HAZED AND CONFUSED:
THE EFFECT OF AIR POLLUTION ON DEMENTIA*

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We track US Medicare beneficiaries' cumulative residential exposures to $PM_{2.5}$ and their cognitive health from 2001 through 2013, leveraging within- and between-county quasi-random variation in $PM_{2.5}$ resulting from the expansion of Clean Air Act regulations. Our main estimates suggest that a 1-$\mu g/m^3$ increase in decadal $PM_{2.5}$ increases the probability of a dementia diagnosis by an average of 1.63-1.84 percentage points. While we find that higher $PM_{2.5}$ also increases the probability of death during the decade, models that control for this selection show that $PM_{2.5}$'s effects on dementia cannot be explained by selecting on survival or by Tiebout-sorting dynamics. We do not find relationships between decadal $PM_{2.5}$ and placebo outcomes nor between other air pollutants and dementia. Our estimates suggest that federal regulation of $PM_{2.5}$ led to 182,000 fewer people with dementia in 2013, yielding $214 billion in benefits. Further, $PM_{2.5}$'s effect on dementia persists below the current regulatory thresholds.

March 2020

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Alzheimer's disease and related forms of dementia degrade human capital, increase medical spending, and reduce the quantity and quality of life. Dementia is the fifth leading cause of death worldwide.[1] In the US alone, 5.5 million dementia patients spent $234 billion on health care services in 2019, with an additional 18.5 billion labor hours by unpaid caregivers (Alzheimer's Association 2019). The precise causes of dementia remain unknown. However, recent medical research raises suspicion that long-term exposure to fine-particulate air pollution smaller than 2.5 microns in diameter ($PM_{2.5}$) may contribute to dementia (Peters et al. 2019, Underwood 2017, Block et al. 2012). Observational studies reinforce this suspicion. For example, Zhang et al. (2018) and Carey et al. (2018) found that long-term exposure to $PM_{2.5}$ is associated with decreased cognitive performance for adults in China and increased rates of dementia for adults in London, respectively. However, these associations may not be causal. Economic research on residential sorting has shown that air pollution triggers some people to move (Banzhaf and Walsh 2008, Cheng, Oliva, and Zhang 2017) and, conditional on moving, people sort themselves across neighborhoods based on their incomes and preferences for air quality and other public goods (Sieg et al. 2004, Bayer, Ferreira and McMillan 2007, Bayer, Keohane and Timmins 2009, Kahn and Walsh 2015, Bayer et al. 2016). This Tiebout sorting could generate correlation between $PM_{2.5}$ and dementia if people who are at a greater risk of developing dementia sort themselves into relatively polluted areas.

This paper is the first nationwide, individual-level study of whether long-term exposure to $PM_{2.5}$ has a causal effect on dementia. We use administrative records from the U.S. Medicare program to develop a longitudinal research design that

[1] The World Health Organization's 10th revision of the International Statistical Classification of Diseases and Related Health Problems defines dementia (codes F00-F03) as "a syndrome due to disease of the brain, usually of a chronic or progressive nature, in which there is disturbance of multiple higher cortical functions, including memory, thinking, orientation, comprehension, calculation, learning capacity, language and judgement. Consciousness is not clouded. The impairments of cognitive function are commonly accompanied, and occasionally preceded, by deterioration in emotional control, social behavior, or motivation. This syndrome occurs in Alzheimer disease, in cerebrovascular disease, and in other conditions primarily or secondarily affecting the brain." (WHO 2011) Below we define Alzheimer's disease specifically, which accounts for 60% to 80% of all dementia cases. Mortality data are from: https://www.who.int/news-room/fact-sheets/detail/the-top-10-causes-of-death.

comprehensively addresses residential sorting. First, we assemble ten years of data on a random sample of millions of Americans age 65 and above to track their diagnosis dates for many illnesses including Alzheimer's disease and related dementias, their use of prescription drugs for symptoms of Alzheimer's disease, their demographics, and their sequence of residential addresses from 2001 through 2013. Then we combine individuals' location histories with $PM_{2.5}$ data from a balanced panel of the Environmental Protection Agency (EPA) air quality monitors to measure long-term $PM_{2.5}$ exposure at the individual level, accounting for migration.

Like the prior observational studies, the data we use show strong, positive relationships between the prevalence of dementia and the average concentration of $PM_{2.5}$ over a decade. Figure A1 illustrates this association by plotting state-level dementia rates among 75, 80, 85, and 90-year-old individuals in 2013 against their average residential $PM_{2.5}$ exposures from 2004 through 2013. Correlation coefficients range from 0.47 to 0.66.

We investigate whether these associations are causal or are spurious due to residential sorting, sample selection, errors in measuring pollution exposure, or other unspecified threats to identification. Our research design leverages quasi-random variation in $PM_{2.5}$ resulting from the EPA's expansion of Clean Air Act regulations. In 2004 the EPA began to enforce a maximum threshold on $PM_{2.5}$, prompting local regulators to clean up polluted areas. The subsequent reductions in emissions created variation in individuals' $PM_{2.5}$ exposures from 2004-2013 conditional on their demographics, pre-regulatory health, and pre-regulatory pollution exposures and other geographic factors. We use this variation to identify how $PM_{2.5}$ exposure from 2004-2013 affected the probability of being diagnosed with dementia during this period among those who did not have dementia in 2004. Our two-stage least-squares (2SLS) models flexibly control for individual characteristics associated with dementia risk, including race, gender-by-integer-age interactions, medical expenditures, fully interacted sets of baseline medical conditions, the socioeconomic

composition of people's baseline neighborhoods, and the pre-regulatory pollution levels of those neighborhoods. Further, we include core-based statistical area (CBSA) fixed effects to absorb spatial variation in diagnostic standards, health care quality and access, and latent environmental quality.

Under our research design, the effect of interest is identified by quasi-random variation in decadal exposure to $PM_{2.5}$ experienced by people of the same age, race, and gender who lived in the same CBSA and who, at the start of the decade, had received the same medical diagnoses for dementia risk factors, had the same level of gross annual medical expenditures, and had sorted themselves into neighborhoods with the same baseline levels of $PM_{2.5}$ and with the same distributions of race, income, educational attainment, and property values. Conditional on these characteristics, our models are identified by three sources of residual variation in $PM_{2.5}$ that prior studies have used to analyze air pollution's effects on housing prices and residential sorting. First, like Chay and Greenstone (2005), we use information on how strengthened EPA regulations affected some counties more than others. Second, like Bento, Freeman, and Lang (2015), we use within-county variation in the effects of these regulations. Third, like Banzhaf and Walsh (2008), we observe changes in exposure among people who moved after the regulations were enforced.

A two-stage linear probability model shows that a 1 $\mu g/m^3$ increase in average residential concentrations of $PM_{2.5}$ over a decade (9.1% of the mean) increases the probability of receiving a dementia diagnosis by 1.68 percentage points (pp) (7.5% of the mean) among those who survived the decade. To put theses estimate in context, the elevated risk of dementia due to a 1 $\mu g/m^3$ increase in decadal $PM_{2.5}$ is approximately twice as large as the elevated risk conditionally associated with having been previously diagnosed with hypertension and half of the elevated risk conditionally associated with having been previously diagnosed with diabetes. A flexible control-function probit model yields an average marginal effect of 1.84 pp.

This model implies that the marginal effects rise as the level of exposure falls, indicating that further reductions in $PM_{2.5}$ would yield even larger marginal reductions in dementia than we estimate for our study period.

Consistent with prior work on the short-term effects of $PM_{2.5}$ (e.g., Deryugina et al. 2019), we find that long-term exposure to $PM_{2.5}$ causes mortality. Specifically, our two-stage linear model shows that a 1 $\mu g/m^3$ increase in decadal $PM_{2.5}$ elevates the decadal mortality risk by 2.37 pp. Because our main estimation sample is limited to people who survived through 2013, our estimates for the effect of $PM_{2.5}$ on dementia could diverge from the population-wide effect if unobserved health that affects survival is correlated with unobserved health that affects dementia. We implement a two-pronged approach to evaluate whether sample selection can explain our observed effects of $PM_{2.5}$ on dementia. First, we employ a partial-identification approach (Manski 1990, Lee 2009) that makes no assumptions on the correlation between survival and the latent health affecting dementia and show that our main results cannot be explained by selection on mortality. Second, we extend our 2SLS model to incorporate a control-function procedure (Heckman and Robb 1986) where we first estimate the probability of survival using additional instruments constructed from data on individuals' diagnoses of cancers that, based on medical literature, are unrelated to dementia. This procedure increases the estimated effect of a 1 $\mu g/m^3$ increase in decadal $PM_{2.5}$ on the dementia diagnosis probability to 2.33 pp. This increase is consistent with the hypothesis that people with lower latent health are both less likely to survive the decade and more likely to develop dementia if they were to survive. We additionally estimate a correlated random coefficient model (Garen 1984) to simultaneously address selection and sorting on latent heterogeneity in sensitivity to $PM_{2.5}$ within the selection-corrected 2SLS model. We find that this model yields estimates almost identical to our preceding selection-corrected models, and we cannot reject the hypothesis of no sorting or selection on $PM_{2.5}$ sensitivities.

Our results persist across a wide range of alternative modeling decisions. The effect of decadal $PM_{2.5}$ on dementia persists when we modify our main specification to use (1) different measures of dementia such as the use of prescription drugs for the symptoms of Alzheimer's disease rather than claims-based diagnosis codes; (2) different samples that include people who select into managed care plans known as Medicare Advantage; (3) monitor-level instruments rather than county-level instruments; (4) different approaches to measuring $PM_{2.5}$ exposure including expanding the set of monitors to include those not present for the entire study period, (5) controls for baseline pollution exposure even more flexible than the fourth-order polynomial function in our main models, and (6) a control that accounts for the possibility that receiving a dementia diagnosis affects people's subsequent decisions about where to live.

Additional analysis supports the validity of using our research design to draw causal inference about the effects of $PM_{2.5}$ on dementia specifically. First, we estimate the same 2SLS model for other chronic illnesses thought *a priori* to be unrelated to $PM_{2.5}$, but that share similarities with dementia in terms of symptoms, diagnostic difficulty, and how diagnosis rates are correlated with age, race, and gender. These placebo tests yield point estimates that are small and statistically indistinguishable from zero at conventional levels. These null effects contrast with our "reverse placebo" finding that long-term exposure to $PM_{2.5}$ also causes mortality, consistent with prior work on short-term exposure. Second, we repeat the estimation using having dementia in 2004 as the outcome. The point estimate is negative, small in absolute value, and statistically indistinguishable from zero. This suggests that our model is unlikely to be confounded by anticipatory Tiebout sorting into more or less polluted areas based on unobserved factors that contribute to differences in dementia diagnoses.

Additional specifications provide further evidence that the higher probability of dementia is due to long-term exposure to $PM_{2.5}$ specifically. First, expanding the

set of pollutants in the model to include ozone, nitrogen dioxide, sulfur dioxide, and carbon monoxide confirms that $PM_{2.5}$ affects dementia, and the estimated effect of $PM_{2.5}$ is unchanged by conditioning on these other pollutants. Second, we find that the effects are driven entirely by long-term exposure. The estimated effects of $PM_{2.5}$ increase as we lengthen the measured exposure duration and become statistically significant at eight years and beyond. Related, we show that our results are not explained by observed strokes that lead to vascular dementia, suggesting that the effects are due to Alzheimer's disease specifically.

These findings indicate that air pollution's effects on dementia make its detriments to health and human capital substantially larger than previously realized. Incorporating these effects will be important for comprehensively evaluating the ongoing efforts to improve air quality worldwide. These include recent efforts to reduce vehicle emissions in China (Li 2017) and industrial emissions in the U.S. (Blundell, Gowrisankaran, and Langer 2018) via the Clean Air Act regulations even beyond the specific one that we consider in this paper. We find that the EPA's expansion of the Clean Air Act to target $PM_{2.5}$ specifically led to improvements in newly regulated areas that averted approximately 182,000 cases of dementia in 2013 among people age 75 and above, yielding $214 billion in benefits. Finally, we find that $PM_{2.5}$'s effect on dementia persists at levels below the EPA's current regulatory threshold, implying that further improvements in air quality would reduce dementia rates.

I. Related Literature

A. Economic research on air pollution, human capital, and Tiebout sorting

Economic research has shown that particulate matter emitted by the transportation, manufacturing, and energy sectors increases mortality. This finding persists around the world and over time, even as pollution has declined and medical technology has improved—from the historically high exposures in London in the 1960s

(McMillan and Murphy 2017) and China in the 2000s (Li et al. 2019) to the historically low exposures in the U.S. in the 2000s (Deryugina et al. 2019). Economic research has also shown that air pollution constrains both the production and productivity of human capital.[2] For instance, daily pollution spikes have been found to increase school absences and reduce students' scores on high stakes exams (Currie et al. 2009, Ebenstein, Lavy, and Roth 2016). Among working age adults, daily pollution spikes have been found to reduce productivity in both manual and cognitive tasks (Chang et al. 2016, Archsmith, Heyes, and Saberian 2017). In contrast, prior studies have not considered whether pollution degrades human capital late in life apart from mortality. While prior studies have shown that cognitive decline impairs older adults' financial decisions, reduces their welfare, and affects market functioning (Agarwal et al. 2009, Keane and Thorpe 2016) our study is the first economic research to investigate whether air pollution plays a role.

Residential sorting poses a difficult econometric challenge for any study of long-term pollution exposure (Kahn and Walsh 2015). The Tiebout sorting literature has shown that heterogeneity in wealth and preferences plays a leading role in determining whether individuals choose to pay housing price premia to live in neighborhoods with better air quality and correlated amenities (e.g., Bayer, Ferreira and McMillan 2007, Banzhaf and Walsh 2008, Bayer, Keohane and Timmins 2009, Bayer et al. 2016, Lee and Lin 2018). This creates a potentially complex endogeneity problem because factors determining individual pollution exposure (e.g., wealth and preferences) may themselves be partially determined by latent aspects of health that affect dementia risk. In addition to leveraging our data and applying methods to overcome this problem, we contribute to the sorting literature by providing the first empirical analysis of long-term pollution exposure that accounts for individual migration.

[2] See Graff-Zivin and Neidell (2013) for a systematic literature review.

A specific concern that our estimation strategy is designed to deal with is the possibility that individuals sort on unobserved risk factors such as genetics. For this to confound our estimates, individuals of the same age, race, sex and baseline health would have to be sorting on the basis of genetics into attainment versus non-attainment counties (or monitors, in some of our specifications) within the same CBSA and with the same baseline pollution levels and neighborhood sociodemographic factors. In addition to our methodological approach, prior research that observed individuals' genetics indicates that sorting into different levels of $PM_{2.5}$ is not associated with the relevant genetic factors known as Apolipoprotein E (APOE). Specifically, Cacciottolo et al.'s (2017) study of nearly 4,000 elderly women in the US finds that individuals' residential exposures to $PM_{2.5}$ do not differ by APOE genotypes. Similarly, Shin, Lillard, and Bhattacharya (2019) find "no correlation between Alzheimer's Disease polygenic risk score and net worth, housing assets and nonfinancial assets" indicating that dementia-related genetics are not associated with sorting into neighborhoods based on economic status.

B. Medical links between air pollution and dementia

Medical and epidemiological research provides reason to suspect that long-term exposure to $PM_{2.5}$ may permanently impair older adults' cognition via dementia (Peters et al. 2019). Compared with other air pollutants, $PM_{2.5}$'s relatively small size allows it to remain airborne for long periods, to penetrate buildings, and to be inhaled easily. Research proposes multiple pathways by which $PM_{2.5}$ may cause dementia. First, $PM_{2.5}$ accumulates in brain tissue (Maher et al. 2016) and causes neuroinflammation, which is associated with symptoms of dementia (Underwood 2017). Individuals living in polluted areas for long periods have been found to have elevated concentrations of $PM_{2.5}$ in their brains, smaller brain volume, and higher rates of brain infarcts or areas of necrosis (Wilker et al. 2015). Second, pollution is linked to increased risk for strokes and subsequent vascular dementia (Wellenius et

al. 2012). Third, exposure of mice to particulates in laboratory experiments results in neuroinflammation and patterns of brain cell damage similar to postmortem analysis of Alzheimer's patients (Block et al. 2012). Fourth, $PM_{2.5}$ has been associated with subclinical measures of cognitive impairment (Power et al 2016) such as laboratory tests, with the strongest associations among individuals over age 65 (Zhang et al. 2018). Finally, $PM_{2.5}$ has been found to increase mortality from cardiovascular conditions (Pope et al. 2002, Landen et al. 2006) that are associated with a higher risk of dementia (Alzheimer's Association 2018). Some studies found associations between dementia and other pollutants, including carbon monoxide, nitrogen oxides and ozone, but the research was not designed to disentangle the contributions of covarying pollutants (Peters et al. 2019).

While suggestive, the current evidence directly linking $PM_{2.5}$ to dementia is based on non-human mammal studies and specialized human cohorts, such as individuals who chose to live near major roadways (e.g., Chen et al. 2017).[3] One such specialized cohort are the elderly US women in the Women's Health Initiative Memory Study studied by Cacciottolo et al (2017). They find that the association between residential $PM_{2.5}$ exposure and dementia are strongest for women with specific APOE genotypes. While genetics may contribute to heterogeneity in individuals' dose-response function for $PM_{2.5}$ and dementia, genes' influence remains probabilistic rather than deterministic, such that "[h]aving the genetic variant associated with an increased risk of late-onset dementia is neither necessary nor sufficient for onset" (Giustinelli, Manski and Molinari 2019).

II. Variation in Long-Term $PM_{2.5}$ Exposure Due to the Clean Air Act

We analyze how decadal exposure to air pollution affects the probability of new

[3] An exception is Carey et al. (2018) which tracked 130,000 older adults in London over a nine-year period and found their likelihood of a dementia diagnosis to be positively correlated with their neighborhood's baseline $PM_{2.5}$ and NO_2 but not ozone. However, that study did not address potential confounding from residential sorting.

dementia diagnoses using within-county and between-county, quasi-random variation in pollution exposure resulting from Clean Air Act (CAA) regulations. The CAA established national standards for maximum-allowable concentrations of air pollutants. Counties containing monitors that violate the standards are designated as being "nonattainment" by the EPA. States are then responsible for developing implementation plans that coordinate local regulatory actions to ensure that nonattainment counties reduce concentrations around pollution "hot spots" enough to meet the standards. States that fail to bring their counties into attainment risk losing federal highway funds and may face additional penalties.

Among the regulated pollutants, particulate matter is believed to have the most pernicious effects on human health (US EPA 2011). Beginning in 1971, the EPA regulated total suspended particulates (TSP). In light of evidence that health effects were driven by the smallest particulates, the EPA replaced the TSP standard with a standard on PM_{10} in 1987 and a standard on $PM_{2.5}$ in 1997. Each new standard was followed by new nonattainment designations.[4] These designations caused the regulated counties to have relatively large reductions in particulates. Further, the sizes of these reductions varied within counties due to local targeting of hot spots and geographic factors that determine particulate dispersion. Because a county's nonattainment status was determined by its "dirtiest" monitor, local regulators took actions that led to the largest pollution reductions around monitors that exceeded the standard or were close to doing so (Auffhammer, Bento, and Lowe 2009).

Prior research has leveraged similar policy changes to evaluate air pollution's effects by assuming that individuals' decisions about where to live prior to these policies did not incorporate anticipation of these regulatory changes and their neighborhood-specific effects on air pollution. Chay and Greenstone (2005) and Isen, Rossin-Slater, and Walker (2017) use county nonattainment for TSP as an

[4] See Kahn (1997) for a review of these policies.

