	2.066
89	1.624	0.410	0.804	2.420
90	1.624	0.439	0.670	2.425
91	1.643	0.509	0.601	2.600
92	2.277	0.541	1.146	3.325
93	1.938	0.619	0.683	3.204
94	1.940	0.716	0.453	3.361
95	2.533	0.801	0.913	4.122
96	2.078	0.889	0.363	3.832
97	2.552	0.992	0.621	4.535
98	3.798	1.079	1.723	5.783
99	3.794	1.287	1.085	6.170
100 and over	3.432	1.307	0.643	5.871
Age x male				
76	-0.338	0.320	-0.946	0.299
77	-0.150	0.325	-0.779	0.487
78	-0.272	0.343	-0.929	0.413
79	-0.244	0.345	-0.893	0.489
80	-0.168	0.342	-0.809	0.534
81	0.029	0.338	-0.612	0.706
82	-0.452	0.333	-1.073	0.235
83	-0.313	0.340	-0.937	0.372
84	-0.400	0.355	-1.052	0.327
85	-0.078	0.363	-0.796	0.652
86	-0.239	0.367	-0.922	0.500
87	-0.759	0.396	-1.522	0.069
88	-0.067	0.420	-0.921	0.776

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: DECADAL $PM_{2.5}$ (2004-2013)

	Coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
89	-0.135	0.421	-0.909	0.721
90	-0.284	0.458	-1.165	0.620
91	-0.149	0.475	-1.031	0.782
92	-0.320	0.520	-1.413	0.687
93	0.921	0.624	-0.255	2.148
94	1.050	0.722	-0.330	2.490
95	0.263	0.840	-1.374	2.032
96	0.905	0.970	-0.949	2.872
97	0.932	1.178	-1.479	3.138
98	-0.257	1.679	-3.585	3.077
99	-2.440	1.995	-6.061	1.778
100 and over	0.447	1.365	-2.265	3.142
2004 Census Block Group Demographics				
median household income / 1000	-0.054	0.006	-0.067	-0.041
per capita income / 1000	0.174	0.012	0.152	0.200
median year built	-0.015	0.006	-0.027	-0.004
median house value / 1000	-0.014	0.001	-0.016	-0.011
average house value / 1000	0.000	0.000	-0.001	0.001
median gross rent / 1000	-0.005	0.017	-0.040	0.028
% over 65	-7.735	1.184	-10.059	-5.404
% white	7.929	1.151	5.639	10.231
% black	6.325	1.201	3.910	8.735
% hispanic	9.512	1.312	6.917	12.037
% 9th through 12th	-10.908	2.539	-15.935	-6.218
% high school graduate	-8.834	2.105	-13.070	-4.943
% some college	-13.446	2.130	-17.751	-9.342
% associate degree	-22.898	2.500	-28.012	-18.185
% bachelor's degree	-4.590	2.123	-8.765	-0.491
% graduate degree	-4.788	2.307	-9.348	-0.515
% owner occupied	-5.385	0.985	-7.284	-3.461
% renter occupied	-0.389	1.060	-2.406	1.749

R^2 = 0.961

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: SURVIVAL TO 2013

	Probit Coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
Breast cancer in 2004	-12.697	0.442	-13.556	-11.890
Prostate cancer in 2004	-1.561	0.427	-2.382	-0.753
Colorectal cancer in 2004	-11.709	0.559	-12.792	-10.481
Endometrial cancer in 2004	-15.767	1.112	-18.137	-13.617
Leukemia / Lymphoma in 2004	-38.477	0.812	-40.070	-36.914
nonattainment	-15.349	63.663	-135.858	111.910
nonattainment x $PM_{2.5}$ (1 µg/m3) (2001-2003)	-5.513	18.127	-42.814	29.810
nonattainment x $(PM_{2.5})^2$	1.799	2.037	-2.335	5.905
nonattainment x $(PM_{2.5})^3$	-0.139	0.106	-0.349	0.070
nonattainment x $(PM_{2.5})^4$	0.003	0.002	-0.001	0.007
$PM_{2.5}$ (1 µg/m3) (2001-2003)	33.157	11.683	10.298	55.951
$(PM_{2.5})^2$	-4.288	1.575	-7.296	-1.212
$(PM_{2.5})^3$	0.238	0.092	0.056	0.413
$(PM_{2.5})^4$	-0.005	0.002	-0.008	-0.001
2004 Gross Medicare Expenditures ($10,000)				
expenditures	-8.665	0.931	-10.452	-6.796
expenditures^2	5.269	0.357	4.570	5.958
expenditures^3	-0.521	0.045	-0.609	-0.430
expenditures^4	0.007	0.002	0.004	0.011
individual demographics				
male	-24.434	0.775	-25.922	-22.872
White	-7.181	1.046	-9.254	-5.202
Black or African American	0.235	1.114	-1.914	2.397
Asian	17.681	1.206	15.400	20.035
Hispanic	13.367	1.146	11.077	15.694