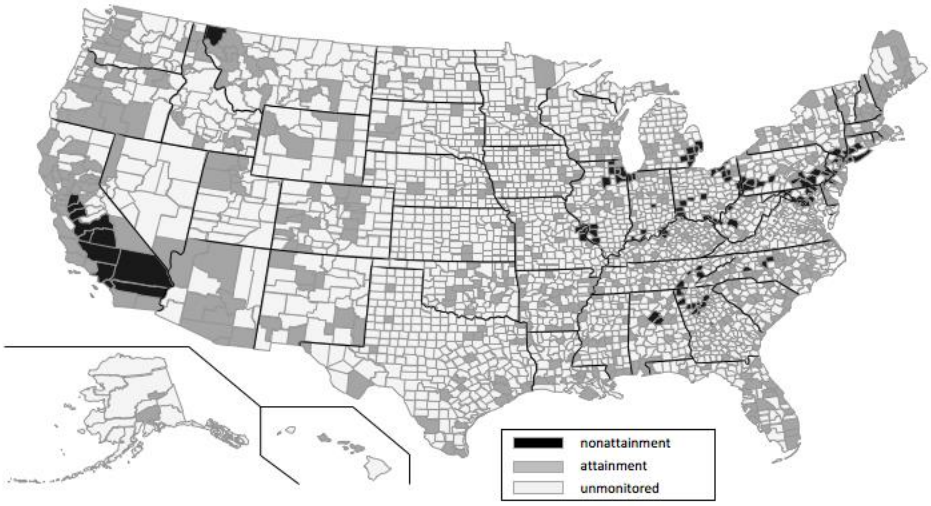
instrument for subsequent changes in county-level TSP concentrations, while Bento, Freedman, and Lang (2015) develop instruments based on within-county variation in monitor-level nonattainment for PM_{10}. In this paper, we exploit the EPA's initial nonattainment designations for $PM_{2.5}$ to develop county-level and monitor-level instruments for decadal $PM_{2.5}$.

In 1997, the EPA established initial monitoring protocols for $PM_{2.5}$ and set the maximum-allowable annual average concentration at 15.05 $\mu g/m^3$. By 1999, a national network of more than 900 air quality monitors was put into place. Several litigants challenged the new $PM_{2.5}$ standard, but it was ultimately upheld by the U.S. Supreme Court and litigation ended in 2002. In April 2003, the EPA asked state and local regulators to provide their three most recent calendar years of $PM_{2.5}$ monitor data and to self-report any nonattainment areas to the EPA by February 2004. The same memo explained how the EPA would use this information to finalize nonattainment designations and outlined procedures and deadlines for becoming compliant. In January 2005, the EPA issued final nonattainment designations using monitor data from 2001-2003.[5]

Figure I shows the locations of attainment and nonattainment counties with air quality monitors. At that time, 132 of the monitored counties containing approximately 27% of the US population were classified as nonattainment. Another 528 counties containing 43% of the US population were classified as attainment. The remaining counties lacked monitoring data and were designated "unclassifiable" and not subjected to additional regulation (US EPA 2005). States were directed to ensure that nonattainment counties met the 15.05 $\mu g/m^3$ standard by 2010.

[5] Nonattainment designations at each monitor were based on an average from 2001-2003 of annual averages over quarterly averages over daily averages over hourly average monitor readings. For counties with multiple monitors, nonattainment designations were based on the monitor with the highest concentration. Details are provided in US EPA (2005).

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



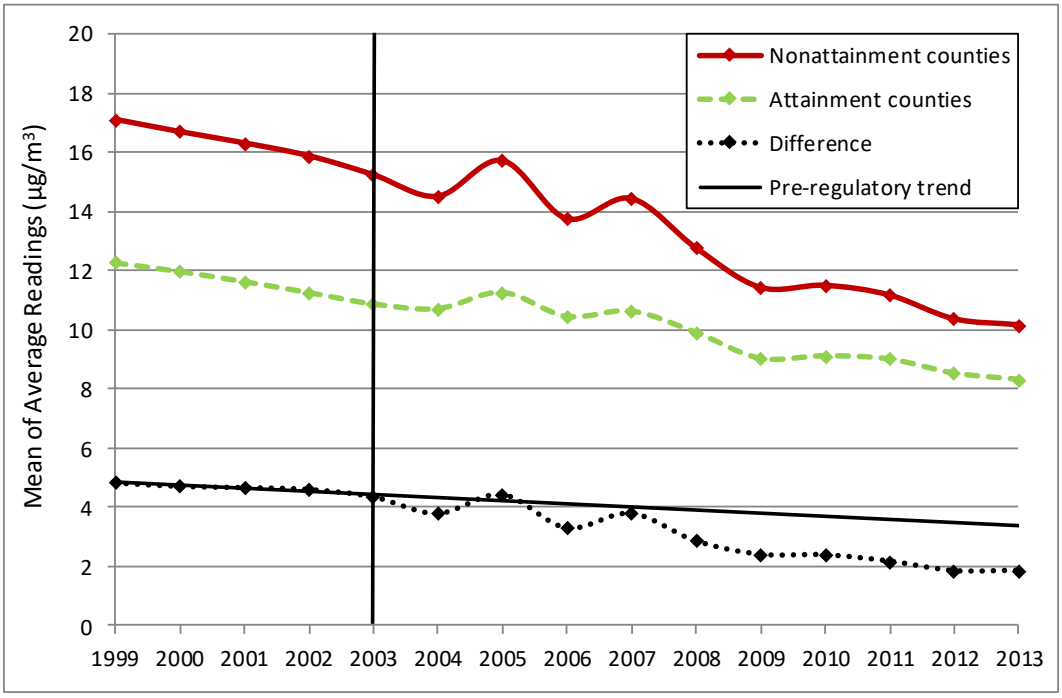
Note: The map shows attainment status in 2005 for US counties that had air quality monitors in place throughout the 2001-2003 evaluation period. There were 132 nonattainment counties located in 21 states and 528 attainment counties in 50 states.

Local regulators' responses to these designations led to quasi-random within- and between-county variation in the change in average $PM_{2.5}$ concentrations over the subsequent decade. Figure II provides initial evidence that nonattainment designations led to greater average $PM_{2.5}$ reductions in newly regulated counties.[6] We define 2004 as the start of the post-regulatory period because local regulators learned which counties were likely to be designated nonattainment at some point between April 2003 (when they received the EPA memo) and February 2004 (when they were required to submit their data). The figure shows that $PM_{2.5}$ concentrations were trending downward similarly in both attainment and nonattainment counties prior to 2004. The dotted line shows that the difference between the two trend lines was fairly stable from 1999 through 2003 with between 4.4 and 4.8 higher $\mu g/m^3$

[6] The figure is based on a balanced panel of 485 $PM_{2.5}$ monitors in operation continuously from 2001-2013. Appendix Figure A2 shows that the figure looks virtually identical if we reconstruct it using an unbalanced panel of all monitors ever in operation from 2001-2013.

in nonattainment counties.[7] After 2003, $PM_{2.5}$ 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 the dotted and solid lines 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.

FIGURE II: 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 dotted line shows the difference between the nonattainment and attainment lines. The pre-regulatory trend line is a projection of the difference from 1999 to 2003 when state and local regulators were notified of the impending nonattainment designations. In 2010 the Census Bureau recorded 41% of the US population age 65 and over living in attainment counties and 27% living in nonattainment counties.

[7] Figure A8 in the appendix shows that difference in annual dementia diagnosis rates between attainment and nonattainment counties was also stable between 1999 and 2003.

Figure II mirrors the analysis that Chay and Greenstone (2005) used to motivate their use of the 1975 nonattainment designations for TSP (see Figure 2 in that paper) as instrumental variables to isolate exogenous between-county variation in TSP changes. We extend their strategy to additionally isolate exogenous within-county variation in $PM_{2.5}$ changes. Specifically, we follow Auffhammer, Bento, and Lowe (2009) in allowing the effects of local regulatory responses to vary with distance from the regulatory threshold. We control for pre-regulatory trends with a flexible function of local $PM_{2.5}$ levels from 2001-2003 and find that, conditional on pre-regulatory levels of $PM_{2.5}$, neighborhoods in nonattainment counties had $PM_{2.5}$ reductions over the following decade larger than neighborhoods in attainment counties, with the size of the difference varying with the distance from the threshold. We further exploit exogenous within-county variation in $PM_{2.5}$ by developing an IV approach in the spirit of Bento, Friedman, and Lang (2005), where we interact county-attainment status with the nearest monitor's attainment status to account explicitly for differential targeting within a county. Section IV-VI formalize these models and report results.

III. Data and Summary Statistics

A. Medicare Data

The U.S. Medicare program provides universal health insurance for citizens over age 65. The traditional form of Medicare (TM) pays health care providers a predetermined fee for each service they provide and exposes enrollees to relatively high cost sharing.[8] Alternatively, beneficiaries can choose to enroll in a Medicare Advantage (MA) managed care plan that charges a monthly premium in exchange

[8] Traditional Medicare is comprised of universal inpatient coverage for hospitals, skilled nursing facilities, and hospice facilities (known as Part A) and coverage for physician services and outpatient treatments (known as Part B). Enrolling in Part B requires paying an additional monthly premium. Over 90% of people over age 65 choose to enroll in Part B.

for lower cost sharing than TM and may use alternative methods of paying providers.[9] We analyze Medicare administrative records from the US Centers for Medicare and Medicaid Services (CMS). CMS maintains a comprehensive national database on beneficiaries, including their addresses, medical claims, and demographics. We start with a random 10% sample of all beneficiaries in 2001 and then add random 10% samples of all new beneficiaries each year from 2002 to 2013.[10]

After compiling these data, we extract records for the subset of individuals for whom we can observe health, residential location, and $PM_{2.5}$ exposure at the point when $PM_{2.5}$ regulation effectively began in 2004. We start with everyone who was 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 who lived in "unclassifiable" counties that lacked $PM_{2.5}$ monitors at the time regulation began. This data cut is standard in air pollution studies due to the increased scope for measurement error.[11] 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 in 2004, and our models require the opportunity to observe within-person changes in dementia. However, for some analysis we expand the sample to include MA enrollees and evaluate the use of ADRD medication as the outcome of interest.

Our third exclusion is to drop 0.3 million individuals who had dementia in 2004

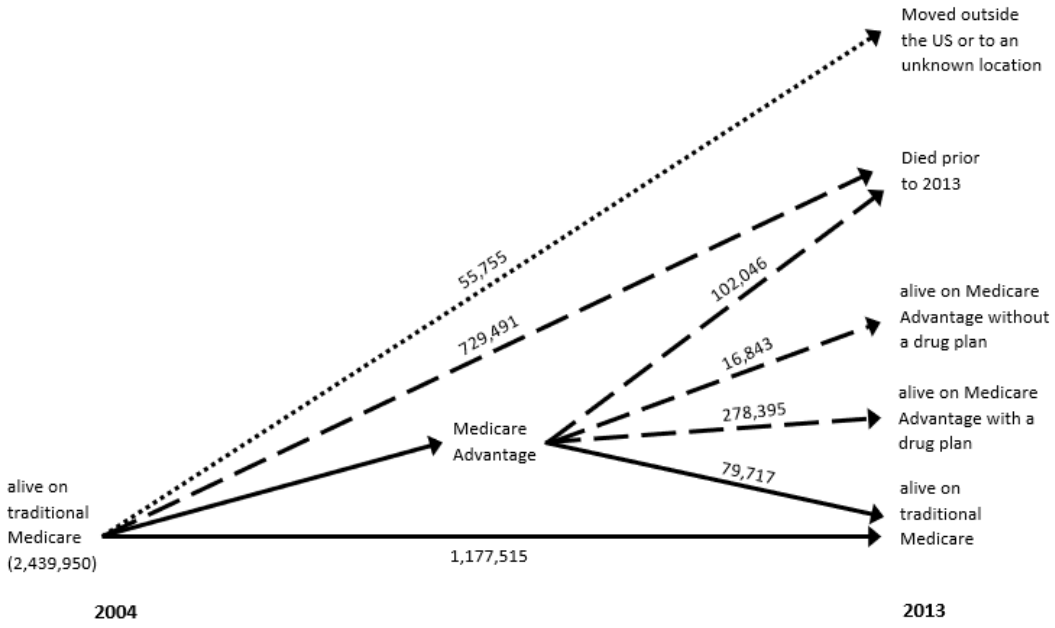
[9] MA enrollees are left out of most studies of Medicare beneficiaries due to data limitations during our study period, but we are able to overcome these limitations and include MA enrollees in some specifications, described below.

[10] Some people become eligible prior to age 65, for example due to disability, but we exclude them from the data until they turn 65. Due to the provenance of our data, we also include an independent, random 20% sample from the universe of age 65 and over beneficiaries who purchased standalone prescription drug insurance plans through Medicare Part D at any point between 2006 and 2010 without the aid of low-income subsidies.

[11] Spatially interpolating their pollution exposures relies exclusively on information from other counties, which may increase measurement error due to the greater distance between people's residences and the monitors. This could pose a threat even to 2SLS estimation if the measurement error tends to be greater in the unmonitored/unclassifiable counties because they were treated the same as attainment counties for regulatory purposes. We avoid this threat to identification by dropping people who lived in unmonitored/unclassifiable counties at the time nonattainment designations were made.

because the disease is currently irreversible, leaving no scope for change.[12] Finally, we drop 0.4 million individuals whose CMS records are missing claims in 2004 or who we could not assign to a Census block group in 2004 based on their mailing address on file or due to the fact that they moved during that year. These sample cuts are unlikely to compromise external validity. Appendix 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.

FIGURE III: SAMPLE SIZES AND TRANSITIONS FROM 2004-2013



Note: The solid arrows denote our primary sample. The dashed arrows indicate samples we use in sensitivity analyses that evaluate any effect on our estimates from selection on survival or selection on type of Medicare plan. The dotted arrow denotes a small subsample that we exclude because they moved to a location outside the United States, or to another location that we were unable to geocode, leaving us unable to reliably estimate their pollution exposure.

The resulting sample consists of 2,439,950 individuals in 2004. Figure III illustrates how between 2004 and 2013, some of these individuals move outside of the

[12] As described below, we perform a model validation test using a sample that includes those with dementia in 2004.

continental US, move out of TM into MA and perhaps back again, or die. Our primary estimation sample is comprised of 1,257,232 individuals who are alive and enrolled in traditional Medicare in 2013 (1,177,515 individuals who were continuously enrolled in TM from 2004 to 2013 plus 79,717 who moved from TM to MA and then back to TM). We explicitly account for potential selection bias caused by focusing on this balanced panel of TM survivors by additionally estimating models with extended samples that include those who die before 2013 and those who move and remain in Medicare Advantage through 2013, as denoted by the dashed arrows in Figure III. Thus, we ultimately estimate models using 98% of the individuals in our data without dementia in 2004. We drop 2% for whom we cannot reliably assign pollution exposure because they move outside the US or to an address that we are unable to geocode.

B. Dementia and its risk factors

For individuals in traditional Medicare, CMS's Chronic Conditions Data Warehouse file uses codes on insurance claims to track if and when each individual is diagnosed with a range of specific chronic medical conditions. A diagnosis of dementia as officially defined by the World Health Organization (see footnote 1) is based on the presence of multiple symptoms of cognitive impairment that significantly impact daily functioning.[13] Examples include memory loss, impaired judgement, loss of spatial awareness, depression, and behavioral changes. Alzheimer's disease is the primary type of dementia, accounting for 60% to 80% of all cases (Alzheimer's Association 2018).[14] Figure IV shows how the fraction of individuals with dementia varies by age and gender in 2013. Approximately 2% of our sample

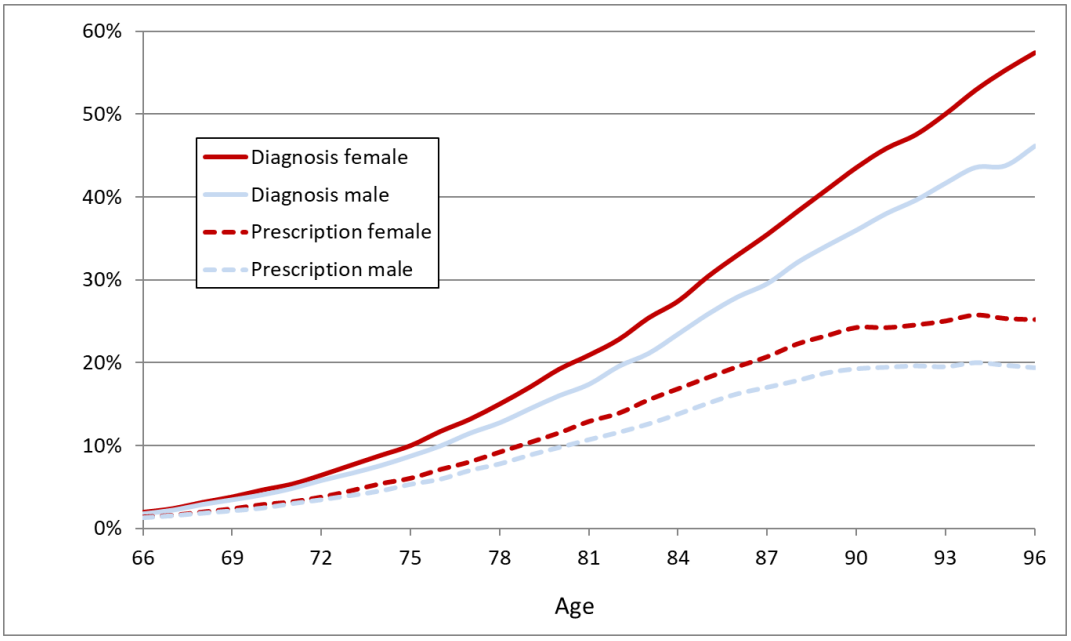
[14] The ICD-10 defines Alzheimer's disease (G30) as "A degenerative disease of the brain characterized by the insidious onset of dementia. Impairment of memory, judgment, attention span, and problem solving skills are followed by severe apraxias and a global loss of cognitive abilities. The condition primarily occurs after age 60, and is marked pathologically by severe cortical atrophy and the triad of senile plaques; neurofibrillary tangles; and neuropil threads" (World Health Organization 2011).

receives a diagnosis by age 66. Diagnosis rates increase gradually with age through the mid-seventies before accelerating in the late seventies and beyond. More than one third of those living to age 90 receive a dementia diagnosis by that point. The diagnosis rate is higher for women, and this gender gap widens with age.

This claims-based approach to identifying dementia cases has been well validated, with Medicare claims from 2007-2012 correctly identifying 85 percent of patients diagnosed with dementia by clinician researchers using in-person assessments (Lee et al. 2019; see also Taylor Jr., Fillenbaum, and Ezell 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 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, 1,098,256 individuals in our sample 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 IV: DEMENTIA DIAGNOSIS AND PRESCRIPTION DRUG USE BY AGE AND GENDER IN 2013



CMS data also provide controls for the known medical risk factors for dementia. These include chronic conditions that reduce the flow of blood and oxygen to the brain (Alzheimer's Association 2019). Most individuals in our data were diagnosed with at least one of these risk factors by 2004: stroke (7%), congestive heart failure (13%), diabetes (22%), ischemic heart disease (36%), and hypertension (67%). Additional behavioral factors associated with lower risk of dementia include higher educational attainment, better nutrition and overall physical health, and a higher degree of social and cognitive engagement. We proxy for these individual-level behaviors by using the average characteristics of individuals living in each individual's 2004 Census block group.[15] From the US Census Summary files, we use block-group averages of household income, per capita income, housing value, gross rent, housing stock age, percent of the housing stock that is owner occupied, share of residents over 65, share of residents by race, and share of residents by

[15] A block group contains 600 to 3,000 residents on average (US Census).

educational attainment.