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: SURVIVAL TO 2013

	Probit Coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
Chronic conditions in 2004				
H	-5.186	0.285	-5.752	-4.631
S	-19.657	1.319	-22.120	-16.884
S, H	-28.957	0.751	-30.381	-27.528
D	-19.291	0.764	-20.852	-17.832
D, H	-23.838	0.427	-24.671	-23.000
D, S	-47.745	3.633	-55.019	-40.486
D, S, H	-50.400	1.196	-52.787	-48.019
I	-5.818	0.518	-6.802	-4.836
I, H	-11.837	0.349	-12.528	-11.126
I, S	-22.623	1.754	-26.193	-19.302
I, S, H	-33.503	0.670	-34.803	-32.219
I, D	-25.042	1.124	-27.204	-22.900
I, D, H	-29.753	0.435	-30.618	-28.902
I, D, S	-45.910	3.941	-53.422	-38.189
I, D, S, H	-52.484	0.866	-54.123	-50.831
C	-52.963	1.239	-55.348	-50.486
C, H	-47.658	0.638	-48.969	-46.447
C, S	-57.695	4.542	-66.941	-48.908
C, S, H	-65.111	1.730	-68.518	-61.841
C, D	-72.426	2.742	-77.685	-67.065
C, D, H	-68.047	0.820	-69.715	-66.496
C, D, S	-62.014	10.448	-82.033	-42.348
C, D, S, H	-87.598	2.244	-92.094	-83.143
C, I	-56.837	1.047	-58.922	-54.797
C, I, H	-54.559	0.427	-55.408	-53.727
C, I, S	-66.272	3.382	-73.165	-59.906
C, I, S, H	-71.862	0.785	-73.561	-70.408
C, I, D	-75.280	2.021	-79.305	-71.223
C, I, D, H	-78.345	0.482	-79.266	-77.397
C, I, D, S	-97.338	5.907	-110.268	-87.213
C, I, D, S, H	-96.245	0.797	-97.849	-94.700
Age				
76	-2.633	0.776	-4.151	-1.102
77	-6.820	0.792	-8.380	-5.270
78	-11.029	0.779	-12.686	-9.478
79	-15.091	0.766	-16.645	-13.668
80	-19.029	0.754	-20.601	-17.638

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: SURVIVAL TO 2013

	Probit Coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
81	-25.744	0.790	-27.362	-24.193
82	-32.994	0.763	-34.493	-31.399
83	-37.152	0.756	-38.708	-35.701
84	-43.227	0.784	-44.712	-41.627
85	-50.962	0.730	-52.360	-49.544
86	-58.346	0.770	-60.015	-56.975
87	-66.066	0.781	-67.605	-64.586
88	-73.374	0.759	-74.904	-71.913
89	-82.584	0.789	-84.114	-81.144
90	-92.259	0.795	-93.799	-90.766
91	-100.974	0.809	-102.579	-99.327
92	-110.871	0.834	-112.514	-109.270
93	-121.799	0.863	-123.583	-120.135
94	-133.690	0.939	-135.686	-132.011
95	-143.847	0.947	-145.848	-142.181
96	-156.420	1.045	-158.544	-154.518
97	-169.733	1.146	-172.152	-167.548
98	-182.185	1.238	-184.734	-179.840
99	-191.265	1.436	-194.146	-188.390
100 and over	-225.657	1.018	-227.855	-223.850
Age x male				
76	0.830	1.108	-1.328	3.002
77	0.871	1.091	-1.306	2.940
78	-0.136	1.125	-2.284	2.121
79	0.691	1.095	-1.434	2.793
80	-0.947	1.059	-2.916	1.186
81	0.039	1.102	-2.117	2.225
82	-0.429	1.085	-2.626	1.711
83	-1.472	1.090	-3.566	0.819
84	-2.349	1.113	-4.607	-0.168
85	-4.045	1.099	-6.246	-1.902
86	-3.013	1.099	-5.028	-0.768
87	-3.429	1.140	-5.709	-1.273
88	-5.611	1.147	-8.072	-3.388

TABLE I3 (CONT'D): RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: SURVIVAL TO 2013

	Probit Coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
89	-5.321	1.163	-7.539	-3.093
90	-5.190	1.213	-7.737	-3.006
91	-5.049	1.277	-7.512	-2.527
92	-6.132	1.246	-8.640	-3.753
93	-6.286	1.356	-9.039	-3.686
94	-8.466	1.485	-11.408	-5.666
95	-9.352	1.628	-12.556	-5.974
96	-8.992	1.793	-12.556	-5.479
97	-11.002	2.102	-15.096	-6.846
98	-7.557	2.363	-12.305	-3.011
99	-17.041	2.774	-22.431	-11.889
100 and over	-6.048	2.106	-10.139	-2.070
2004 Census Block Group Demographics				
median household income / 1000	0.031	0.007	0.017	0.045
per capita income / 1000	-0.018	0.012	-0.042	0.004
median year built	0.080	0.007	0.067	0.093
median house value / 1000	0.017	0.001	0.015	0.019
average house value / 1000	0.001	0.000	0.000	0.002
median gross rent / 1000	-0.036	0.021	-0.081	0.005
% over 65	26.877	0.955	24.996	28.678
% white	-5.126	1.139	-7.352	-2.848
% black	-5.640	1.218	-8.105	-3.300
% hispanic	1.797	1.271	-0.548	4.342
% 9th through 12th	-19.179	2.833	-24.631	-13.462
% high school graduate	1.457	2.230	-2.992	6.093
% some college	12.496	2.225	8.134	16.981
% associate degree	17.347	2.851	11.810	22.960
% bachelor's degree	24.928	2.274	20.438	29.347
% graduate degree	32.224	2.454	27.359	36.972
% owner occupied	11.978	1.065	9.867	14.005
% renter occupied	0.151	1.163	-2.168	2.296