C. Using Address Histories to Measure Long-Term Pollution Exposure

CMS uses information from the US Social Security Administration to track Medicare beneficiaries' residential addresses. We obtain ZIP+4 Codes for each individual's sequence of addresses from 2004 to 2013. ZIP+4 Codes are close to street addresses in terms of spatial precision: each code corresponds to a single mail delivery point such as a house, one floor of an apartment building, or one side of a street on a city block. The US includes more than 34 million ZIP+4 Codes, or about one for every four households.

Migration rates in our sample are similar to those reported by the Census Bureau for individuals aged 65 and above. Over two-thirds of individuals live in the same ZIP+4 throughout our study period. Of the 31% of individuals who move at least once, 17% move between counties and 10% move between states. We use this information to measure each individual's long-term exposure to air pollution, incorporating changes in pollution experienced as a result of moving.[16]

Individuals in our estimation samples live in 2.7 million distinct ZIP+4 Codes during 2004-2013. We measure residential exposure to $PM_{2.5}$ based on the concentrations at the centroids of the residential ZIP+4 using data from the EPA's air quality system. These data include a balanced panel of 485 monitors that monitored $PM_{2.5}$ continuously for our entire study period (2001-2013) and a total of 1,722 monitors over this time.

We use the latitude and longitude coordinates of each monitor along with the

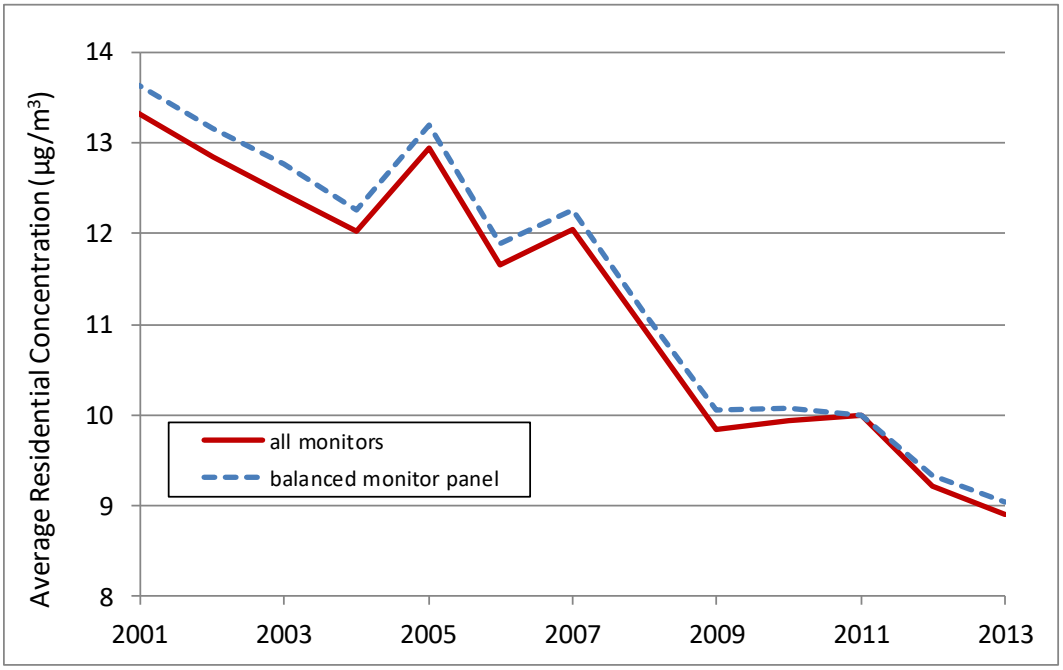
[16] We are unable to observe seasonal migration by people with more than one residence (e.g., snowbirds) because we only observe the residential address on record with the Social Security Administration and CMS. Fortunately, the scope for measurement error is small. Jeffery (2015) estimates that seasonal migrators only account for 2% to 4.1% of the Medicare population based on addresses on Medicare claims for individuals' primary care and emergency room visits.

coordinates of each ZIP+4 to assign the annual average concentration at each residence.[17] Specifically, we calculate the shortest distance between each ZIP+4 centroid and each monitor. Then, for each centroid-year combination, we calculate a weighted average of ambient concentrations recorded at all monitors with the weights given by the square of the inverse distance.[18] Thus, as the distance from a ZIP+4 centroid to a monitor increases, the weight assigned to that monitor decreases. We combine the resulting set of ZIP+4-specific local $PM_{2.5}$ readings with individuals' residential ZIP+4 histories to construct individual-specific exposure histories. Finally, we repeat this process to measure $PM_{2.5}$ from 2001 to 2003 at the locations where individuals lived in 2004. By using these data to control for pre-regulatory $PM_{2.5}$ levels, we can identify $PM_{2.5}$'s effect on dementia from variation in post-regulatory exposures among individuals who lived in similarly polluted neighborhoods at the time regulation began but differed in whether their neighborhoods were in or out of attainment.

These exposure histories are the most comprehensive data ever developed to study how air pollution affects cognitive impairment among older adults. Like all existing methods for measuring pollution exposure, the constructed histories may embed measurement error because of our inability to fully observe factors such as avoidance behavior, the location and duration of activities taking place outside of the home, variation in indoor air penetration rates due to heterogeneity in home sealing, and variation in respiration due to health and physical activity. Our instrumental variables approach also helps to address these sources of measurement error.

[17] Geographic coordinates of ZIP+4 centroids were purchased from GeoLytics, which created them from the Census Bureau's TIGER/line Shapefiles and US Postal Service records.
[18] This method of interpolation, with weights given by the distance raised to a negative exponent, is a predominant method in the environmental economics literature.

FIGURE V: AVERAGE RESIDENTIAL CONCENTRATION OF $PM_{2.5}$ BY YEAR



Note: The figure reports the annual average concentrations of fine particulate matter based on place of residence for our sample of Medicare beneficiaries.

Exposure to air pollution among the US Medicare population declined substantially during the 2000s. Figure V shows that annual average residential exposure to $PM_{2.5}$ declined from over 13 $\mu g/m^3$ in 2001 to about 9 $\mu g/m^3$ in 2013. This is true regardless of whether we measure exposure using the 2001-2013 balanced panel of 485 monitors (the dashed line) or the unbalanced panel of all 1,722 monitors in operation each year (solid line). We feature this balanced panel in our main econometric analysis to avoid measurement error that could be introduced if new monitors tend to be located in more or less polluted areas (Muller and Rudd 2017, Grainger, Schreiber, and Chang 2018, Grainger and Schreiber 2019). We also show that our results are robust to instead using the unbalanced monitor panel.

IV. Main Econometric Model and Results

A. Identification of the linear 2SLS model

Let $y_{i,t}$ indicate whether individual i has dementia in year t and let $\Delta y_i = y_{i,2013} - y_{i,2004}$ denote the change in dementia status between 2004 and 2013. Because dementia has no cure, it is an absorbing state and, by definition, Δy_i is equal to zero for all individuals who have dementia in 2004. Therefore we model whether individual i is *newly* diagnosed with dementia by the end of 2013 (i.e., we model dementia onset) and restrict our primary sample to individuals who had not received a dementia diagnosis before the end of 2004.[19] This measure of new dementia diagnosis is the dependent variable in our primary, linear probability model,[20]

$$(1)\ \Delta y_i = \alpha \sum_{t=2004}^{2013} \frac{PM2.5_{i,t}}{10} + \eta_{c(i)} + \beta X_i + \gamma H_i + \theta W_i + f\left(\sum_{t=2001}^{2003} \frac{PM2.5_{i,t}}{3}\right) + \epsilon_i.$$

The coefficient of interest in equation (1), α, measures the effect of the average concentration of $PM_{2.5}$ at the individual's residence over the decade (from 2004 to 2013) on Δy_i.[21] While this model assumes that any effect of $PM_{2.5}$ is linear and constant over time, below we present results from non-linear models and from models that vary the length of history of $PM_{2.5}$ accumulation.

We control for individual and neighborhood characteristics that may be correlated with both dementia and $PM_{2.5}$. First, we add dummy variables, $\eta_{c(i)}$, for the 2013 core-based statistical area (CBSA) in which individuals live.[22] This absorbs

[19] The results presented in Section IV restrict the sample to those who were still alive in 2013. Section V addresses potential biases arising from this sample selection.

[20] We begin with a model of dementia onset, which is standard in the research on dementia. In principle, we could instead begin with a model describing an individual's dementia status in both 2004 and 2013 to derive equation (1), which would directly specify the relationship between the instruments and unobserved determinants of dementia in each time period. Such a model is shown in Appendix B. Our model of onset allows us to make an equivalent strict-exogeneity assumption and our discussion of identification below explicitly accounts for the fact that error in the onset equation captures changes in unobservable dementia determinants, conditional on not having dementia in 2004.

[21] This model is similar to stress models discussed in Deaton and Paxson (1998).

[22] There are approximately 1,000 CBSAs, which are defined according to the Office of Management and Budget as of one or more counties anchored by an urban center of at least 10,000 people plus adjacent counties that are socioeconomically tied to the urban center by commuting. For people living outside of CBSAs, we create a state-specific, rural dummy variable.

the effects of environmental factors that could be spatially correlated with both pollution and dementia. Examples include extreme temperatures, the presence of lead pipes, and chemical exposures via hazardous waste sites. In particular, extreme temperatures are known to cause morbidities that serve as risk factors for dementia (Deschenes 2014). Equally important, these dummies will absorb variation across CBSAs in access to medical care and doctors' diagnostic procedures that could lead to spatial variation in dementia diagnosis rates. Additionally, for the majority of individuals who never move during our study period, the CBSA dummies will control for pre-regulatory sorting across CBSAs on the basis of latent characteristics that may serve as risk factors for dementia (Finkelstein, Gentzkow, and Williams 2016).

To control for heterogeneity in dementia risk among individuals living in each CBSA, we utilize all of their demographic information in Medicare records along with relevant information about their health at the start of the decade. The X_i vector includes indicators for race and gender-specific indicators for integer age at the end of 2013 (from 75 through 100).[23] These flexible age-by-gender controls absorb the nonlinear trends in dementia rates shown in Figure IV.

Because our dependent variable is dementia onset (i.e., we condition on not having dementia at baseline), we include H_i, which is a vector characterizing baseline health in 2004. We employ a full-factorial design to control for pre-existing medical conditions that are known risk factors for dementia, adding dummy variables for each of 32 possible combinations of hypertension, diabetes, congestive heart failure, ischemic heart disease, and stroke.[24] We further control for unob-

[23] 75 is the minimum age in 2013 because the sample is limited to people who were 65 or older on January 1, 2004. Centenarians are grouped into two gender-specific bins because their relatively small numbers prevent us from precisely estimating age-specific coefficients. Our findings on air pollution are unaffected by adding age-specific bins beyond age 100.

[24] Because air pollution is a risk factor for these morbidities, controlling for them will also help to absorb the manifested effects of individual differences in pollution exposure prior to our study period. The full list of these interaction terms is provided in Table A2.B.

served heterogeneity in baseline health by adding a fourth-order polynomial function of gross expenditures on all health care services covered by Medicare Parts A and B in 2004.[25]

To proxy for socioeconomic characteristics that we do not observe for individuals, such as wealth, education, and degree of social engagement, we add a series of covariates, W_i, describing the residents of individual i's 2004 Census block group. Specifically, we include median household income, income per capita, mean and median house value, median rent, median house age, fractions of the housing stock that are owner occupied, renter occupied and vacant, fraction of the residents over age 65, fractions of residents who report being white, black, and Hispanic, and the fractions of residents in each of seven educational-attainment bins. These neighborhood-level measures also serve to control for within-CBSA heterogeneity in other amenities known to attract wealthier households with higher education.

Finally, we add a fourth-order polynomial function, $f(\cdot)$, in baseline $PM_{2.5}$ exposure from 2001 through 2003 at individual i's residential location in 2004. This controls for any residual effects of pre-regulatory sorting into more polluted neighborhoods by individuals who are more likely to receive a future dementia diagnosis. Controlling for baseline neighborhood concentration also makes the identification of α in equation (1) similar to a first-differences model. That is, α is identified by how cumulative $PM_{2.5}$ exposure from 2004 to 2013 affects the probability of a new dementia diagnosis, conditional on pre-regulatory concentrations in the individuals' baseline neighborhoods.

Despite the rich set of controls in equation (1), two potential threats to identification remain: measurement error in pollution exposure and omitted variable bias.

[25] Medicare Parts A and B cover virtually all medical services aside from prescription drugs and long-term care. This includes doctors' services, preventive care, durable medical equipment, hospital outpatient services, laboratory tests, imaging, hospital inpatient services, nursing facilities, and hospice care.

We address both concerns by instrumenting for decadal exposure. Equation (2) provides the first stage of the 2SLS model:

$$(2)\ \sum_{t=2004}^{2013}\frac{PM2.5_{i,t}}{10} = \pi Z_i + \xi_{c(i)} + \sigma X_i + \tau H_i + \omega W_i + f\left(\sum_{t=2001}^{2003}\frac{PM2.5_{i,t}}{3}\right) + \varepsilon_i.$$

Z_i is a vector of instrumental variables created by interacting an indicator for individuals who resided in nonattainment counties in 2004 with the fourth-order polynomial function of baseline exposure that enters the second-stage model. This flexibility capitalizes on the within-county variation in subsequent $PM_{2.5}$ exposure due to local regulators' responses to nonattainment designations.[26]

In our 2SLS models, α is identified by variation in (instrumented) decadal exposure to $PM_{2.5}$ experienced by individuals of the same age, race, and gender who lived in the same CBSA and who, at the start of the decade, had not received a dementia diagnosis and had received the same medical diagnoses for dementia risk factors, had the same level of gross annual medical expenditures, and had sorted themselves into neighborhoods with the same baseline levels of $PM_{2.5}$ and with the same distributions of race, income, educational attainment, and property values. The identifying variation in $PM_{2.5}$ arises from three sources. First, some CBSAs include both attainment and nonattainment counties, yielding between-county differences in post-regulatory exposures similar to the identifying variation in Chay and Greenstone (2005) and Isen, Rossin-Slater, and Walker (2017). Second, within each county, residential locations differ in their initial distance from the attainment threshold, yielding within-county differences in post-regulatory exposure due to local targeting of pollution hot spots similar to the identifying variation in Auffhammer, Bento, and Lowe (2009) and Bento, Freedman, and Lang (2015).[27] Third, individuals who moved between 2004 and 2013 experienced variation in exposure

[26] Formally, instrument validity requires that cov(Z, ϵ) = 0 where, as discussed above, ϵ represents unobserved attributes that affect dementia onset for those who did not have dementia at the beginning of our sample.

[27] Appendix Figure A4 illustrates the first two sources of identifying variation by showing within-CBSA and within-county variation in nonattainment status conditional on baseline $PM_{2.5}$ concentrations, using New York and Chicago as examples.

due to their migration paths, similar to the identifying variation in Banzhaf and Walsh (2008).

B. First-stage results

The pollution exposure histories and first-stage estimates reveal that the EPA's $PM_{2.5}$ regulation was followed by systematic changes in exposure. First, average exposures declined for more than 95% of individuals between 2001-2003 and 2004-2013. Second, the declines were larger for individuals whose 2004 neighborhoods were more polluted at baseline (2001-2003). Third, conditional on baseline neighborhood pollution, the declines were larger for individuals whose 2004 neighborhoods were in nonattainment counties. Figure VI.A illustrates these trends. It plots estimates for the average decline in exposures between 2001-2003 and 2004-2013, conditional on county attainment status and baseline pollution levels. These estimates are derived by regressing changes in individual exposure on indicators for 0.33 $\mu g/m^3$ bins of baseline exposure interacted with county attainment status. Additional covariates include the CBSA dummies, block group variables, and all individual variables from equations (1)-(2). The resulting trend lines mirror Auffhammer, Bento, and Lowe's (2009) estimates for the partial effect of the EPA's 1990 county nonattainment designations for PM_{10} on subsequent PM_{10} concentrations.

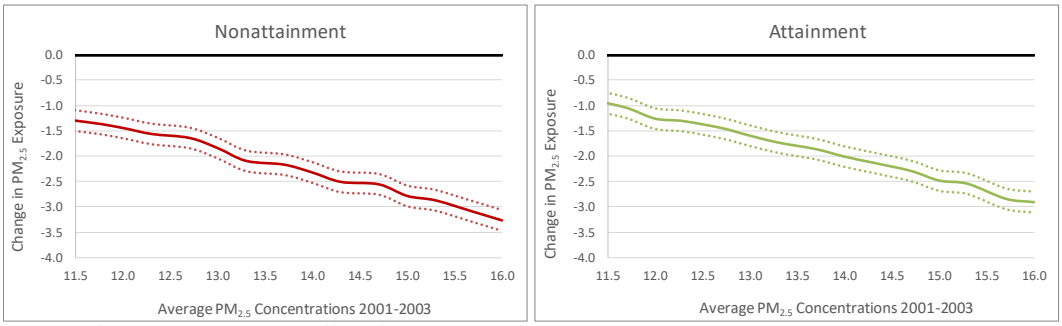
The identifying variation for our 2SLS model comes from the difference between the attainment and nonattainment trend lines in Figure VI.A. Visual inspection shows that $PM_{2.5}$ declined by more in nonattainment counties, even conditional on baseline exposure. Figure VI.B provides a more formal illustration of the identifying variation. It uses the coefficients on the instruments from equation (2) to

plot the estimated partial effect of nonattainment on post-regulatory $PM_{2.5}$ exposure, conditional on baseline exposure.[28] Intuitively, the partial effect of nonattainment is negative. The size of the effect declines in baseline concentrations as we approach the regulatory threshold from below. Potential explanations for the declining difference include spatial spillovers from pollution control effort in nonattainment areas and incentives for attainment area regulators to target known hot spots that could cause them to be reclassified in the future. Finally, the first-stage F statistic is 637, suggesting that any finite sample bias is negligible.

The partial effect of nonattainment on individual $PM_{2.5}$ exposure in Figure VI.B is noticeably smaller than the reduction implied by visual comparison between attainment and nonattainment counties' average concentrations in Figure II. This is because the covariates in (1)-(2) absorb much of the regulation's effect. In particular, spatial dummies absorb the between-CBSA variation in $PM_{2.5}$ reductions. To illustrate the regulation's full effect on average $PM_{2.5}$ reductions, we regress differences between individuals' decadal exposures and their baseline exposures on the county nonattainment indicator. This difference-in-differences regression shows that average $PM_{2.5}$ exposure declined by 1.24 $\mu g/m^3$ more among those in nonattainment counties than those living in attainment counties, with declines of 3.04 $\mu g/m^3$ and 1.80 $\mu g/m^3$, respectively. We interpret this difference as the regulation's approximate effect on exposure in nonattainment. This reduction is slightly larger than in Figure II mainly because of within-county variation in where individuals live in relation to monitors.