5. *Association between Dementia and its Clinical Risk Factors*

Table I4 shows that the estimated AME of a 1 $\mu g/m^3$ increase in decadal $PM_{2.5}$ in Column (5) of Table I is about three times as large as the estimated increase in dementia risk associated with having been diagnosed with hypertension at the beginning of the decade but not diagnosed with any of the other health risk factors (0.8 pp) using the same decadal model. Our estimate is similar in size to the increase in risk associated with a pre-existing diagnosis of ischemic heart disease alone. Our estimate is smaller than the risks associated with pre-existing diagnoses of the other chronic conditions individually, which range from a 4.1 pp increase for ischemic heart disease alone to an 8.5 pp increase for stroke alone. We estimate that someone diagnosed with all five risk factors by 2004 had a 27.6 pp higher probability of being diagnosed with dementia by the end of 2013, all else equal. Aging provides another opportunity for comparison. All else constant, aging from 75 to 80 is associated with an AME of 5.8 pp and aging from 75 to 85 is associated with an AME of 15.5 pp.

TABLE I4—COMPARING RELATIVE RISKS FOR $PM_{2.5}$ AND OTHER RISK FACTORS

Risk Factor	Percentage point increase in dementia diagnosis probability	95% confidence interval	
hypertension in 2004	0.8	0.6	1.1
ischemic heart disease in 2004	2.0	1.6	2.3
decadal $PM_{2.5}$ (1 μg/m3)	2.4	1.3	3.5
diabetes in 2004	4.1	3.4	4.6
aging from 75 to 80	5.8	5.5	6.1
congestive heart failure in 2004	7.5	6.1	8.9
stroke in 2004	8.5	7.4	9.7
aging from 75 to 85	15.5	14.9	16.0
All five chronic conditions in 2004	27.6	25.4	29.4

Note: The table reports average marginal effects and 95% confidence intervals for dementia risk factors based on the model in Table I, Column (5). Model coefficients are reported in Table J4.

6. *Year-specific average marginal effects from Equations (6) and (7)*

Table I5 reports the year specific AMEs from Equations (6) and (7) in the main text, as referenced in Section V.B.

TABLE I5—AVERAGE MARGINAL EFFECT OF CUMULATIVE $PM_{2.5}$ SINCE 2004 ON THE PROBABILITY OF A NEW DEMENTIA DIAGNOSIS WITHIN THE YEAR

	2005	2006	2007	2008	2009	2010	2011	2012	2013
Probit model average marginal effect	0.063	0.630	-0.140	0.309	0.285	0.430	0.504*	1.07**	0.427
(1 μg/m^3 increase in decadal $PM_{2.5}$)	(0.430)	(0.417)	(0.322)	(0.272)	(0.247)	(0.324)	(0.280)	(0.315)	(0.306)
F-statistic on instruments for $PM_{2.5}$	164.6	236.6	255.9	309.7	349.9	438.6	462.0	489.0	488.9
number of individuals: dementia function	2,293,270	2,051,489	1,844,045	1,650,175	1,490,142	1,362,545	1,236,493	1,109,628	989,751
Chi-square statistic on instruments for survival	1166.0	1337.8	1717.5	1953.6	2204.5	2228.5	2274.3	2244.8	2153.3
number of individuals: survival function	2,439,904	2,439,904	2,439,904	2,439,904	2,439,904	2,439,904	2,439,904	2,439,904	2,439,904
share of survivors diagnosed with dementia	2.9	3.0	3.2	3.4	3.7	3.8	4.0	4.3	4.4

Note: The outcome is scaled to equal 100 if an individual was diagnosed with dementia during the year and 0 otherwise. Asterisks indicate statistical significance at the 10% (*) and 5% (**) levels using robust standard errors clustered at the block group. Standard errors are bootstrapped using 1,000 repetitions.

J. Sensitivity analyses referenced in Section VI

Table II in the main text present tests of model validation for our main specification (Column (6) of Table I). In this section we present results from a series of specifications that include alternative measures of pollution, alternative measures of dementia, varying the sample based on distance from the nearest air-quality monitor, and alternative outcomes.