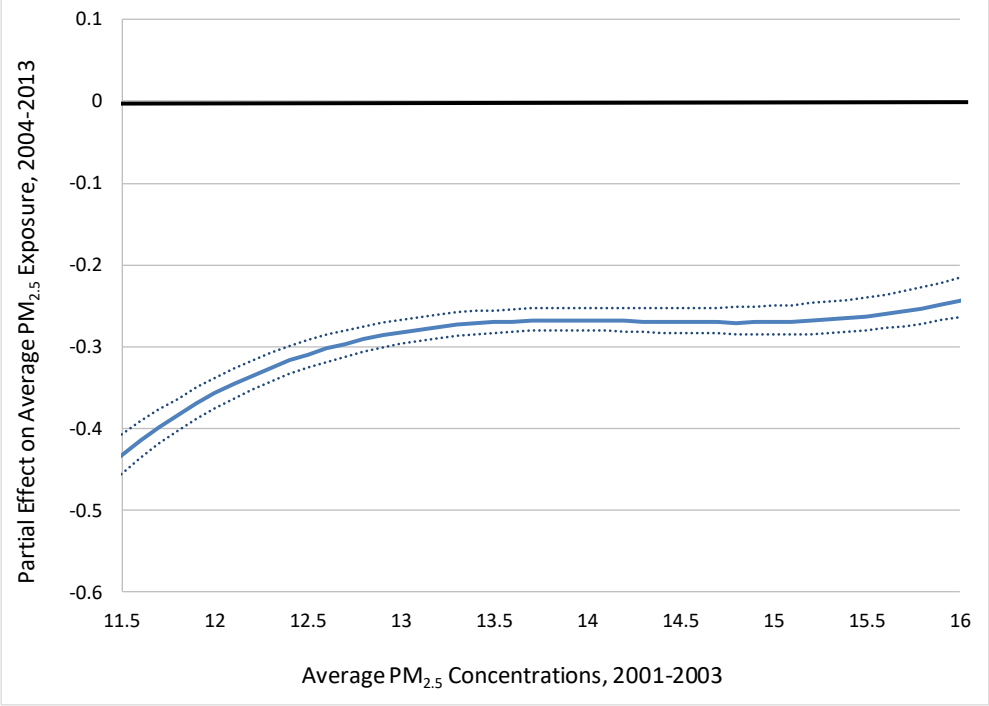
[28] Table A2 reports the model coefficients.

FIGURE VI.A: CHANGES IN POST-REGULATORY $PM_{2.5}$ EXPOSURE, BY ATTAINMENT STATUS AND PRE-REGULATORY CONCENTRATIONS 2001-2003



Note: The figure shows the average effect of the nonattainment designation on the average conditional change in decadal $PM_{2.5}$ concentrations. The dotted lines denote 95% confidence bands with clustering on Census block group.

FIGURE VI.B: ESTIMATED PARTIAL EFFECT OF NONATTAINMENT ON POST-REGULATORY $PM_{2.5}$ EXPOSURE, BY PRE-REGULATORY CONCENTRATIONS 2001-2003



Note: The figure shows the average effect of the nonattainment designation on the average conditional change in decadal $PM_{2.5}$ concentrations. The dotted lines denote 95% confidence bands constructed from 1,000 bootstrap replications, with clustering on Census block group.

C. Second-stage results

Table I presents results from models with and without covariates and instruments. The dementia indicator is multiplied by 100 so that $PM_{2.5}$ coefficients represent percentage point (pp) changes in the probability of receiving a dementia diagnosis. Standard errors are robust to heteroscedasticity and are clustered at the Census block group level to allow for spatial correlation in diagnoses.[29]

Column (1) shows the result from an OLS regression that includes only decadal $PM_{2.5}$ and CBSA-specific intercepts. A 1-$\mu g/m^3$ increase in average residential concentrations of $PM_{2.5}$ from 2004 through 2013 is associated with a 0.75 pp increase in the probability of receiving a dementia diagnosis by the end of 2013. About 28% of this association persists in Column (2) when we add all observed measures of baseline health and $PM_{2.5}$ exposure, demographics and socioeconomic status.

TABLE I—DECADAL EXPOSURE TO $PM_{2.5}$ AND DEMENTIA IN 2013

	(1)	(2)	(3)	(4)	(5)	(6)
decadal $PM_{2.5}$ (1 $\mu g/m^3$)	0.751***	0.209*	1.164***	1.679***	1.626***	1.844***
	(0.06)	(0.11)	(0.09)	(0.49)	(0.49)	(0.47)
ind. & neigh. covariates		x		x	x	x
specification	OLS	OLS	2SLS	2SLS	IV Probit	IV Probit
first-stage F statistic			19,161	637	637	44
number of individuals	1,257,232	1,257,232	1,257,232	1,257,232	1,257,232	1,257,232
share with dementia in 2013	22	22	22	22	22	22

Note: The dependent variable equals 100 if an individual was diagnosed with dementia prior to the end of 2013 and 0 otherwise. Col (1) is a univariate OLS regression 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. Columns (3) and (4) are the 2SLS analogues to Columns (1) and (2), respectively. The first row of Columns (1)-(4) presents the coefficient on decadal $PM_{2.5}$, which is the average marginal effect in these models. Coefficients on all other covariates in the first and second stage models in Col (4) are reported in Appendix Table A2. Col (5) is the control-function probit analogue to the 2SLS model in Col (4). Col (6) is a control-function probit that allows for additionally flexibility in both stages of estimation. The first row of Columns (5) and (6) present the average marginal effect of decadal $PM_{2.5}$ on dementia. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered by block group. Standard errors in Columns (5) and (6) are bootstrapped using 1,000 repetitions.

[29] Because our instrumental-variables-based measure of pollution varies at the fine level of the ZIP+4, we cluster our standard errors at the coarser level of the block group. Our results are robust to clustering at the even coarser county level.

Columns (3) and (4) show the 2SLS analogs to the OLS models in columns (1) and (2). The first stage results are reported in Table A2. The p-value from a Sargan test of over-identifying restrictions is 0.34, so we fail to reject the joint hypothesis that our instruments are valid and that the model is correctly specified. Using instrumental variables increases the estimates for $PM_{2.5}$'s effect and makes the estimates less sensitive to the inclusion of individual covariates. The second-stage coefficient on $PM_{2.5}$ in our main specification, Column (4), is about seven times larger than the corresponding OLS estimate from Column (2), consistent with substantial measurement error in pollution exposure.[30] The coefficient implies that a 1-$\mu g/m^3$ increase in average $PM_{2.5}$ from 2004 through 2013 increased the probability of a dementia diagnosis by the end of 2013 by 1.68 pp.[31]

The fitted probabilities of receiving a dementia diagnosis from the linear 2SLS model lie between zero and one for 99.9% of individuals. Nonetheless, we evaluate whether the results in Column (4) are robust to the linearity assumption. First, we estimate a probit model that controls for the endogeneity of $PM_{2.5}$ using the control-function approach developed in Rivers and Vuong (1988) and, second, we estimate a flexible version of this model using the insights of Blundell and Powell (2003, 2004) for estimating non-parametric, binary-response models with endogenous regressors. For these models, we make the identifying assumptions that the errors in equations (1) and (2) are jointly normal and independent of the instruments.

To estimate the first control-function probit model, we proceed in two stages; in the first stage, equation (2) is estimated via OLS and, in the second stage, a probit version of equation (1) is estimated via maximum likelihood, where we include the

[30] Deryugina et al. (2019) report similar differences between OLS and 2SLS results in estimating the effect of daily pollution spikes on mortality among the Medicare population.

[31] Coefficients on the remaining covariates are reported in Appendix Table A2. We find that diagnosis rates tend to be higher for African-Americans (+3.7 pp) and Hispanics (+3.4 pp) relative to Asians (+0.5 pp) and whites (+0.8 pp), with "other race" as the omitted category. Diagnosis rates also decline by about 1% for every $100,000 of additional neighborhood income per capita and tend to be lower in neighborhoods with higher educational attainment. For example, 10 pp higher fraction of block group residents with graduate degrees (relative to less than 8th grade education) is associated with a 0.5 pp lower probability of a dementia diagnosis.

first-stage residuals as a control. The estimated average marginal effect of $PM_{2.5}$ on dementia shown in Column (5) implies that a 1-$\mu g/m^3$ increase in average $PM_{2.5}$ from 2004 through 2013 increased the probability of a dementia diagnosis by the end of 2013 by 1.63 pp.

Our second probit model is a flexible extension of this control-function probit model. While the controls themselves enter all specifications in a flexible manner, the model underlying Column (5) specifies a latent dementia propensity that is linear in $PM_{2.5}$ and additively separable in $PM_{2.5}$ and the controls. Therefore, we extend the probit model by allowing $PM_{2.5}$ to flexibly enter as a fourth-order polynomial and include interactions between $PM_{2.5}$ and the vectors X_i, H_i, and W_i (individual demographics, individual health and health-spending controls, and Census block-group demographics, respectively).[32] In this approach, the effect of $PM_{2.5}$ on the latent propensity to be diagnosed with dementia can vary flexibly with both the level of $PM_{2.5}$ and with the levels of individual and neighborhood characteristics. We additionally specify a flexible first-stage regression that allows for interactions between attainment status and all of the included controls.[33] As shown in Column (6), the average marginal effect from this model implies that a 1-$\mu g/m^3$ increase in average $PM_{2.5}$ from 2004 through 2013 increases the probability of a dementia diagnosis by 1.84 pp.[34]

This flexible probit model provides estimates of the heterogeneity in the marginal effects of $PM_{2.5}$ across the level of decadal exposure to $PM_{2.5}$ and across individual and neighborhood characteristics. The results show that for the range currently relevant for the US, the marginal effects are larger at lower levels of $PM_{2.5}$. This implies that, all else equal, further reductions in $PM_{2.5}$ would have greater

[32] The results are not sensitive to using even higher-order polynomials. When using an eighth-order polynomial in exposure, we find an average marginal effect of 1.80.
[33] Note that using the same linear first stage as in Columns (4) and (5) results in a slightly lower estimate of 1.78, versus the estimate of 1.84 shown in Column (6) of Table I.
[34] It is convenient in models of this class to focus on average marginal effects as that allows the researcher to begin with a flexible representation of the probability of dementia instead of deriving this probability from the structural model (Wooldridge 2015).

effects on reducing dementia than we estimate for the reductions in 2004-2013. For example, the current regulatory threshold of 12 $\mu g/m^3$ that was established in 2012 is near the 75th percentile of decadal exposure in our data (11.94 $\mu g/m^3$). The results from the flexible probit model indicate that individuals with decadal exposures within a one-unit window of 12 $\mu g/m^3$ (i.e., between 11.5 and 12.5 $\mu g/m^3$) experienced an average marginal effect of 0.99 pp. But among those within a one-unit window of 11 $\mu g/m^3$ (around the median of 11.19 $\mu g/m^3$ in our sample, and below the current regulations) the model yields a larger average marginal effect of 1.67 pp. At the 25th percentile of decadal exposure in our data (10.04 $\mu g/m^3$), the average marginal effect is 2.52 pp. As calculated, the differentials in these average marginal effects take into account both the non-linearity in $PM_{2.5}$ and the impacts of the heterogeneous effects of individual and neighborhood characteristics.

D. Assessing the magnitude of $PM_{2.5}$'s effects on dementia

The point estimate from our main 2SLS model (Table I, Column (4)) indicates that a 1 $\mu g/m^3$ increase in 10-year average residential concentrations of $PM_{2.5}$ from 2004 to 2013 increases the probability of receiving a dementia diagnosis by 1.68 pp. This is equivalent to a 7.5% increase relative to the dementia diagnosis rate among our sample. To provide context for these results, a 1 $\mu g/m^3$ change is equivalent to 9.1% of the average person's exposure during our study period and 59% of a standard deviation. Thus, a 1 $\mu g/m^3$ increase is a moderate change in exposure.

Table II compares our $PM_{2.5}$ result to the coefficients we estimate on other dementia risk factors that were included as covariates in the model.[35] For instance, our estimate for the effect of a 1 $\mu g/m^3$ increase in decadal $PM_{2.5}$ is about twice 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

[35] The estimates presented here come from our main, linear 2SLS specification summarized in Table I Column (4). Corresponding estimates from the probit-model specifications yield similar results.

of the other health risk factors (0.8 pp). Our $PM_{2.5}$ estimate is smaller than the risks associated with pre-existing diagnoses of the other chronic conditions individually, which range from a 2.1 pp increase for ischemic heart disease alone to a 8.0 pp increase for stroke alone. We estimate that someone diagnosed with all five risk factors by 2004 had a 20.6 pp higher probability of being diagnosed with dementia by the end of 2013, all else equal. Aging provides another opportunity for comparison. Focusing on females, our $PM_{2.5}$ estimate is approximately one-quarter of the conditional increase associated with aging from 75 to 80 and one-tenth of the conditional increase associated with aging from 75 to 85.

TABLE II. 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	1.0
decadal $PM_{2.5}$ (1 μg/m3)	1.7	0.7	2.6
ischemic heart disease in 2004	2.1	1.7	2.5
diabetes in 2004	3.3	2.8	3.8
congestive heart failure in 2004	4.3	3.1	5.5
Aging from 75 to 80 (women)	6.0	5.6	6.4
stroke in 2004	8.0	6.9	9.1
aging from 75 to 85 (women)	15.2	14.8	15.7
All five chronic conditions in 2004	20.6	19.5	21.6

Note: The table reports point estimates and 95% confidence intervals for dementia risk factors based on the model in Table I, Column (4). Appendix Table A2 reports the full set of model coefficients.

V. Using Partial Identification and Instruments to Assess Selection Bias

A. Selection on Survival

Prior work has found that $PM_{2.5}$ kills people on Medicare (Di et al. 2017,

Deryugina et al. 2019). For example, Deryugina et al. uses an instrumental-variables regression to conclude that a one-day 1 $\mu g/m^3$ increase in $PM_{2.5}$ causes a 0.18% increase in mortality over three days. When we estimate the 2SLS specification in equations (1)-(2) with decadal mortality as the dependent variable, we find that a 1-$\mu g/m^3$ increase in average $PM_{2.5}$ from 2004 through 2013 increases mortality by 2.37 pp, equivalent to 6% of the decadal mortality rate.[36] These results, combined with the concern that unobserved aspects of health that determine survival may be correlated with unobserved aspects of heath that determines dementia, suggest that sample selection may condition the interpretation of our estimates. For example, suppose that unobserved health determining survival is negatively correlated with unobserved health determining dementia, i.e., sicker people who are more likely to die are also more likely to be diagnosed with dementia if they live. In this case, selection would induce a negative correlation between the error in equation (1) and our instrumented measure of $PM_{2.5}$, biasing downward the estimate of $PM_{2.5}$'s direct effect on dementia in the selected sample.[37]

If selection bias were present, then the preceding IV estimates capture the total effect of $PM_{2.5}$ on the dementia rate, which combines 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 could change for two reasons. First, the change in $PM_{2.5}$ could have a causal effect on dementia. Second, the change in $PM_{2.5}$ could have a causal effect on survival and, if the underlying propensity to develop dementia for the marginal individuals (who are induced to die by the change in pollution) 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. Lee (2009) discusses this concept in

[36] Table A3 reports results from mortality models that parallel the specifications used in Table I.
[37] A less intuitive, but nonetheless possible, concern would be that the unobserved health determining survival was positively correlated with the unobserved health determining dementia. This would induce a positive correlation between the error in equation (1) and our instrumented measure of $PM_{2.5}$ and cause an upward bias in our estimate.

detail in the context of a randomly assigned job-training program that affects whether individuals work as well as their subsequent wages.[38]

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 context, 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 develop dementia and the propensity to survive (e.g., Manski 1990, Horowitz and Manski 2000, Lee 2009).[39] Next, we sharpen the bounds by adding plausible assumptions about the relationship between the propensity to develop 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 move from partial identification to point identification by adding additional instruments and distributional assumptions (e.g., Heckman 1979).

We consider a marginal change in $PM_{2.5}$ and denote the total effect of $PM_{2.5}$ on the dementia rate as Δ. This total effect is comprised of the causal effect of $PM_{2.5}$ on dementia, which we continue to denote as α, and the compositional effect due to selection on survival (Lee 2009). The compositional effect is the product of two terms: the share of marginal individuals who die as a result of the change in $PM_{2.5}$ (we denote this share as ρ) and the difference in the underlying probabilities of developing dementia between this group of marginal individuals (group A) and the inframarginal individuals who survive the change in $PM_{2.5}$ (group B). We denote this difference as $(P_y^B - P_y^A)$. The total effect can then be written as:

[38] See also Blundell, Gosling, Ichimura, and Meghir (2007), which analyzes changes in wage distributions in the presence of compositional changes.

[39] Lee (2009) requires a monotonicity assumption, which in our case would require that all individuals' probability of survival, conditional on observables, responds with the same sign to an increase in $PM_{2.5}$. As our model specifies homogeneous effects of $PM_{2.5}$, conditional on observable characteristics, this is trivially satisfied. Thus, the bounds of Lee (2009) and the bounds of Horowitz and Manski (2000) are equivalent. With heterogenous effects, the Horowitz and Manski (2000) bounds would become wider, while the Lee (2009) bounds would stay the same (but would be interpreted as average treatment effects for the inframarginal households). We consider heterogenous effects of $PM_{2.5}$ in Section IV.B.

$$\Delta = \alpha + \rho * (P_y^B - P_y^A)$$

and re-arranged to isolate the effect of interest, α:

$$\alpha = \Delta + \rho * (P_y^A - P_y^B).$$

We use this equation to calculate simple bounds for α. Further details may be found in Appendix B. Our results from Section IV represent estimates of the total effect Δ. Our analogous results for mortality can be used to construct an estimate of ρ. Bounds for α may be constructed by recognizing that the scaling of our dementia indicator, $\left(P_y^B - P_y^A\right)$, must lie between -100 and 100 (Peterson 1976, Manski 1990), as both P_y^A and P_y^B must each lie between 0 and 100. Tighter bounds may be constructed by using information on the overall dementia rate. For example, an insight of Lee (2009) is that while $(P_y^A - P_y^B)$ is unobserved, the data reveal the overall dementia rate, P_y, which is a weighted average over marginal and inframarginal individuals:

$$P_y = \rho * P_y^A + (1 - \rho)P_y^B.$$

Thus, for any given value of P_y^A, we can solve for P_y^B. By plugging 0 and 100 for P_y^A (and then solving for the corresponding value of P_y^B), bounds for α may be written as:

$$\left[\Delta - \rho * P_y^B(0), \quad \Delta + \rho * \left(100 - P_y^B(100)\right)\right]$$

We first calculate these bounds using the estimates from our linear models for the mean value of $PM_{2.5}$.[40] The estimate of 1.68 in Table I, Column (4) is then interpreted as an estimate of Δ. We additionally have an estimate of the effect of a 1-μg/m^3 increase in $PM_{2.5}$ on the probability of dying of 2.37 percentage points

[40] As the results from the Rivers and Vuong (1988) IV-Probit model are similar to our main results, the analogous bounds for that model are very similar. The identification region for the Lee (2009)-style bounding approach using these IV-Probit estimates is given by [0.78, 4.63]. The identification region using the assumption that $P_y^A \geq P_y^B$ is [1.63, 4.63].

(shown in Table A.3). Combining this estimate with the overall mean survival rate in our sample of 60.51 yields $\rho = 0.04$.[41] The mean dementia rate in our sample provides us with $P_y = 21.98$.

The estimated identification region is shown in brackets in the top row of Table III, Column (1). The lower bound of this region is still 0.82 despite embedding the extreme assumption that everyone who is induced to die by an increase in $PM_{2.5}$ would have zero probability of developing dementia had they survived. Further, the lower bound indicates that only half of the estimated total effect of 1.68 can be explained by a compositional effect. A 95% confidence interval for α is shown in the second row and calculated following the method of Imbens and Manski (2004). While these lower-bound results show that the existence of no causal effect of $PM_{2.5}$ on dementia is unlikely, the upper-bound results cannot rule out effects much larger than what we find when we altogether ignore the potential for selection.

TABLE III—ESTIMATES ALLOWING FOR SELECTION ON SURVIVAL

	(1)	(2)	(3)	(4)	(5)
decadal $PM_{2.5}$ (1 μg/m^3)	[0.82, 4.73]	[1.68, 4.73]	[0.95, 4.17]	[1.84, 4.17]	2.334***
	(-0.02, 6.08)	(0.87, 6.08)	(0.08, 5.32)	(1.07, 5.32)	(0.51)
total number of ind.	2,384,195	2,384,195	2,384,195	2,384,195	2,384,195
num. who survive through 2013	1,257,232	1,257,232	1,257,232	1,257,232	1,257,232
share with dementia in 2013	22	22	22	22	22
share who survive through 2013	61	61	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. Columns (3) and (4) report analogous identification regions to (1) and (2) but with the underlying estimates coming from our most flexible IV Probit specification. Finally, Column (5) reports results from a selection-correction model that uses an exclusion restriction to arrive at a point estimate. Underneath the identification regions in Columns (1)-(4), we report in parentheses the 95% confidence intervals on α using the method described in Imbens and Manski (2004). Underneath the point estimate in Column (5), we report a standard error. The standard errors underlying the confidence intervals in Columns (1)-(4) and the standard error in Column (5) are calculated using a bootstrap with 1,000 replications, clustered by initial Census block group. The first-stage F statistics for Column (5) are shown in Table IV Column (1). Asterisks indicate statistical significance for Column (5) at the 10% (*), 5% (**), and 1% (***) levels.

[41] This share is calculated for a marginal change at the mean as 2.37/60.51=0.039.

Next we sharpen these bounds by assuming a plausible form of monotone treatment selection (Manski and Pepper 2000) in which those who would be induced to die following a change in $PM_{2.5}$ were, on average, no less likely to develop dementia than those whose survival was unaffected by the change $\left(i.e., P_y^A \geq P_y^B\right)$. Intuitively, this assumes that the latent health driving mortality is positively correlated with the latent health driving dementia. Table III Column (2) shows that, under this assumption, the lower bound of the identification region increases to our 2SLS estimate of 1.68.[42]

We alternatively calculate bounds using estimates of Δ, ρ, and P_y from our most flexible IV-Probit model.[43] The bounds for this case are shown in Table III Column (3) with the corresponding monotone treatment selection bounds shown in Column (4). These are of a similar magnitude, respectively, to the bounds in Columns (1) and (2), but tighter.[44] Because this model also allows us to calculate heterogeneity in the average marginal effects of $PM_{2.5}$, we calculate bounds separately for the three subsamples with average decadal $PM_{2.5}$ within the 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 [1.88, 4.27], [0.76, 4.07], and [-0.15, 3.90], 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 the increased $PM_{2.5}$-induced mortality across the bins. These lower bounds reflect the extreme assumption that those who suffered from $PM_{2.5}$-driven mortality would have been immune to dementia had they survived. Applying the assumption that

[42] Making arbitrary assumptions can also sharpen the bounds, e.g., assuming that the dementia rate among the marginal individuals is between half and double the rate of the inframarginal individuals. This would yield an identification region of [1.25, 2.54].

[43] Because this model allows the effects of $PM_{2.5}$ on mortality and dementia to vary by observed characteristics, we use individual-specific values of Δ, ρ, and P_y to create individual-specific marginal effects and then calculate the averages based on specifying the lower and upper bounds of P_y^A for all individuals.

[44] The difference in the estimated effects of $PM_{2.5}$ on mortality (shown in Table A.3) between the two models leads to the tighter bounds.

these individuals were merely no less sensitive than the survivors in terms of developing dementia sharpens the bounds. Under this assumption, the identification regions become [2.52, 4.27], [1.67, 4.07], and [0.99, 3.90], respectively.

The preceding bounds do not require any assumptions regarding the validity of instruments for the selection process. Use of valid instruments, however, allows for point identification of the effect of $PM_{2.5}$ on dementia. The medical literature provides a set of instruments that affect survival but not dementia: prior diagnoses of non-smoking related cancers, which are uncorrelated with dementia outcomes (Driver et al. 2012, Ganguli 2015). Therefore, we complement the partial identification results by adding a control-function approach based on Heckman (1979) and Heckman and Robb (1986) to obtain a selection-corrected point estimate. We begin by estimating a linear probability model of decadal survival, S_i, with the same covariates as equation (2) plus an additional vector of instruments, M_i.

$$(3)\ S_i = \lambda Z_i + \zeta_{c(i)} + \varphi X_i + \mu H_i + \kappa W_i + f\left(\sum_{t=2001}^{2003} \frac{PM2.5_{i,t}}{3}\right) + \delta M_i + \upsilon_i.$$

We define M_i to include indicators for baseline diagnoses of non-smoking-related cancers (leukemia, lymphoma, and cancers of the breast, prostrate, colon, rectum, and endometrium) from the CMS's Chronic Conditions Data Warehouse file. These cancers, which affect decadal survival, are assumed to be unrelated to latent features of health that affect the probability of a dementia diagnosis.[45] In our estimation of equation (3), the instruments are jointly significant at the 1% level and individually significant at the 1% level with the exception of prostate cancer, as shown in Appendix Table A4.

We then use the survival-function residuals, $\hat{\upsilon}_i$, to define an additional control

[45] A potential concern is that non-smoking related cancers, while not causing dementia, could be correlated with dementia through other omitted factors. For example, a competing-risks framework could lead to a negative correlation between non-smoking related cancers and latent health affecting dementia and lead to an upward-biased estimate of α in our selection-correction model. Such a framework would likewise suggest that our 2SLS model provides a downward-biased estimate. On this basis, one could interpret non-smoking related cancers as "imperfect instruments," as defined by Nevo and Rosen (2012), and use them to partially identify α. The estimated identification region is then simply the interval between the 2SLS estimate and the selection-corrected estimate, i.e., [1.68, 2.33].

variable that we include in equations (1) and (2). Given the well-documented equivalence of 2SLS and control-function estimation in linear models (Hausman 1978), we estimate the following control-function equation,

$$(4)\ \Delta y_i = \alpha \sum_{t=2004}^{2013} \frac{PM2.5_{i,t}}{10} + \eta_{c(i)} + \beta X_i + \gamma H_i + \theta W_i + f\left(\sum_{t=2001}^{2003} \frac{PM2.5_{i,t}}{3}\right) + \phi_1 \hat{v}_i + \phi_2 \hat{\varepsilon}_i + \tilde{\epsilon}_i, \text{ where } \tilde{\epsilon}_i = \epsilon_i - \phi_1 \hat{v}_i - \phi_2 \hat{\varepsilon}_i.$$

$\hat{v}_i$ is the control formed by the residuals from the survival equation in (3) and $\hat{\varepsilon}_i$ is the control formed by the residuals from the first-stage equation, i.e., a modified version of equation (2) that includes $\hat{v}_i$ as an additional control. Because we estimate $\hat{v}_i$ and $\hat{\varepsilon}_i$ in prior stages, we bootstrap standard errors over all three regressions, clustering at the Census block-group level.

Table III Column (5) reports the estimate for α using equation (4) to augment our main 2SLS model and recover a point estimate that controls for selection on survival. This point estimate of 2.33 is larger than the corresponding 2SLS estimate from Table I. Likewise, the selection-corrected point estimates using our two probit models (employing a probit survival equation) increase to 2.19 and 2.29, respectively.[46] These increases are consistent with classic selection bias caused by positively correlated latent health factors. In other words, individuals who were less likely to survive the decade were also more likely to develop dementia, which is consistent with the monotone treatment selection on latent health that was used to tighten the bounds in Table III Columns (2) and (4).

B. Selection models with heterogeneous sensitivities to $PM_{2.5}$

We now extend the selection-correction model to specify potentially correlated, random coefficients on $PM_{2.5}$. We present results from models that apply the Garen (1984) framework in several novel ways. First, we implement a model that allows

[46] Van de Ven and Van Praag (1981) and Dubin and Rivers (1989) provide discussions of selection bias when the outcome equation is binary.

the unobserved heterogeneity in the sensitivity of dementia to $PM_{2.5}$ to be correlated with survival. Second, we estimate a version that allows the unobserved heterogeneity in these sensitivities to be correlated with endogenous location choice, similar to the approaches in Chay and Greenstone (2005), Bento, Friedman, and Lang (2015), and Schlenker and Walker (2017). Third, we estimate a specification that nests the prior two by allowing the unobserved heterogeneity to be correlated with both survival and endogenous location choice. Finally, we present a version of this general case where we relax one of the strong linearity assumptions of the original Garen approach.

Our first approach begins by specifying a heterogeneous coefficient on $PM_{2.5}$ exposure: $\alpha_i = \bar{\alpha} + \tau_i$ where $\bar{\alpha}$ is the population mean of α_i and τ_i captures deviations from this mean.[47] Assuming the heterogeneity in sensitivities is linearly related to latent health, $\bar{\alpha}$ can be identified by extending equation (4) to include an interaction between the survival-equation residual and decadal $PM_{2.5}$ exposure following Garen (1984) and Wooldridge (2015).[48]

$$(5)\ \Delta y_i = \bar{\alpha} \sum_{t=2004}^{2013} \frac{PM2.5_{i,t}}{10} + \eta_{c(i)} + \beta X_i + \gamma H_i + \theta W_i$$
$$+ f\left(\sum_{t=2001}^{2003} \frac{PM2.5_{i,t}}{3}\right) + \phi_1 \hat{v}_i + \phi_2 \hat{\varepsilon}_i + \psi_1 \hat{v}_i \sum_{t=2004}^{2013} \frac{PM2.5_{i,t}}{10} + \tilde{\epsilon}_i,$$
$$\text{where } \tilde{\epsilon}_i = \epsilon_i - \phi_1 \hat{v}_i - \phi_2 \hat{\varepsilon}_i - \psi_1 \hat{v}_i \sum_{t=2004}^{2013} \frac{PM2.5_{i,t}}{10}.$$

Results from this specification are shown in Table IV Column (2). The coefficient is effectively unchanged from the Heckman-selection specification with homogenous sensitivities reported in Table III, Column (5) and repeated in Table IV, Column (1), suggesting that heterogeneity in $PM_{2.5}$ sensitivities is not correlated with

[47] Heckman and Vytacil (1998), Card (2001) and Wooldridge (2003) provide discussions of this and similar approaches.
[48] Formally, the identifying assumptions are that $E[\epsilon_i | v_i, \varepsilon_i] = \phi_1 v_i + \phi_2 \varepsilon_i$, $E[\tau_i | v_i, \varepsilon_i] = \psi_1 v_i$, and that unobservables are independent of the regressors in equations (2)-(3).

survival.[49]

A separate concern is that sensitivity could correlate with residential sorting, e.g., individuals who are more sensitive to $PM_{2.5}$ may live in more polluted neighborhoods. In this case, the instrument for pollution could be correlated with the endogenous location choice, causing 2SLS to recover a local average treatment effect among a non-random subset of the population. We therefore modify equation (5) to address this concern by interacting decadal $PM_{2.5}$ exposure with the attainment-based control function instead of the survival-based control function, i.e., replacing $\psi_1 \hat{v}_i \sum_{t=2004}^{2013} \frac{PM2.5_{i,t}}{10}$ with $\psi_2 \hat{\varepsilon}_i \sum_{t=2004}^{2013} \frac{PM2.5_{i,t}}{10}$. The resulting model is similar to the specifications used in Chay and Greenstone (2005), Bento, Friedman, and Lang (2015), and Schlenker and Walker (2017) to study air pollution's effects on housing prices and hospital admissions. It provides a consistent estimator of the population-wide dementia-sensitivity to $PM_{2.5}$ in the presence of Tiebout sorting on random coefficients. Table IV Column (3) shows the results from this specification. Just as with the prior model, we find that the estimate for mean sensitivity is effectively unchanged from the Heckman-selection specification with homogeneous sensitivities shown in Table IV Column (1), suggesting that individuals were not sorting based on their sensitivity to $PM_{2.5}$.[50]

We then generalize equation (5) to allow heterogeneity in $PM_{2.5}$ sensitivity to be correlated with both survival-based sample selection and choice-based residential sorting. By including both interaction terms, $\hat{v}_i \sum_{t=2004}^{2013} \frac{PM2.5_{i,t}}{10}$ and $\hat{\varepsilon}_i \sum_{t=2004}^{2013} \frac{PM2.5_{i,t}}{10}$, this model nests the prior two specifications.[51] As shown in Table IV Column (4), we find that a 1 $\mu g/m^3$ increase in average $PM_{2.5}$ from 2004

[49] Our point estimate of ψ_1 is -0.02 (s.e. 0.08) implying that those who survived were slightly less sensitive than those who died. The average sensitivity among survivors is calculated as $\bar{\alpha} + \hat{\psi}_1 E[\hat{v}_i | S_i = 1] = 2.34 - 0.02 * 0.30 = 2.33$, which is trivially lower than the sensitivity of the population as a whole.

[50] Our point estimate of ψ_2 is 0.03 (s.e. 0.05).

[51] The third and fourth specifications also assume that the expected value of the parameter governing heterogeneity is linearly

through 2013 increases the probability of a dementia diagnosis by 2.33 percentage points, virtually identical to the estimates in Columns (1)-(3).

Finally, to weaken the assumption that heterogeneity in sensitivity is linearly related to $\hat{v}_i$ and $\hat{\varepsilon}_i$, we estimate a specification that includes interactions between $PM_{2.5}$ and fourth-degree polynomials in $\hat{v}_i$ and $\hat{\varepsilon}_i$. Table III Column (5) presents these results. They indicate that a 1-$\mu g/m^3$ increase in average $PM_{2.5}$ from 2004 through 2013 increases the probability of a dementia diagnosis by 2.59 percentage points, a slightly higher estimate than the specifications that assume the effects are linear in latent health.

TABLE IV—ESTIMATES ALLOWING FOR CORRELATED RANDOM COEFFICIENTS

	(1)	(2)	(3)	(4)	(5)
decadal $PM_{2.5}$ (1 $\mu g/m^3$)	2.334***	2.339***	2.328***	2.332***	2.586***
	(0.51)	(0.51)	(0.51)	(0.51)	(0.52)
Corrects for selection based on survival	x	x	x	x	x
α_i allowed to vary with survival only		x			
α_i allowed to vary with attainment only			x		
α_i allowed to vary with survival and attainment				x	
α_i allowed to vary flexibly with surv. and attain.					x
first-stage F statistic, survival	707	707	707	707	707
first-stage F statistic, attainment	637	637	637	637	637
number of individuals who survive through 2013	1,257,232	1,257,232	1,257,232	1,257,232	1,257,232
total number of individuals	2,384,195	2,384,195	2,384,195	2,384,195	2,384,195
share with dementia in 2013	22	22	22	22	22
share who survive through 2013	61	61	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) repeats the results from Table II Column (5). It controls for selection on mortality. Column (2) extends Column (1) to allow individuals to differ in their sensitivity to $PM_{2.5}$, with sensitivity being potentially correlated with latent factors affecting survival. Column (3) extends Column (1) to allow individuals to differ in their sensitivity to $PM_{2.5}$, with sensitivity being potentially correlated with latent factors affecting residential sorting on air pollution. Column (4) nests the models in the first three columns to control for all three mechanisms simultaneously. Column (5) extends Column (4) by relaxing the identifying linear-in-latent-health assumption underlying Column (4). Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using standard errors clustered by initial Census block group and bootstrapped over all stages of estimation.

related to the survival-function residuals and first-stage residuals: $E[\tau_i \,|v_i, \varepsilon_i] = \psi_2 \varepsilon_i$ and $E[\tau_i \,|v_i, \varepsilon_i] = \psi_1 v_i + \psi_2 \varepsilon_i$, respectively.

Overall, the collective results in Table IV suggest that classic selection on survival may cause our main estimates to be attenuated relative to the population average treatment effect. They also suggest that our main estimates are not influenced by differential sensitivities to $PM_{2.5}$ through either selection or sorting.

VI. Additional Analyses of Robustness and Validity

A. Alternative measures of dementia including for those in Medicare Advantage

Table V shows results from models that first repeat the estimation after adding individuals who self-selected out of TM and into MA and then decompose our main result into $PM_{2.5}$'s effects on different types of dementia diagnoses. Column (1) repeats our main estimate for convenience. In Column (2), we define the dependent variable as taking a prescription for the symptoms of Alzheimer's disease at any point from 2006 through 2013. This allows us to expand the sample to include individuals who exited TM at some point after 2004 to enroll in a MA plan that included prescription drug coverage. This excludes individuals on TM who did not receive drug coverage through Medicare. On net, this expands the sample by 278,395 individuals (accounting for 94% of the sample who switched to MA and survived through 2013, as shown in Figure III). The results indicate that a 1-$\mu g/m^3$ increase in average $PM_{2.5}$ over the decade increases the probability of taking an Alzheimer's drug by 9.6%, slightly larger than the percent effect observed for diagnosis rates. This demonstrates that our main results are not dependent on either the use of claims-based diagnosis or the exclusion of MA. Column (3) maximizes the sample by defining the dependent variable as having either a claims-based diagnosis or a claim for a prescription drug to treat symptoms of Alzheimer's disease. The net effect of expanding the sample and altering the measure of dementia is to lower the sample dementia rate in 2013 to 21%. The resulting 2SLS coefficient, 1.69 pp, is nearly identical to our main 2SLS estimate, giving further evidence that

this estimate is not biased by selection into Medicare Advantage.

Columns (4) and (5) repeat the estimation of equation (1) for the TM sample after stratifying the dependent variable to decompose the relative impacts on dementia cases with and without an associated diagnosis of Alzheimer's disease. Our decomposition suggests that Alzheimer's accounts for 64% of the dementia cases that our 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 and other forms of dementia without an autopsy or extensive brain imaging, leaving some doctors reluctant to diagnose living patients with Alzheimer's specifically, as opposed to dementia generally.

TABLE V—ESTIMATES USING ALTERNATIVE MEASURES OF DEMENTIA

	(1)	(2)	(3)	(4)	(5)	(6)
decadal $PM_{2.5}$ (1 μg/m^3)	1.679*** (0.49)	1.203** (0.55)	1.692*** (0.46)	0.611 (0.38)	1.068*** (0.39)	1.696*** (0.48)
dependent variable	claim-based diagnosis	dementia drug	claim-based diagnosis or drug	claim-based diagnosis without Alzheimer's	claim-based diagnosis with Alzheimer's	claim-based diagnosis
first-stage F statistic	637	571	718	637	637	637
total number of individuals	1,257,232	1,044,271	1,535,746	1,257,232	1,257,232	1,257,232
% in traditional Medicare in 2013	100	73	82	100	100	100
% in Medicare Advantage in 2013	1	28	19	1	1	1
dependent variable mean	22	12	21	12	10	22

Note: Col (1) repeats the main specification from Table I. Col (2) defines the dependent variable to be 100 for individuals who took a prescription drug for Alzheimer's disease and alters the sample to include everyone who had drug coverage through Medicare including those on Medicare Advantage. Col (3) defines the dependent variable to be 100 for individuals who are diagnosed with dementia and/or take prescription drugs for Alzheimer's disease. Col (4) is the same as (1) but defines the dependent variable as dementia cases without an Alzheimer's diagnosis and Col (5) defines it as Alzheimer's disease specifically. Col (6) is the same as (1) but adds an indicator for whether individuals had a stroke by 2013. Summing the percentages of individuals enrolled in traditional Medicare and Medicare Advantage rounds to just over 100% because a small fraction of individuals switched between the two programs in 2013. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered by initial Census block group.