Estimates from our decadal model (Column (5) of Table I) show that a 1 μg/m^3 increase in average decadal $PM_{2.5}$ increases the probability of a new dementia diagnosis by an average of 2.38 pp. This specification is comparable to the existing economic literature on the impacts of pollution exposure on health outcomes, and it is parsimonious compared to the more computationally intensive, year-specific model in Column (6) of Table I that yields an aggregated AME of 2.15 pp. The similarity in results between the two models suggests that, in our setting, the decadal model is not substantially biased by aggregating exposure over the decade. For these reasons, the following tests are performed using the decadal model described by Equations (2)-(5) in the main text.

1. *Sensitivity Analysis: Alternative Measures of Pollution Exposure*

Table J1 reports the sensitivity of the decadal specification shown in Column (5) of Table I to replacing our preferred measure of air pollution exposure with five alternatives. For convenience, Column (1) repeats Column (5) of Table I. In Column (2) we utilize within-county variation in

monitor readings, similar to Bento, Freedman, and Lang (2015). Specifically, we replace the CBSA dummy variables with county dummy variables, and we stratify the nonattainment indicator according to whether the average $PM_{2.5}$ concentration from 2001 to 2003 at the air quality monitor closest to an individual's residence exceeded the federal standard. This generates three indicators that vary across individuals within counties: (i) nonattainment county with the individual's nearest monitor exceeding the standard, (ii) nonattainment county without the individual's nearest monitor exceeding the standard, and (iii) attainment county with the individual's nearest monitor exceeding the standard. Each indicator is interacted with the fourth-order polynomial function of baseline exposure.

TABLE J1—AVERAGE MARGINAL EFFECTS USING ALTERNATIVE MEASURES OF $PM_{2.5}$ EXPOSURE

	(1)	(2)	(3)	(4)	(5)	(6)
1 $\mu g/m^3$ increase in decadal $PM_{2.5}$	2.384***	1.685***	1.738***	1.163***	2.524***	2.380***
	(0.568)	(0.479)	(0.548)	(0.400)	(0.580)	(0.571)
1 $\mu g/m^3$ increase in decadal PM_{10}			0.035			
			(0.038)			
1 ppb increase in decadal O_3			-0.055			
			(0.046)			
1 ppb increase in decadal NO_2			-0.083			
			(0.050)			
1 ppb increase in decadal SO_2			0.182			
			(0.182)			
1 ppm increase in decadal CO			0.727			
			(2.024)			
<u>modification to main specification</u>						
IV = county x monitor attainment		x				
control for other air pollutants			x			
unbalanced monitor panel				x		
5-digit ZIP assignment of $PM_{2.5}$					x	
spline function of baseline $PM_{2.5}$						x
F-statistic on instruments for $PM_{2.5}$	498	399	345	759	521	119
number of individuals: dementia function	1,179,094	1,179,094	1,179,094	1,179,094	1,179,094	1,179,094
Chi-square statistic on instruments for survival	3,813	3,812	3,815	3,812	3,813	3,811
number of individuals: survival function	2,439,904	2,439,904	2,439,904	2,439,904	2,439,904	2,439,904
share of survivors with dementia in 2013	20	20	20	20	20	20

<u>Note</u>: Column (1) repeats Column (5) of Table I. Column (2) modifies this specification by stratifying the nonattainment county instrument according to whether the monitor closest to an individual's residence was in attainment while replacing CBSA dummies with county dummies. Column (3) adds measures of decadal exposure to other federally regulated air pollutants. Column (4) replaces our preferred measure of decadal pollution exposure (based on a balanced panel of continuously operating monitors) with data from an unbalanced panel of all monitors in operation each year. Column (5) measures pollution at the coarser 5-digit ZIP code level. Column (6) replaces the fourth-order polynomial function of baseline pollution exposure with a "spline" function based on dummies for 72 baseline exposure bins, each of which has a width of 0.33 micrograms per cubic meter. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered at the block group. Standard errors are bootstrapped using 1,000 repetitions.

Column (3) adds measures of decadal exposure to five other federally regulated air pollutants with extensive monitoring networks: coarse particulate matter (particulates smaller than 10 microns in diameter), ozone, nitrogen dioxide, sulfur dioxide, and carbon monoxide. Each measure is constructed following the same procedures that we used to construct decadal $PM_{2.5}$.

Column (4) replaces our "balanced monitor panel" measure of exposure with a measure constructed from an unbalanced panel of all monitors in operation each year (between 871 and 1,137 monitors per year). The unbalanced panel may improve efficiency by using all available ground-level information on pollutant concentrations, but it also may introduce additional measurement error, as explained by Muller and Rudd (2017), Grainger, Schreiber, and Chang (2018), and Grainger and Schreiber (2019).

In Column (5), we measure $PM_{2.5}$ at the centroids of individuals' 5-digit ZIP code areas instead of their 9-digit ZIP mail delivery points. This coarser approach recognizes that exposures may occur over larger areas as individuals travel outside their immediate neighborhoods for activities such as shopping and recreation.

Column (6) replaces the fourth-order polynomial function of baseline (2001-2003) residential $PM_{2.5}$ concentrations with a more flexible spline function. We partition neighborhoods into 72 bins by baseline concentrations (in 0.33 $\mu g/m^3$ increments) and add an indicator variable for each bin.

In summary, we find that a variety of different measures for decadal $PM_{2.5}$ exposure reinforce the conclusion that decadal exposure to $PM_{2.5}$ increases the probability of receiving a new dementia diagnosis.

2. Sensitivity Analysis: Medicare Advantage

The claims-based approach to identifying dementia cases has been well validated, with traditional Medicare (TM) claims from 2007-2012 correctly identifying 85 percent of patients diagnosed with dementia by clinician researchers using in-person assessments (Lee et al. 2019, Taylor et al. 2002). The overall dementia rate in our traditional Medicare data for 2012 is 12.8 percentage points, compared with 10.5 percentage points determined by a panel of clinicians using an in-person set of cognitive tests given to 888 individuals age 65 and above in the Health and Retirement Study (HRS) (Hudiomet et al. 2018). The higher cross-sectional rate in the traditional Medicare sample may be due to several factors, including sampling error in the HRS, underdiagnosis in the HRS (Agarwal et al. 2009), non-representativeness of the HRS (Hudiomet et al. 2018), or

selection of healthier individuals out of TM and into Medicare Advantage (MA) during our study period (Newhouse et al. 2016).

We assess whether the use of claims-based diagnosis for the TM sample influences our conclusions by also evaluating whether $PM_{2.5}$ affects the probability that individuals fill a prescription for drugs used to treat the symptoms of Alzheimer's disease. In the CMS data, we observe if and when each individual, including those on MA plans, began taking one of these five drugs: donepezil, galantimine, rivastigmine, memantine, and donepezil and memantine in combination. Beginning in 2006, there were approximately 1.1 million individuals in our sample who had drug coverage through Medicare, and 12% of them initiated one of these medications between 2006 and 2013. Among the TM enrollees for whom we can observe both drug use and dementia diagnoses, we see that 90% of those prescribed these drugs also received a dementia diagnosis by 2013. Figure J1 contrasts the share of people having ever taken an Alzheimer's drug with the share of people having ever been diagnosed with dementia by age and gender.

FIGURE J1: DEMENTIA DIAGNOSIS AND PRESCRIPTION DRUG USE BY AGE AND GENDER IN 2013

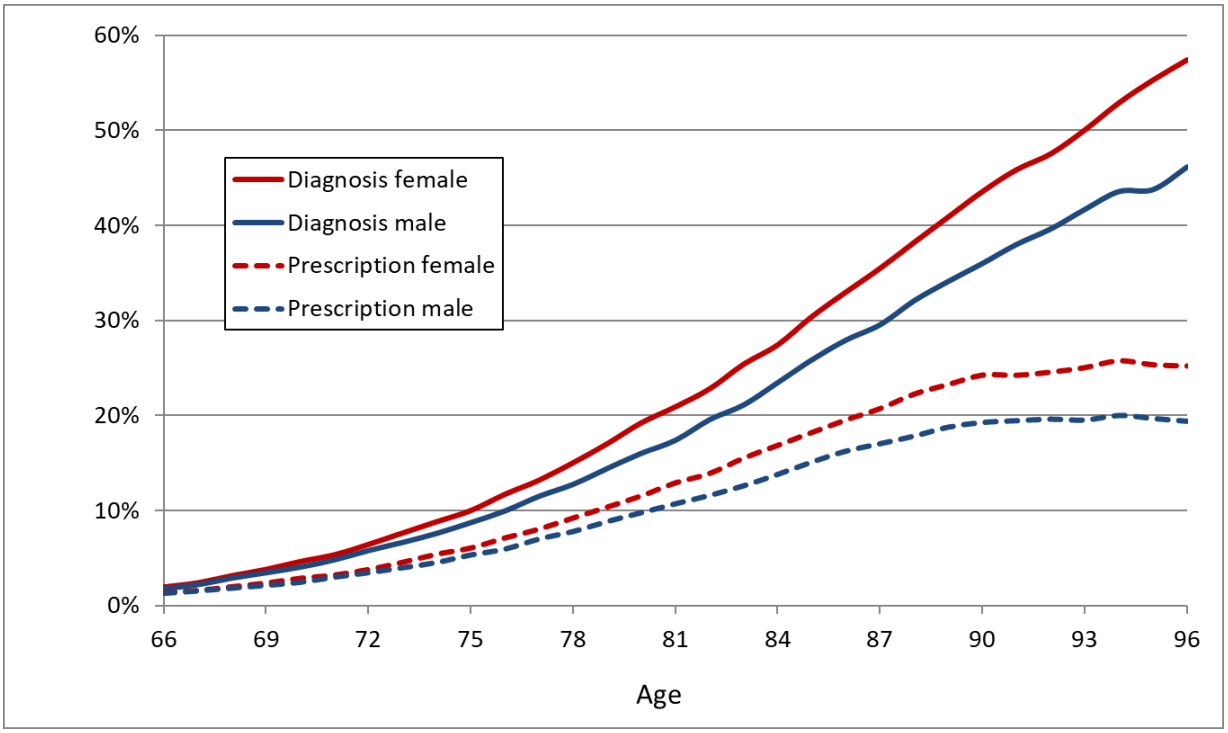


Table J2 contrasts the results shown in Column (5) of Table I that we reported for the TM population with ancillary estimates that include people from the MA population on drug plans. As

a benchmark for comparison, Column (1) repeats the result from Column (5) of Table I. This is based on 2SCML estimation of Equations (2)-(5) in the main text. We use this same specification to estimate models that incorporate the MA population in the following two columns.