As a further test of which types of dementia drive our results, we repeat 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, 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. 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 (6). This suggests that long-term exposure to $PM_{2.5}$ increases the risk of Alzheimer's disease, specifically.

B. Alternative measures of $PM_{2.5}$ exposure

Table VI summarizes results from alternative approaches to measuring $PM_{2.5}$ exposure. 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. As in our main specification, each indicator is interacted with the fourth-order polynomial function of baseline exposure. This yields an estimate of 1.67 pp, nearly the same as our main estimate in Column (1).[52]

[52] Appendix Figure A5 shows the estimated partial effects of each interaction. We find patterns consistent with strategic regulatory targeting. Our estimates suggest that nonattainment designations produced slight increases in $PM_{2.5}$ for people in attainment counties living near nonattainment monitors in adjacent counties. This pattern could result from regulatory actions diverting pollution from areas near nonattainment monitors to areas in adjacent attainment counties (e.g., siting of new production facilities).

TABLE VI—ESTIMATES USING ALTERNATIVE MEASURES OF $PM_{2.5}$ EXPOSURE

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
decadal $PM_{2.5}$ (1 μg/m^3)	1.679***	1.666***	1.262***	1.658***	1.650***	1.855***	1.921***
	(0.49)	(0.43)	(0.49)	(0.51)	(0.49)	(0.54)	(0.54)
baseline specification	x						
IV = county x monitor attainment		x					
unbalanced monitor panel			x				
5-digit ZIP assignment of $PM_{2.5}$				x			
spline function of baseline $PM_{2.5}$					x		
exposure fixed at post-diagnosis move						x	
exposure fixed at diagnosis							x
first-stage F statistic	637	491	395	645	135	515	476
number of individuals	1,257,232	1,257,232	1,257,232	1,257,232	1,257,232	1,257,232	1,257,232
share with dementia in 2013	22	22	22	22	22	22	22

Note: Col (1) repeats our main result that is modified for each remaining Column. Column (2) stratifies 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) replaces our preferred measure of pollution (based on a balanced panel of continuously operating monitors) with data from an unbalanced panel of all monitors in operation each year. Column (4) measures pollution at the coarser 5-digit ZIP code level. Column (5) 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. Column (6) stops tracking cumulative exposure among dementia patients at the time they move to new residences. Col (7) strops tracking cumulative exposure at the point when we first observe their dementia diagnosis.

Column (3) 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. We find that using the unbalanced panel reduces the instrument's power to explain decadal $PM_{2.5}$ exposures in the first stage and yields a smaller but still economically and statistically significant second-stage estimate of 1.26 pp.

In Column (4) 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. The

estimated effect of $PM_{2.5}$ on dementia is 1.66 pp, virtually identical to our main result.

Column (5) 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. This again produces a similar $PM_{2.5}$ coefficient (1.65 pp).

A remaining concern is that our estimates could reflect reverse causality via Tiebout sorting if dementia diagnoses cause people to move to more polluted areas, e.g., if assisted living facilities tend to be in more polluted areas. We test this hypothesis by fixing annual average exposure at the point of an individual's first post-diagnosis move. For example, if an individual is diagnosed with dementia in 2010 and moves to a new residence in 2012, then we replace the decadal measure of their $PM_{2.5}$ exposure with their annual average exposure from 2004 through 2011. Column (6) shows that this approach increases our estimate slightly. This is the opposite of what would be implied by reverse causality. It results from the fact that individuals who move with dementia tend to move to less-polluted areas than individuals who move without dementia.[53] Column (7) takes this logic one step further by fixing dementia patients' cumulative exposures in their diagnosis years so that, in the prior example, we would use annual average exposure from 2004-2010. Once again, the coefficient increases slightly, further reinforcing that our main approach to measuring pollution exposure does bias our estimates upward.

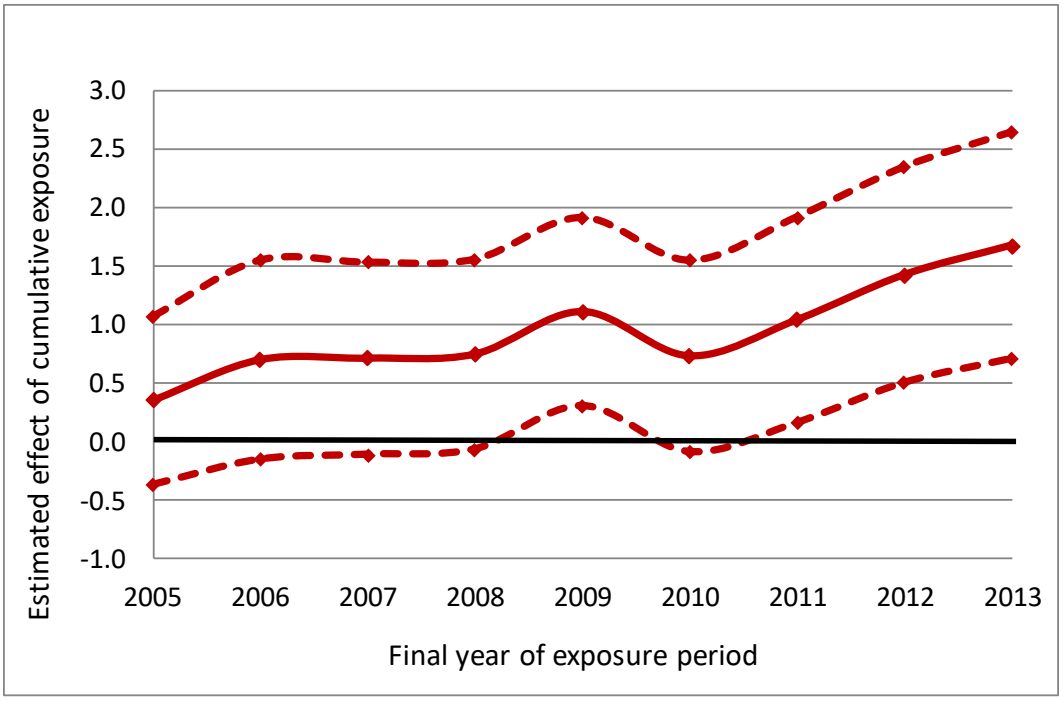
C. Alternative measures of air pollution

Appendix Table A5 shows that our linear 2SLS results for $PM_{2.5}$ are robust to additionally conditioning on other federally regulated air pollutants: coarse particulate matter (PM_{10}), ozone (O_3), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and

[53] Appendix Figure A6 shows exposure conditional on migration status and dementia diagnosis.

carbon monoxide (CO). Specifically, we extend the covariate set to include fourth-order polynomial functions of baseline concentrations of each pollutant while treating subsequent decadal exposures to all six pollutants as endogenous. Similar to Bento, Friedman, and Lang (2015), this relies on the instrument vector described in Section VI.B to obtain an overidentified model.[54] The resulting coefficient on $PM_{2.5}$ is virtually unchanged, and we fail to reject the hypothesis that PM_{10}, O_3, NO_2, SO_2, and CO jointly have no additional effect on dementia at the 10% level.

FIGURE VII: ESTIMATED EFFECTS OF $PM_{2.5}$ ON DEMENTIA BY EXPOSURE DURATION



D. Alternative exposure durations

We focus on decadal $PM_{2.5}$ exposure because 10 years is the longest interval over which our research design and data enable us to identify an effect, but it is

[54] For consistency we measure concentrations of each pollutant using the same balanced panel of monitoring stations that we use to construct measures of $PM_{2.5}$.

straightforward to use the same design to estimate effects for shorter intervals. To examine how $PM_{2.5}$'s effect on the probability of a dementia diagnosis varies with exposure duration, we estimate models for two years to ten years of exposure (i.e., from 2004-2005, 2004-2006,… , 2004-2013). These estimates are from models that parallel our main 2SLS specification but replace the decadal exposure measure with a shorter integer-year duration. At the two-year mark in 2005 the estimation sample includes 2.4 million individuals. As we move from 2005 to 2013, the sample diminishes due to death and switching into Medicare Advantage.

Figure VII shows our estimates for the effects of 1 $\mu g/m^3$ increases in average residential concentrations from 2004 to the interval endpoints shown on the horizontal axis, along with 95% confidence intervals. The estimates increase steadily with exposure duration and remain statistically distinguishable from zero beyond the eighth year (2011). Appendix figure A7 shows that the figure looks nearly the same when we reconstruct it after restricting the sample to individuals who survived to 2013; i.e., holding the longitudinal sample fixed as we adjust exposure duration. This comparison reinforces our conclusion that our main findings are not biased away from zero due to attrition from death and transition to MA.

E. Placebo tests

As previously discussed, we test whether pollution has a causal impact on death among the Medicare population. Specifically, using the 2SLS specification in equations (1) and (2) with decadal mortality as the dependent variable, we find that a 1-$\mu g/m^3$ increase in average $PM_{2.5}$ from 2004 through 2013 increases mortality by 2.37 percentage points, equivalent to 6% of the decadal mortality rate. The full results of this specification are shown in Table A3 in the appendix. The consistency between this finding for long-term exposure and the results from well-identified mortality effects from short term spikes in $PM_{2.5}$ (e.g., Deryugina et al. 2019) lends credence to our research design.

We estimate an additional set of traditional placebo models designed to test whether unspecified threats to identification cause spurious positive relationships between pollution and the onset of poor health generally. We examine five chronic conditions that are not known or suspected to be caused by air pollution but 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. These include glaucoma, fibromyalgia, breast cancer, prostate cancer, and peripheral vascular disease. Glaucoma is a progressive disorder with nerve degeneration that is strongly associated with age; fibromyalgia affects mood and behavior and can be difficult to diagnose; breast cancer and prostate cancer can be slow to progress and have gender-specific diagnosis rates; and peripheral vascular disease is associated with reduced blood circulation. Conditional on age and gender, dementia, glaucoma, and peripheral vascular disease are all more common among African-Americans and Hispanics relative to non-Hispanic whites.[55]

TABLE VII—ESTIMATES OF $PM_{2.5}$ ON PLACEBO HEALTH OUTCOMES

	Dementia in 2013	Glaucoma	Fibro-myalgia	Breast cancer	Prostate cancer	Peripheral vascular disease
decadal $PM_{2.5}$	1.679***	-1.026*	-0.465	-0.077	-0.189	0.581
($1\ \mu g/m^3$)	(0.49)	(0.53)	(0.53)	(0.21)	(0.23)	(0.60)
first-stage F statistic	637	600	626	624	625	631
number of individual	1,257,232	1,065,603	1,182,076	1,248,239	1,249,959	1,186,008
share with outcome	22	17	18	3	4	27

Note: the first column repeats our main result for comparison. The next six columns report results using the same model but replacing dementia with each of the placebos. Asterisks indicate statistical significance at the 10%, 5%, and 1% levels based on robust standard errors clustered by Census block group.

[55] The placebo model samples are slightly smaller than our main dementia sample. 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%).

Finding large, positive, and statistically significant effects of $PM_{2.5}$ on these placebo morbidities would signal that our 2SLS research design may be compromised. Table VII shows that this is not the case. We fail to reject the null hypothesis of zero effect at the 5% significance level for each placebo outcome. Our criteria for selecting the placebos in Table VII excludes cardiopulmonary conditions and other illnesses that have previously been linked to air pollution. When we instead ignore these criteria and repeat estimation of our main specification for each of the 15 most common chronic conditions among the Medicare population (Centers for Medicare and Medicaid Services 2012) including those linked to pollution exposure, we find a positive effect of $PM_{2.5}$ at the 5% level for only one disease besides dementia: chronic obstructive pulmonary disease. This reinforces findings from prior large cohort studies that found $PM_{2.5}$ to cause and exacerbate COPD (e.g., Guo et al. 2018).[56]

The last column of Table V summarizes a final placebo specification that repeats 2SLS estimation on a larger sample using a dementia diagnosis in 2004 as the outcome. Anticipatory Tiebout sorting on factors that contribute to dementia and are correlated with $PM_{2.5}$ but are not accounted for by our model could yield a relationship between dementia in 2004 and $PM_{2.5}$ exposure over the subsequent decade. However, this is not the case. The resulting coefficient is negative, close to zero, and estimated relatively precisely.[57] As with our observed effects on mortality, the lack of effects on placebo outcomes affirms the credibility of using our research design to draw causal inferences about the effects of $PM_{2.5}$ on dementia.

[56] We leave a comprehensive analysis of $PM_{2.5}$ on morbidity to future research.

[57] Figure A8 provides the informal visual inspection confirming parallel pre-treatment trends in diagnoses rates the spirit of Autor (2003). While parallel pre-trends is neither necessary nor sufficient for drawing causal inference from our 2SLS research design, the absence of pre-instrument 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 prescribing decisions.

VIII. Welfare Implications and Conclusion

Our findings provide the first large-scale, nationwide evidence to support the hypothesis from medical research that long-term exposure to fine-particulate air pollution increases the individual risk of dementia among older adults. Our results show that $PM_{2.5}$'s effect on dementia is driven by cumulative exposure and that this effect is not explained by other pollutants, selection on mortality, sorting between traditional Medicare and Medicare Advantage, residential sorting based on anticipating future pollution changes, or other forms of Tiebout sorting based on unobserved health, genetics, income, and preferences for neighborhood amenities.

Dementia's global social costs continue to grow with the aging populations of many countries, causing the World Health Organization to label it a "public health priority" and the US Centers for Disease Control to describe it as a "public health crisis." Because no medical preventions or cures exist, policy discussions have focused on investment in research and health infrastructure and modifying behaviors related to smoking, diet and exercise (World Health Organization 2012, US Centers for Disease Control and Prevention 2018). Our findings reveal another lever available to policy makers. We show that EPA regulation of $PM_{2.5}$ during the 2000s lowered dementia rates in the United States. Specifically, we estimate that county nonattainment designations determined in late 2003 and early 2004 lowered average $PM_{2.5}$ across the subsequent decade in nonattainment counties by 1.24 $\mu g/m^3$. Multiplying this reduction by our main estimate for the effect of a 1 $\mu g/m^3$ increase in decadal exposure on the probability of a dementia diagnosis (1.68 pp) implies that the regulation reduced the dementia rate in nonattainment counties by 2.1 pp, amounting to 182,000 fewer dementia cases among individuals age 75 and above in nonattainment counties in 2013.[58]

[58] This value comes from scaling our estimates by Census data indicating that 8.7 million people age 75 and over lived in counties in 2013 that were nonattainment in 2005.

To provide insights to the magnitude of the monetary value of these avoided cases, we combine our data with estimates from prior estimates of the value of quality-adjusted life years. Appendix C provides details of our approach. To summarize, we estimate that for individuals over age 75, a new dementia diagnosis is equivalent to losing 7.4 years of life in otherwise average health without dementia. Eighty-three percent of this loss is due to lower life expectancy and seventeen percent comes from reduced quality of remaining life. Assuming a value per statistical life year of $160,000 for individuals over 75 in average health without dementia implies that each case avoided is worth about $1.2 million, generating benefits of $214 billion for the age 75+ cohort living in nonattainment counties in 2013.[59]

In addition to these economic implications of US environmental policy, the finding that air pollution elevates the risk of dementia has implications for household finance and retirement planning. First, having dementia has been found to alter the process and outcomes of individuals' financial decisions (e.g., Keane and Thorp 2015, Keane et al. 2020). Second, an individual's prior exposure to $PM_{2.5}$ is an important variable for predicting her future risk of dementia. Better predictions can help individuals improve their financial decisions, as prior research has shown that individuals' expectations about their likelihood of acquiring dementia influence retirement planning, savings, and the purchase of long-term care insurance (Agarwal et al. 2009, Shin, Lillard, and Bhattacharya 2019).

[59] With a 3% discount rate, $160,000 is less than half the value per life year implied by the constant value of a statistical life used by the EPA to calculate the benefits of regulating air pollution to reduce mortality among the Medicare population (Aldy and Viscusi 2007). In comparison, Murphy and Topel (2006) report that an average 80 year old has a VSLY around $200,000 (in 2018 dollars). In any case, our benefit measures can be easily rescaled by alternative assumptions for the value of statistical life years, but will remain large enough to matter for evaluating air quality regulations under any estimates from the current literature.

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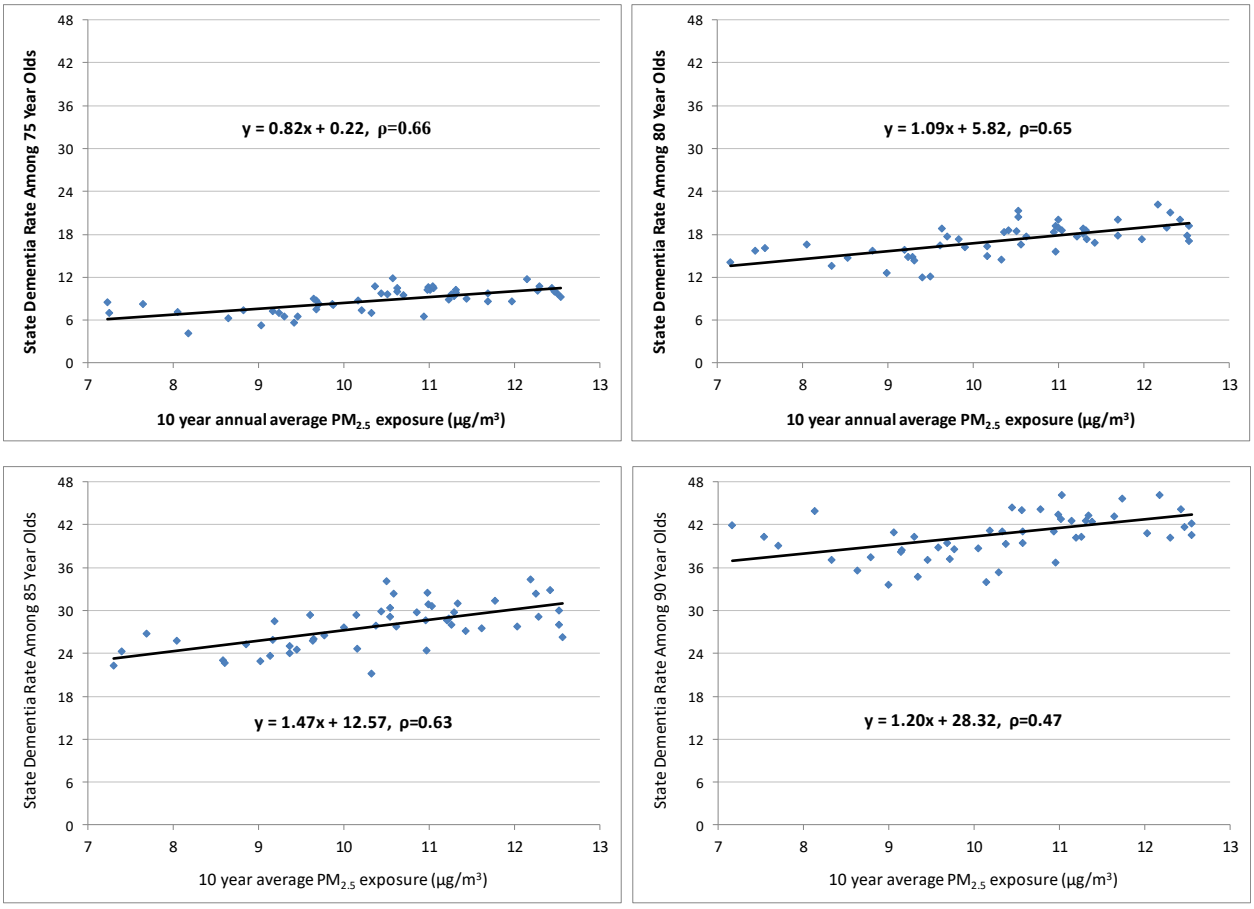
Supplemental Material: For Online Publication Only