Column (2) reports the AME from adding MA enrollees with prescription drug coverage while simultaneously redefining the dementia indicator to be either a claims-based diagnosis at any point from 2005 through 2013 or a claim for a prescription drug to treat symptoms of Alzheimer's disease at any point from 2006 through 2013. This specification expands the sample to include individuals who exited TM at some point after 2004 to enroll in a MA plan that included prescription drug coverage. On net, this expands the sample by 287 thousand individuals (accounting for 94% of the sample who switched to MA and survived through 2013, as shown in Figure A1). The resulting AME is similar to that shown in Column (1).

TABLE J2—AVERAGE MARGINAL EFFECTS INCLUDING MEDICARE ADVANTAGE ENROLLEES

	(1)	(2)	(3)
1 μg/m^3 increase in decadal $PM_{2.5}$	2.384*** (0.568)	2.449*** (0.528)	1.476*** (0.578)
approach to measuring dementia	Diagnoses from Traditional Medicare claims	Diagnoses from Traditional Medicare claims or dementia drugs	dementia drugs
F-statistic on instruments for $PM_{2.5}$	498	562	420
number of individuals: dementia function	1,179,094	1,466,475	1,074,596
Chi-square statistic on instruments for survival	3,813	3,949	3,813
number of individuals: survival function	2,439,904	2,439,904	2,439,904
dependent variable mean	20	19	12

Note: Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered at the block group. Standard errors are bootstrapped using 1,000 repetitions.

Column (3) reports the AME from a model that measures a new dementia diagnosis based solely on whether an individual filled a prescription for an Alzheimer's drug. This specification limits the sample to 1,074,596 people in TM and MA who enrolled in Medicare prescription drug plans. The results indicate that a 1 μg/m^3 increase in average $PM_{2.5}$ over the decade increases the

probability of taking an Alzheimer's drug by 12.3%, slightly larger than the percent effect observed for diagnosis rates.

In summary, the results in Table J2 demonstrate that the results we find for our decadal model are not dependent on either the use of claims-based diagnoses or the exclusion of MA.

3. Sensitivity Analysis: Alzheimer's and Other Dementias

Table J3 reports results from modifying the specification shown in Column (5) of Table I to focus on the specific types of dementia that are directly or indirectly measurable in CMS data. Column (1) repeats the specification shown in Column (5) of Table I for convenience. Columns (2) and (3) repeat estimation of the same specification after stratifying the "Alzheimer's and related dementias" variable from CMS's chronic conditions warehouse file into dementia cases with and without an associated diagnosis of "Alzheimer's disease". The sample size declines slightly in Columns (2) and (3) relative to Column (1) because the Alzheimer's disease variable is missing for a small number of individuals and because changing the outcome measure results in a small number of individuals being dropped during estimation due to lack of within-CBSA variation in the outcome measure. Our estimated AMEs reveal that diagnoses of Alzheimer's disease can account for 77% of the overall dementia AME that our decadal model attributes to long-term $PM_{2.5}$ exposure. A caveat to this interpretation is that it is difficult for doctors to distinguish between Alzheimer's disease and other forms of dementia without an autopsy or extensive brain imaging, leaving some doctors reluctant to diagnose living patients with Alzheimer's disease specifically, as opposed to dementia generally.

TABLE J3—AVERAGE MARGINAL EFFECTS FOR ALZHEIMER'S AND OTHER DEMENTIAS

	(1)	(2)	(3)	(4)
1 μg/m^3 increase in decadal $PM_{2.5}$	2.384***	0.616	1.831***	2.432***
	(0.568)	(0.430)	(0.478)	(0.570)
dependent variable	claim-based diagnosis	claim-based diagnosis without Alzheimer's	claim-based diagnosis with Alzheimer's	claim-based diagnosis with stroke control
F-statistic on instruments for $PM_{2.5}$	498	497	497	498
number of individuals: dementia function	1,179,094	1,178,490	1,178,616	1,179,094
Chi-square statistic on instruments for survival	3,813	3,813	3,813	3,932
number of individuals: survival function	2,439,904	2,439,904	2,439,904	2,439,904
dependent variable mean	20	11	9	20

Note: Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered at the block group. Standard errors are bootstrapped using 1,000 repetitions.

Column (4) reports results from repeating estimation of the model in Column (1) after adding a dummy for whether the individual had a stroke by the end of 2013. Strokes cause vascular dementia, the second most common form of dementia behind Alzheimer's disease, and may be caused by short-term spikes in air pollution. Hence, the stroke variable absorbs any effects of $PM_{2.5}$ on dementia that occur due to observed strokes (although many smaller strokes are clinically unobserved). Our results suggest that the probability of being diagnosed with dementia is 19.1 pp higher for those who had a prior stroke. However, controlling for stroke has virtually no effect on the $PM_{2.5}$ coefficient, as shown in Column (4). This suggests that long-term exposure to $PM_{2.5}$ increases the risk of Alzheimer's disease, specifically. Overall, the results in Table J3 are consistent with the hypothesis that long-term exposure to $PM_{2.5}$ increases the risk of being diagnosed with Alzheimer's disease specifically.

4. Sensitivity Analysis: Distance to Nearest Monitoring Station

Table J4 reports results from modifying the decadal specification shown in Column (5) of Table I to limit the estimation sample to people who never live more than a fixed distance from a $PM_{2.5}$ monitoring station. The results from this specification, with no limit on distance to the nearest monitoring station, is shown in Column (4) for convenience. Column (5) shows results from

repeating estimation after dropping everyone who lived more than 20 km from the nearest monitoring station at any point during our study period. Column (6) shows results from repeating estimation after dropping everyone who lived more than 10 km from the nearest monitoring station at any point during our study period. Our sample size for the dementia function declines by 20% as we move from Column (4) to Column (5), and it declines by 47% as we move from Column (4) to Column (6). However, even as we reduce the size of the estimation sample, the AME for $PM_{2.5}$ remains precisely estimated and similar in magnitude.

TABLE J4—AVERAGE MARGINAL EFFECTS CONDITIONAL ON DISTANCE TO MONITOR

	(1)	(2)	(3)	(4)	(5)	(6)
1 $\mu g/m^3$ increase in decadal $PM_{2.5}$	0.170	0.218*	0.245	2.384***	2.012***	2.031***
	(0.108)	(0.126)	(0.157)	(0.568)	(0.618)	(0.726)
Maximum distance from nearest monitor	no limit	20 km	10 km	no limit	20 km	10 km
$PM_{2.5}$ control function				x	x	x
survival control function	x	x	x	x	x	x
heterogeneity in effects	x	x	x	x	x	x
F-statistic on instruments for $PM_{2.5}$				498	231	115
number of individuals: dementia function	1,179,094	947,797	637,370	1,179,094	947,797	630,370
Chi-square statistic on instruments for survival	3,813	3,230	2,189	3,813	3,230	2,189
number of individuals: survival function	2,439,904	1,963,293	1,339,634	2,439,904	1,963,293	1,339,634
dependent variable mean	19.5	19.4	19.1	19.5	19.4	19.1

Note: Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered at the block group. Standard errors are bootstrapped using 1,000 repetitions.

The first three columns in the table show results from performing similar estimations to those shown in Columns (4), (5), and (6), but without instrumenting for $PM_{2.5}$. Because we reduce the scope for measurement error in $PM_{2.5}$ by reducing the maximum distance to the nearest monitoring station, the AME for $PM_{2.5}$ increases by 28% and 44% respectively.

5. Placebo Outcomes

Table J5 presents results for five placebo diagnoses: glaucoma, fibromyalgia, breast cancer, prostate cancer, and peripheral vascular disease. As explained in the main text, these five chronic conditions that are not known or suspected to be caused by air pollution, but they share similarities with dementia in terms of how they affect the body, how they are diagnosed, and how diagnosis rates are correlated with age, race, and gender. Our placebo models use the same 2SCML estimator from the decadal specification in Column 5 of Table I with one modification: we omit the selection correction for mortality. The reason that we omit the selection correction is that while the medical

literature suggests that our cancer-based instruments for mortality are unrelated to dementia, these same instruments are known or suspected to increase the likelihood of being diagnosed with each of the placebo outcomes. Despite this caveat, the placebo tests are still informative in the sense that our estimate for the AME of $PM_{2.5}$ on dementia remains large and precisely estimated (1.707, p=0.002) when we modify our the specification in Table I, Column (5) to omit the selection correction. This benchmark result is shown in the first column of Table J10.

The placebo model sample sizes in the remaining columns of the table are slightly smaller than the one underlying Column (1). This is because the placebo models parallel our dementia specification in excluding people who had been diagnosed with the placebos by 2004. While the placebo models also add people who had been diagnosed with dementia in 2004, but not the placebos, this addition is more than offset by the prior-diagnosis-with-placebo exclusions because the 10-year survival rate for people with dementia in 2004 is low (16%). None of AMEs on the placebos are statistically distinguishable from zero at the 10% level.

TABLE J5—AVERAGE MARGINAL EFFECTS FOR PLACEBO SPECIFICATIONS

	Dementia	Glaucoma	Fibro-myalgia	Breast cancer	Prostate cancer	Peripheral vascular disease
1 $\mu g/m^3$ increase in decadal $PM_{2.5}$	1.707***	0.424	-0.603	-0.121	-0.121	0.788
	(0.559)	(0.590)	(0.540)	(0.237)	(0.255)	(0.645)
F-statistic on instruments for $PM_{2.5}$	496	448	476	474	472	483
number of individuals: outcome equation	1,179,094	997,106	1,104,693	1,162,565	1,164,689	1,115,560
dependent variable mean	20	18	18	3	4	25

Note: Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered at the block group. Standard errors are bootstrapped using 1,000 repetitions.