SUPPLEMENTAL APPENDIX A: ADDITIONAL TABLES AND FIGURES

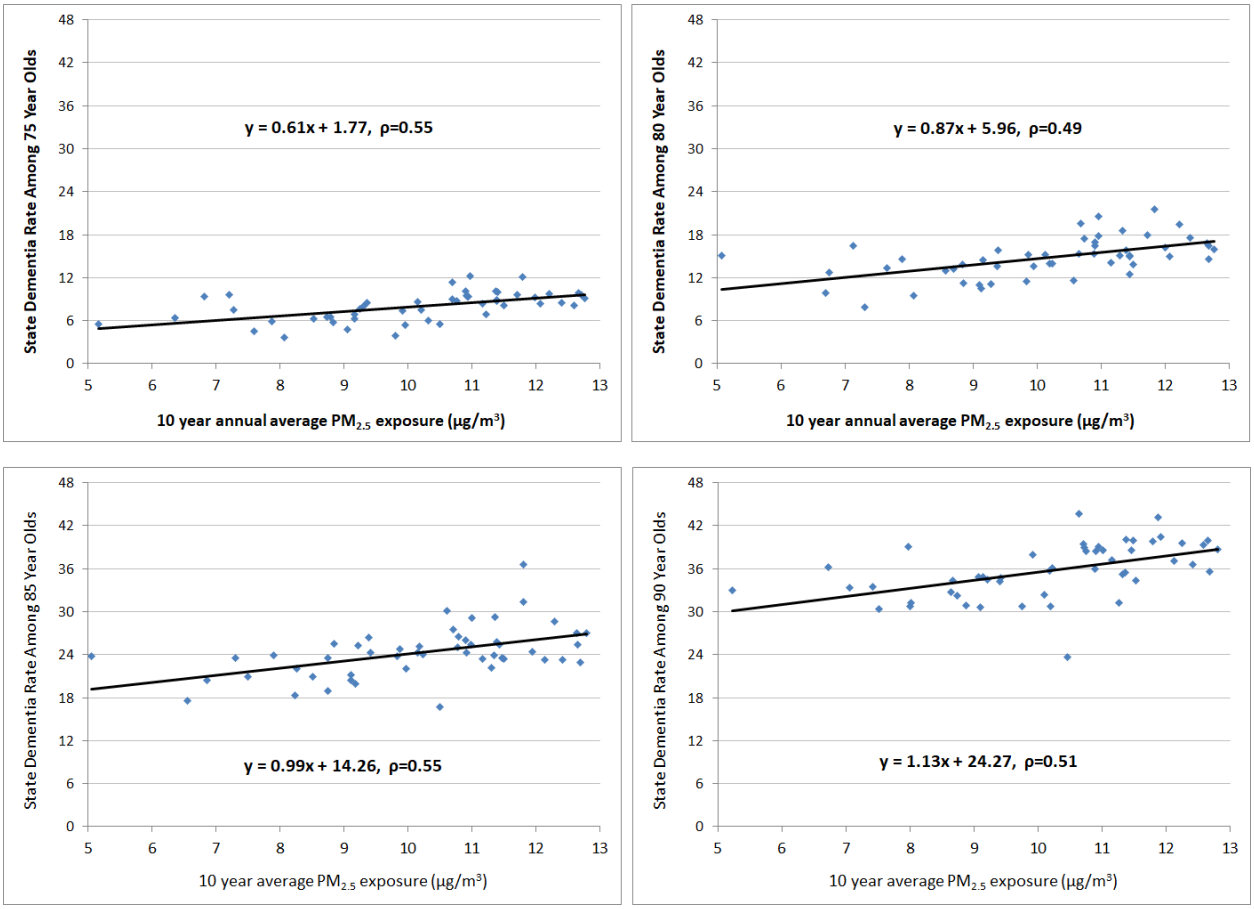
FIGURE A1: ASSOCIATION BETWEEN $PM_{2.5}$ AND DEMENTIA AMONG MEDICARE ENROLLEES, 2013



Note: Each data point represents the fraction of individuals living in a state who had been diagnosed with dementia prior to the end of 2013 plotted against their average decadal exposure to $PM_{2.5}$ based on place of residence. The figures are conditional on integer age: 75 (upper left), 80 (upper right), 85 (lower left) and 90 (lower right). Each figure also shows linear regression equations and correlation coefficients. The figures are based on dementia diagnoses observed for all enrollees in traditional Medicare in 2013.

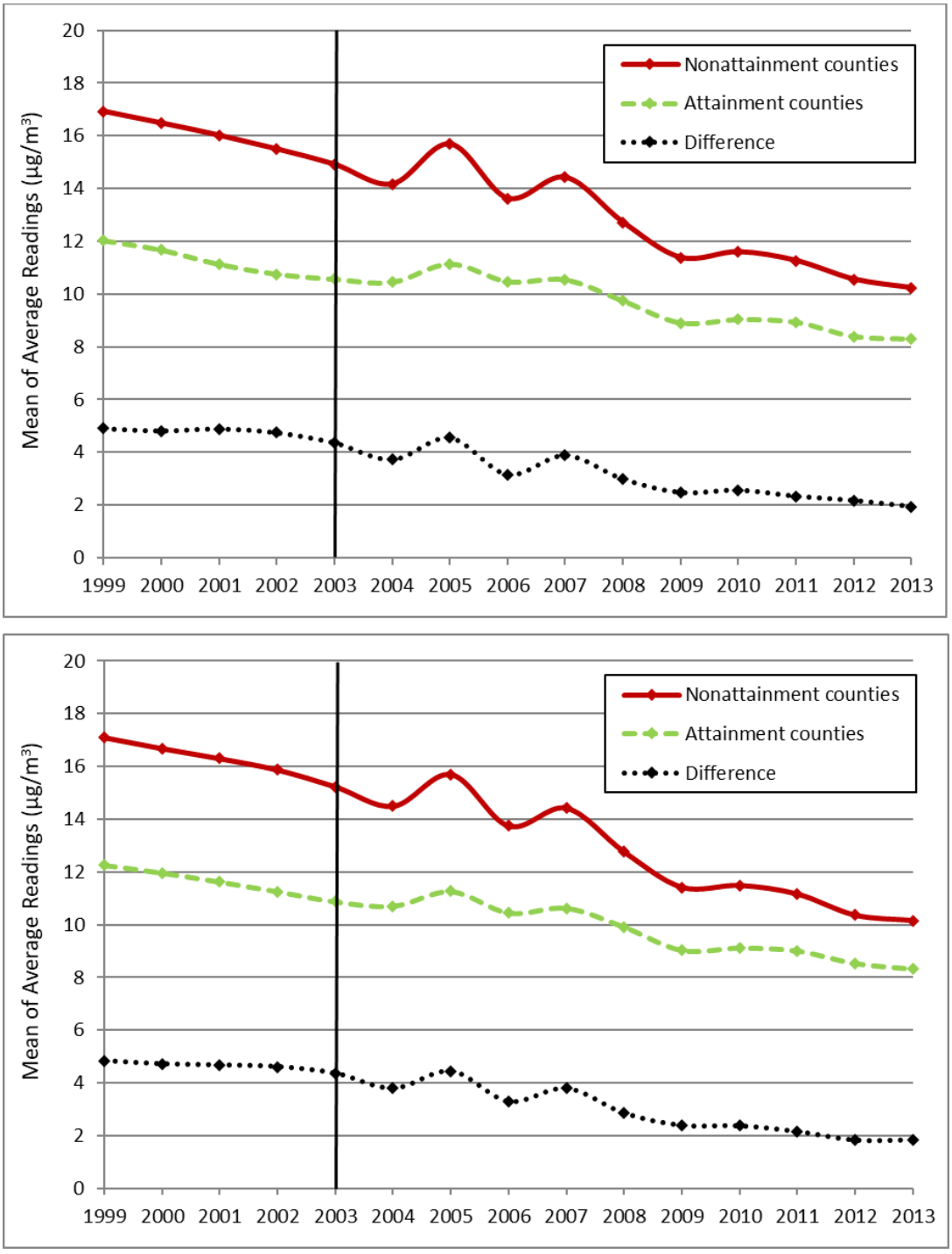
FIGURE A1 (CONTINUED):

ASSOCIATION BETWEEN $PM_{2.5}$ AND DEMENTIA IN MAIN ESTIMATION SAMPLE, 2013



Note: The figure is the same as the prior figure, except that it is constructed using only the individuals included in our main estimation sample. Differences between Figures A1 and A2 are mainly due to dropping individuals living in counties without pollution monitors.

FIGURE A2: AIR POLLUTION TRENDS: UNBALANCED AND BALANCED MONITOR PANELS



The bottom figure is identical to Figure II. It displays air pollution trends based on a balanced panel of monitors in operation continuously from 2001-2013. For comparison, the top figure is based on averages taken each year over an unbalanced panel of operating monitors.

TABLE A1: SUMMARY STATISTICS FOR MEDICARE BENEFICIARY SAMPLES

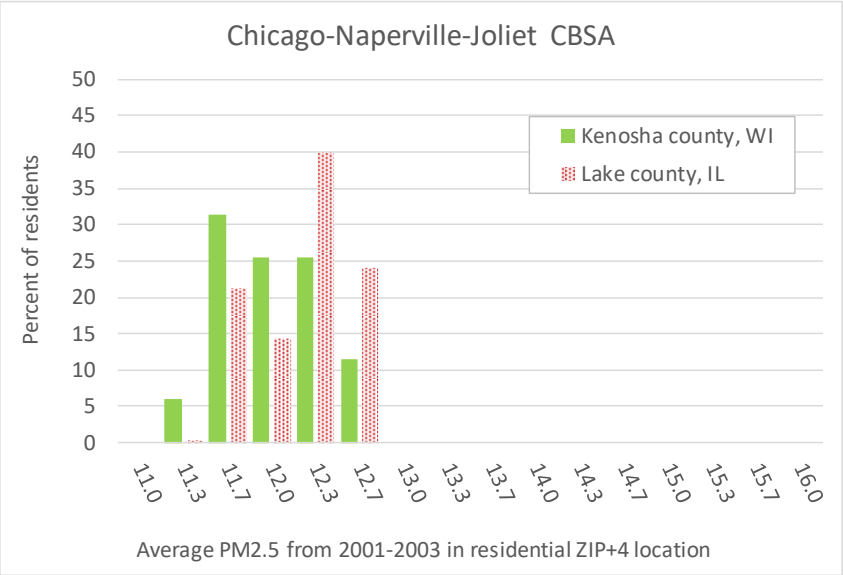
	(1)	(2)	(3)	(4)	(5)	(6)
	Main estimation sample: 2004 - 2013 survivors	Full estimation sample: traditional Medicare enrollees in 2004	EXCLUDED lived in county without pollution monitors	EXCLUDED enrolled in Medicare Advantage in 2004	EXCLUDED had dementia in 2004	EXCLUDED missing data or moved in 2004
# people	1,257,232	2,384,195	2,695,762	772,071	339,539	418,067
Individual demographics						
mean age at sample entry	69.5	71.1	71.3	71.3	77.3	69.2
mean age in 2013	82.8	84.5	84.7	84.8	91.2	82.0
male (%)	38	41	43	41	32	48
white (%)	83	83	87	75	80	77
black (%)	8	9	6	10	11	10
asian (%)	3	3	1	4	2	4
hispanic (%)	5	5	6	10	6	8
alive at beginning of 2013 (%)	100	65	60	64	20	74
ever moved (%)	31	31	36	36	52	67
ever moved county (%)	17	16	21	20	29	51
ever moved state (%)	10	10	15	12	19	37
2013 gross Medicare expenditures ($)	4,838	6,726	7,101		16,265	
Medical diagnoses as of 2004						
dementia (%)	0	0	10		100	
stroke (%)	7	10	11		34	
congestive heart failure (%)	13	21	21		45	
diabetes (%)	22	25	23		34	
ischemic heart disease (%)	36	42	37		61	
hypertension (%)	67	70	63		84	
Neighborhood characteristics						
$PM_{2.5}$ (hourly $\mu g/m^3$) 2001-2003	13.24	13.29	12.86	13.57	13.39	
Nonattainment county (%)	39.99	39.50		42.32	42.25	
household income (median)	65,387	62,041	52,738	60,424	59,800	
income per capita	33,498	31,822	26,815	29,954	31,095	
year built (median)	1970	1969	1973	1967	1968	
house value (median)	265,944	246,780	170,730	278,731	244,764	
house value (average)	136,748	124,553	88,543	132,277	119,108	
gross rent (median)	2,807	2,546	1,723	2,281	2,361	
population over 65 (%)	18	18	19	18	19	
population white not hispanic (%)	68	67	83	58	64	
population black (%)	12	13	7	12	15	
population hispanic (%)	13	13	7	21	14	
education: 9th to 12th (%)	7	8	9	8	8	
education: high school grad (%)	27	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 (%)	27	28	23	31	32	

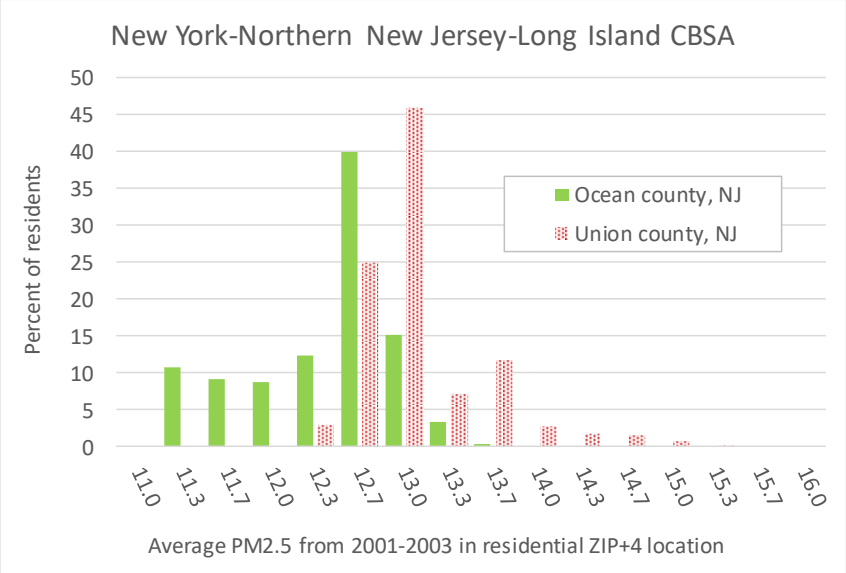
Note: Column (1) describes the sample used in our main longitudinal models. It is a balanced panel of individuals who were in traditional Medicare (TM) in 2004 and survived to 2013, at which point they were still enrolled in TM. Column (2) describes the full estimation sample used in models that include individuals who were in TM in 2004 but died or switched to Medicare Advantage (MA) before 2013. Column (3) describes individuals who were in TM in 2004 but not used in estimation because they lived in counties that were designated by EPA as "unclassifiable" for regulatory purposes due to a lack of pollution monitors. 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 use din 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.

FIGURE A3: LOCATIONS OF EPA MONITORING STATIONS FOR FINE PARTICULATE MATTER

The map shows the locations of air quality monitors for particulate matter smaller than 2.5 microns in diameter ($PM_{2.5}$). The maps 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 A4: WITHIN-CBSA VARIATION IN NONATTAINMENT STATUS BY BASELINE $PM_{2.5}$ LEVELS





The figures provide examples of within-county and between-county variation in nonattainment status conditional on baseline residential $PM_{2.5}$ concentrations from 2001-2003 in two CBSAs. The vertical axes report the fractions of individuals in 0.33 microgram per cubic meter bins describing baseline $PM_{2.5}$ concentrations for residential areas in specific nonattainment and attainment counties at the time nonattainment designations were made. For example, the bottom figure shows that about 45% of individuals living in Union county, New Jersey in 2004 were living in neighborhoods

that had baseline concentrations between 13.0 and 13.3 micrograms per cubic meter. The corresponding fraction in Ocean county, New Jersey was about 15%. Both counties are part of the New York – Northern New Jersey – Long Island CBSA but differed in their regulatory designations. Union county contains monitors above and below the regulatory threshold whereas all of Ocean county's monitors were below the threshold.

The top figure compares two adjacent counties in the Chicago – Naperville – Joliet CBSA. While Lake county's monitors were below the regulatory threshold it was designated as a nonattainment county. This illustrates the fact that the EPA designated counties as nonattainment if they were believed to contribute to violations in other nearby counties due to spatial dispersion of emissions.

TABLE A2.A: SECOND STAGE RESULTS FROM THE MAIN 2SLS SPECIFICATION

	coefficient	Robust standard error	95% Confidence Interval	
$PM_{2.5}$ (1 μg/m^3) (Decadal, 2004-2013)	1.679	0.490	0.717	2.640
Chronic conditions in 2004				
H	0.769	0.094	0.586	0.953
S	8.018	0.571	6.899	9.138
S, H	9.130	0.344	8.455	9.805
D	3.291	0.266	2.771	3.812
D, H	3.592	0.149	3.300	3.884
D, S	14.072	1.857	10.433	17.711
D, S, H	13.438	0.607	12.248	14.629
I	2.101	0.183	1.743	2.459
I, H	2.598	0.124	2.356	2.840
I, S	9.854	0.832	8.223	11.485
I, S, H	11.059	0.338	10.396	11.722
I, D	4.653	0.460	3.752	5.553
I, D, H	5.591	0.175	5.247	5.935
I, D, S	8.609	1.967	4.754	12.464
I, D, S, H	14.605	0.483	13.658	15.552
C	4.293	0.596	3.124	5.462
C, H	4.232	0.314	3.616	4.848
C, S	9.136	2.702	3.841	14.432
C, S, H	12.714	1.027	10.701	14.726
C, D	8.217	1.544	5.191	11.244
C, D, H	8.289	0.460	7.388	9.191
C, D, S	18.205	6.093	6.262	30.147
C, D, S, H	18.227	1.414	15.456	20.999
C, I	4.079	0.521	3.057	5.100
C, I, H	5.383	0.205	4.981	5.785
C, I, S	9.891	1.780	6.402	13.381
C, I, S, H	13.613	0.485	12.663	14.563
C, I, D	7.987	1.097	5.837	10.136
C, I, D, H	9.245	0.243	8.769	9.721
C, I, D, S	20.333	3.847	12.792	27.874
C, I, D, S, H	20.552	0.525	19.523	21.580

Note: The chronic conditions in 2004 are hypertension (H), stroke (S), diabetes (D), ischemic heart disease (I), and congestive heart failure (C).