K. Appendix references

Agarwal, Sumit, John C. Driscoll, Xavier Gabaix, and David Laibson. 2009. "The Age of Reason: Financial Decisions over the Life Cycle and Implications for Regulation." *Brookings Papers on Economic Activity*. Fall: 51-117.

Bento, Antonio, Matthew Freedman and Corey Lang. 2015. "Who Benefits from Environmental Regulation? Evidence from the Clean Air Act Amendments." *Review of Economics and Statistics*, 97(3): 610-622.

Bhattacharya, Jay, Azeem M. Shaikh, and Edward Vytlacil. 2012. "Treatment Effect Bounds: An

Application to Swan–Ganz Catheterization." *Journal of Econometrics,* 168.2: 223-243.

Block M.L., et al. 2012. "The Outdoor Air Pollution and Brain Health Workshop." *NeuroToxicology,* 33: 972-984.

Blundell, Richard, Amanda Gosling, Hikehiko Ichimura, and Costas Meghir. 2007. "Changes in the Distribution of Male and Female Wages Accounting for Employment Composition Using Bounds. *Econometrica, 75*(2), pp.323-363.

Chay, Kenneth Y. and Michael Greenstone. 2005. "Does Air Quality Matter? Evidence from the Housing Market." *Journal of Political Economy,* 113(2): 376-424.

Di, Qian, Yan Wang, Antonella Zanobetti, Yun Wang, Petros Koutrakis, Christine Choirat, Francesca Dominici, and Joel D. Schwartz. 2017. "Air Pollution and Mortality in the Medicare Population." *The New England Journal of Medicine*.

Fowlie, Meredith, Edward Rubin, and Reed Walker. 2019. "Bringing Satellite-Based Air Quality Estimates Down to Earth," *AEA Papers and Proceedings,* 109: 283-88.

Grainger, Corbett, and Andrew Schreiber. 2019 "Discrimination in Ambient Air Pollution Monitoring?." *AEA Papers and Proceedings,* 109, 277-82.

Grainger, Corbett, Andrew Schreiber and Wonjun Chang. 2018. "Do Regulators Strategically Avoid Pollution Hotspots when Siting Monitors? Evidence from Remote Sensing of Air Pollution". *Working Paper.*

Heckman, James J. and Edward Vytlacil. 1998. "Instrumental Variables Methods for the Correlated Random Coefficient Model: Estimating the Average Rate of Return to Schooling When the Return is Correlated with Schooling." *Journal of Human Resources*, 33(4): 974-987.

Honore, Bo E., and Adriana Lleras-Muney. 2006. "Bounds in Competing Risks Models and the War on Cancer." *Econometrica*, 74(6), 1675-1698.

Horowitz, Joel L., and Charles F. Manski. 2000 "Nonparametric Analysis of Randomized Experiments with Missing Covariate and Outcome Data." *Journal of the American Statistical Association* 95(449), 77-84.

Hudiomet, P, MD Hurd and S Rohwedder. 2018. "Dementia Prevalence in the United States in 2000 and 2012: Estimates Based on a Nationally Representative Study." *Journals of Gerontology: Social Sciences* 73(S1), S10-S19.

Imbens, Guido W., and Charles F. Manski. 2004. "Confidence Intervals for Partially Identified Parameters." *Econometrica* 72.6: 1845-1857.

Isen, Adam, Maya Rossin-Slater and W. Reed Walker. 2017. "Every Breath You Take- Every Dollar You'll Make: The Long-Term Consequences of the Clean Air Act of 1970". *Journal of Political Economy,* 125(3): 849-909.

Manski, Charles F., and John V. Pepper. 2000. "Monotone Instrumental Variables with an Application to the Returns to Schooling." *Econometrica,* 68(4):997-1012.

Muller, Nicholas Z and Paul Rudd. 2017. "What Forces Dictate the Design of Pollution Monitoring

Networks?" *Environmental Modeling and Assessment*, 23(1):1-14.

Newhouse, Joseph P., Mary Price, John Hsu et al. 2015. "How Much Selection Is Left in Medicare Advantage?" *American Journal of Health Economics*, 1(1):1-26.

Schlenker, Wolfram and W. Reed Walker. 2016. "Airports, Air Pollution, and Contemporaneous Health." *Review of Economic Studies*, 83(2): 768-809.

Taylor Jr., Donald H., Gerda G. Fillenbaum and Michael E. Ezell. 2002. "The accuracy of medicare claims data in identifying Alzheimer's disease." *Journal of Clinical Epidemiology*, 55, 929-937.

Underwood, Emily. 2017. "The Polluted Brain". *Science*. 355(6323): 342-345.