TABLE A2.A (CONT'D): SECOND STAGE RESULTS FROM THE MAIN 2SLS SPECIFICATION

	coefficient	Robust standard error	95% Confidence Interval	
2004 Gross Medicare Expenditures ($10,000)				
expenditures	3.859	0.119	3.625	4.092
expenditures^2	-0.484	0.033	-0.548	-0.421
expenditures^3	0.019	0.002	0.015	0.023
expenditures^4	0.000	0.000	0.000	0.000
Age (females)				
76	0.751	0.180	0.398	1.104
77	1.689	0.188	1.321	2.057
78	2.907	0.194	2.526	3.288
79	4.307	0.202	3.911	4.704
80	6.025	0.214	5.606	6.444
81	7.152	0.216	6.729	7.576
82	8.838	0.227	8.393	9.283
83	11.253	0.233	10.797	11.710
84	12.696	0.242	12.222	13.170
85	15.244	0.252	14.751	15.737
86	17.625	0.262	17.112	18.138
87	19.841	0.277	19.299	20.384
88	22.560	0.290	21.992	23.127
89	25.081	0.305	24.483	25.679
90	27.224	0.330	26.576	27.871
91	29.571	0.355	28.875	30.267
92	31.013	0.375	30.278	31.747
93	33.346	0.418	32.528	34.165
94	36.313	0.481	35.370	37.256
95	38.380	0.530	37.342	39.419
96	40.681	0.611	39.484	41.878
97	42.037	0.709	40.647	43.427
98	43.329	0.822	41.717	44.940
99	47.367	0.918	45.568	49.167
100 and over	46.058	0.687	44.712	47.403

Note: The excluded reference category for age is 75.

TABLE A2.A (CONT'D): SECOND STAGE RESULTS FROM THE MAIN 2SLS SPECIFICATION

	coefficient	Robust standard error	95% Confidence Interval	
male	-0.837	0.186	-1.201	-0.473
Age (males)				
76	-0.063	0.270	-0.592	0.466
77	-0.245	0.281	-0.795	0.305
78	-0.557	0.292	-1.130	0.016
79	-0.869	0.304	-1.464	-0.273
80	-1.310	0.322	-1.941	-0.678
81	-1.495	0.327	-2.136	-0.854
82	-1.469	0.343	-2.142	-0.796
83	-2.247	0.355	-2.942	-1.552
84	-1.912	0.373	-2.643	-1.181
85	-2.530	0.394	-3.303	-1.758
86	-2.604	0.413	-3.413	-1.794
87	-3.697	0.440	-4.560	-2.833
88	-3.976	0.470	-4.897	-3.055
89	-4.283	0.501	-5.265	-3.302
90	-4.555	0.550	-5.633	-3.476
91	-5.861	0.593	-7.023	-4.700
92	-4.591	0.651	-5.867	-3.314
93	-5.226	0.738	-6.671	-3.780
94	-6.498	0.869	-8.200	-4.796
95	-7.181	0.998	-9.137	-5.225
96	-7.097	1.179	-9.409	-4.786
97	-6.282	1.446	-9.115	-3.448
98	-7.976	1.731	-11.370	-4.583
99	-11.812	2.136	-15.999	-7.625
100 and over	-9.463	1.653	-12.703	-6.224

Note: The excluded reference category for age is 75.

TABLE A2.A (CONT'D): SECOND STAGE RESULTS FROM THE MAIN 2SLS SPECIFICATION

	coefficient	Robust standard error	95% Confidence Interval	
White	0.803	0.353	0.110	1.495
Black	3.718	0.392	2.951	4.486
Asian	0.517	0.410	-0.287	1.321
Hispanic	3.432	0.394	2.660	4.204
2004 Census Block Group Demographics				
median household income / 1000	-0.004	0.003	-0.009	0.001
per capita income / 1000	-0.010	0.004	-0.018	-0.001
median year built	0.002	0.003	-0.003	0.007
median house value / 1000	-0.002	0.000	-0.003	-0.002
average house value / 1000	0.000	0.000	0.000	0.000
median gross rent / 1000	0.020	0.007	0.007	0.034
% over 65	0.235	0.382	-0.513	0.982
% white	1.185	0.438	0.327	2.043
% black	2.434	0.484	1.485	3.383
% hispanic	1.057	0.506	0.065	2.050
% 9th through 12th	-0.158	1.185	-2.480	2.164
% high school graduate	-3.933	0.903	-5.703	-2.163
% some college	-5.971	0.899	-7.733	-4.209
% associate degree	-7.511	1.134	-9.733	-5.289
% bachelor's degree	-5.759	0.907	-7.536	-3.982
% graduate degree	-5.284	0.960	-7.165	-3.403
% owner occupied	-2.484	0.414	-3.295	-1.673
% renter occupied	1.908	0.462	1.002	2.814
$PM_{2.5}$ (1 μg/m^3) (Baseline, 2001-2003)				
exposure	-1.853	1.697	-5.179	1.474
$exposure^2$	0.156	0.185	-0.206	0.518
$exposure^3$	-0.010	0.009	-0.026	0.007
$exposure^4$	0.000	0.000	0.000	0.000

Note: The excluded reference categories are "other" for race, "% with 8th grade or less" for block group education attainment, and "% vacant" for block group housing stock.

TABLE A2.B: FIRST STAGE RESULTS FROM THE MAIN 2SLS SPECIFICATION

	coefficient	Robust standard error	95% Confidence Interval	
Chronic conditions in 2004				
H	-0.0024	0.0008	-0.0040	-0.0007
S	0.0010	0.0048	-0.0085	0.0104
S, H	0.0006	0.0024	-0.0042	0.0054
D	-0.0051	0.0024	-0.0099	-0.0004
D, H	-0.0048	0.0012	-0.0071	-0.0024
D, S	-0.0033	0.0121	-0.0270	0.0203
D, S, H	-0.0007	0.0042	-0.0089	0.0074
I	-0.0022	0.0017	-0.0055	0.0011
I, H	-0.0026	0.0011	-0.0047	-0.0005
I, S	-0.0059	0.0065	-0.0187	0.0069
I, S, H	-0.0017	0.0024	-0.0065	0.0031
I, D	-0.0042	0.0038	-0.0116	0.0033
I, D, H	-0.0055	0.0014	-0.0083	-0.0027
I, D, S	-0.0246	0.0162	-0.0564	0.0071
I, D, S, H	-0.0026	0.0036	-0.0096	0.0044
C	-0.0012	0.0046	-0.0103	0.0079
C, H	-0.0031	0.0026	-0.0082	0.0019
C, S	-0.0401	0.0231	-0.0853	0.0052
C, S, H	-0.0095	0.0075	-0.0241	0.0052
C, D	-0.0041	0.0126	-0.0289	0.0206
C, D, H	-0.0011	0.0035	-0.0080	0.0058
C, D, S	-0.0321	0.0329	-0.0966	0.0324
C, D, S, H	-0.0016	0.0114	-0.0238	0.0207
C, I	0.0059	0.0042	-0.0024	0.0141
C, I, H	0.0011	0.0017	-0.0022	0.0044
C, I, S	-0.0029	0.0139	-0.0301	0.0243
C, I, S, H	-0.0026	0.0038	-0.0101	0.0048
C, I, D	0.0050	0.0088	-0.0121	0.0222
C, I, D, H	-0.0002	0.0019	-0.0040	0.0035
C, I, D, S	0.0273	0.0326	-0.0367	0.0913
C, I, D, S, H	-0.0007	0.0041	-0.0086	0.0073

Note: The chronic conditions in 2004 are hypertension (H), stroke (S), diabetes (D), ischemic heart disease (I), and congestive heart failure (C).

TABLE A2.B (CONT'D): FIRST STAGE RESULTS FROM THE MAIN 2SLS SPECIFICATION

	coefficient	Robust standard error	95% Confidence Interval	
2004 Gross Medicare Expenditures ($10,000)				
expenditures	0.0001	0.0009	-0.0017	0.0019
expenditures^2	-0.0001	0.0002	-0.0006	0.0004
expenditures^3	0.0000	0.0000	0.0000	0.0000
expenditures^4	0.0000	0.0000	0.0000	0.0000
Age (females)				
76	0.0032	0.0020	-0.0008	0.0072
77	0.0023	0.0021	-0.0018	0.0063
78	0.0019	0.0020	-0.0020	0.0059
79	0.0009	0.0021	-0.0032	0.0051
80	0.0038	0.0021	-0.0004	0.0079
81	0.0026	0.0021	-0.0015	0.0066
82	0.0054	0.0021	0.0012	0.0095
83	0.0046	0.0021	0.0004	0.0087
84	0.0039	0.0022	-0.0003	0.0081
85	0.0046	0.0022	0.0003	0.0089
86	0.0054	0.0022	0.0010	0.0098
87	0.0050	0.0023	0.0004	0.0095
88	0.0057	0.0023	0.0011	0.0103
89	0.0075	0.0025	0.0027	0.0123
90	0.0048	0.0026	-0.0002	0.0099
91	0.0052	0.0027	-0.0001	0.0106
92	0.0084	0.0029	0.0027	0.0141
93	0.0042	0.0033	-0.0022	0.0106
94	0.0029	0.0037	-0.0043	0.0101
95	0.0052	0.0040	-0.0025	0.0129
96	0.0028	0.0044	-0.0058	0.0114
97	0.0037	0.0053	-0.0067	0.0140
98	0.0130	0.0059	0.0014	0.0246
99	0.0035	0.0071	-0.0104	0.0175
100 and over	0.0000	0.0053	-0.0103	0.0103

Note: The excluded reference category for age is 75.

TABLE A2.B (CONT'D): FIRST STAGE RESULTS FROM THE MAIN 2SLS SPECIFICATION

	coefficient	Robust standard error	95% Confidence Interval	
male	0.0026	0.0023	-0.0018	0.0070
Age (males)				
76	-0.0024	0.0032	-0.0086	0.0037
77	-0.0015	0.0032	-0.0078	0.0047
78	-0.0021	0.0032	-0.0084	0.0041
79	-0.0032	0.0033	-0.0095	0.0032
80	-0.0015	0.0033	-0.0079	0.0049
81	-0.0009	0.0032	-0.0071	0.0054
82	-0.0040	0.0033	-0.0104	0.0024
83	-0.0029	0.0033	-0.0094	0.0036
84	-0.0055	0.0033	-0.0120	0.0010
85	-0.0027	0.0034	-0.0094	0.0040
86	-0.0039	0.0034	-0.0106	0.0029
87	-0.0072	0.0037	-0.0144	0.0000
88	-0.0023	0.0038	-0.0097	0.0051
89	-0.0033	0.0040	-0.0111	0.0045
90	-0.0033	0.0042	-0.0115	0.0049
91	-0.0053	0.0045	-0.0142	0.0036
92	-0.0052	0.0048	-0.0147	0.0042
93	0.0014	0.0054	-0.0092	0.0119
94	0.0067	0.0066	-0.0063	0.0197
95	0.0030	0.0074	-0.0115	0.0176
96	0.0001	0.0086	-0.0167	0.0168
97	-0.0048	0.0100	-0.0244	0.0148
98	-0.0097	0.0133	-0.0358	0.0163
99	-0.0193	0.0154	-0.0495	0.0108
100 and over	0.0075	0.0115	-0.0150	0.0301

Note: The excluded reference category for age is 75.

TABLE A2.B (CONT'D): FIRST STAGE RESULTS FROM THE MAIN 2SLS SPECIFICATION

	coefficient	Robust standard error	95% Confidence Interval	
White	-0.0094	0.0038	-0.0168	-0.0021
Black	0.0000	0.0041	-0.0081	0.0080
Asian	0.0108	0.0051	0.0008	0.0208
Hispanic	0.0183	0.0043	0.0098	0.0269
2004 Census Block Group Demographics				
median household income / 1000	-0.0005	0.0001	-0.0006	-0.0004
per capita income / 1000	0.0018	0.0001	0.0015	0.0020
median year built	-0.0002	0.0001	-0.0003	-0.0001
median house value / 1000	-0.0001	0.0000	-0.0002	-0.0001
average house value / 1000	0.0000	0.0000	0.0000	0.0000
median gross rent / 1000	-0.0001	0.0002	-0.0005	0.0002
% over 65	0.0800	0.0115	0.0574	0.1025
% white	0.0587	0.0118	0.0355	0.0819
% black	0.0907	0.0134	0.0645	0.1169
% hispanic	-0.1238	0.0252	-0.1731	-0.0744
% 9th through 12th	-0.0886	0.0209	-0.1294	-0.0477
% high school graduate	-0.1383	0.0211	-0.1796	-0.0969
% some college	-0.2163	0.0254	-0.2661	-0.1664
% associate degree	-0.0631	0.0209	-0.1041	-0.0221
% bachelor's degree	-0.0472	0.0227	-0.0917	-0.0027
% graduate degree	-0.0447	0.0093	-0.0629	-0.0265
% owner occupied	0.0045	0.0104	-0.0159	0.0248
% renter occupied	0.0000	0.0000	0.0000	0.0000
$PM_{2.5}$ (1 μg/m^3) (Baseline, 2001-2003)				
exposure	0.9679	0.1504	0.6732	1.2627
$exposure^2$	-0.0862	0.0200	-0.1255	-0.0469
$exposure^3$	0.0068	0.0011	0.0045	0.0090
$exposure^4$	-0.0002	0.0000	-0.0002	-0.0001
Nonattainment * $PM_{2.5}$ (1 μg/m^3) (2001-2003)				
Nonattainment	-23.5482	1.3850	-26.2627	-20.8337
Nonattainment * exposure	4.9770	0.3495	4.2920	5.6619
Nonattainment * $exposure^2$	-0.3615	0.0343	-0.4286	-0.2943
Nonattainment * $exposure^3$	0.0094	0.0016	0.0063	0.0125
Nonattainment * $exposure^4$	0.0000	0.0000	-0.0001	0.0000

R^2 = 0.9631

Note: The excluded reference categories are "other" for race, "% with 8th grade or less" for block group education attainment, and "% vacant" for block group housing stock.

TABLE A3—DECADAL EXPOSURE TO $PM_{2.5}$ AND MORTALITY

	(1)	(2)	(3)	(4)	(5)	(6)
decadal $PM_{2.5}$ ($1\ \mu g/m^3$)	0.537***	0.365***	0.734***	2.369***	2.331***	1.791***
	(0.06)	(0.09)	(0.09)	(0.45)	(0.44)	(0.40)
ind. & neigh. covariates		x		x	x	x
specification	OLS	OLS	2SLS	2SLS	IV Probit	IV Probit
first-stage F statistic			24,224	799	799	55
number of individuals	2,384,195	2,384,195	2,384,195	2,384,195	2,384,195	2,384,195
share who survive through 2013	61	61	61	61	61	61

Note: The dependent variable equals 100 if an individual died prior to the end of 2013 and 0 otherwise. Col (1) is a univariate OLS regression 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. Columns (3) and (4) are the 2SLS analogues to Columns (1) and (2), respectively. The first row of Columns (1)-(4) presents the coefficient on decadal $PM_{2.5}$, which is the average marginal effect in these models. Col (5) is the control-function probit analogue to the 2SLS model in Col (4). Col (6) is a control-function probit that allows for additionally flexibility in both stages of estimation. The first row of Columns (5) and (6) present 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 by block group. Standard errors in Columns (5) and (6) are bootstrapped using 500 repetitions.

The table shows results from repeating estimation of the model in Table I using mortality as the outcome. The main specification in column (4) implies that a 1-$\mu g/m^3$ increase in average $PM_{2.5}$ exposure from 2004 through 2013 increased the probability of a death by the end of 2013 by 2.37 percentage points. This is six times larger than the comparable OLS specification in column (2). The OLS model in (2) yields an estimate that is about half the size of the estimate reported by Di et al. (2017) based on hazard function estimation using CMS data on the Medicare population from 2000 to 2012.

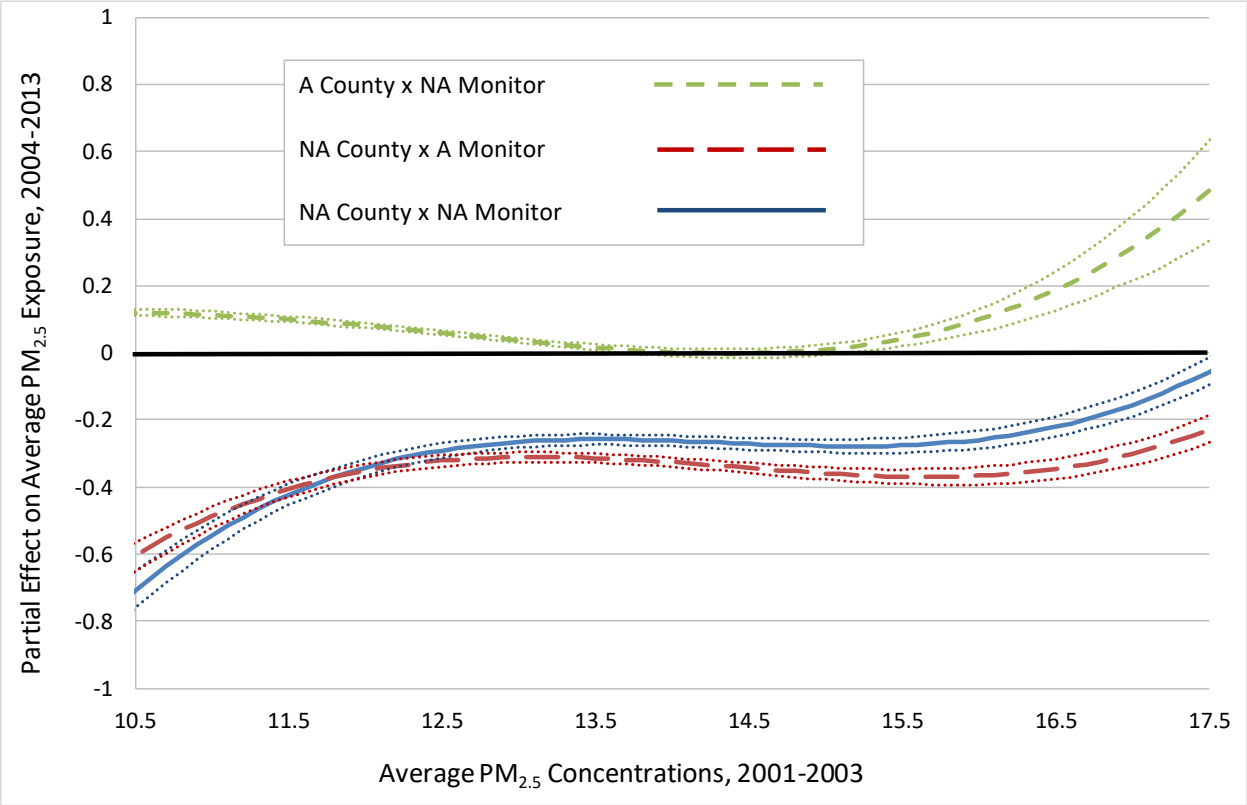
TABLE A4—COEFFICIENTS ON CANCER INSTRUMENTS IN THE SURVIVAL REGRESSION

Breast cancer in 2004	-3.66***
	(0.14)
Prostate cancer in 2004	0.11
	(0.14)
Colorectal cancer in 2004	-3.37***
	(0.17)
Endometrial cancer in 2004	-5.03***
	(0.37)
Leukemia/Lymphoma in 2004	-11.94***
	(0.25)
number of individuals	2,384,195
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 by initial Census block group.

The table shows coefficients on the instruments from the survival regression. The dependent variable is scaled to enable the coefficients to be interpreted as percentage point changes in the probability of survival.

FIGURE A5: PARTIAL EFFECT OF COUNTY-BY-MONITOR NONATTAINMENT ON $PM_{2.5}$ EXPOSURE



The figure reports conditional variation in decadal $PM_{2.5}$ exposures that arises from nonattainment status of the air quality monitor closest to the individual's residence, conditional on county nonattainment designation. Each solid line is constructed by using our first-stage coefficients on the excluded instruments to predict how nonattainment designations affected average decadal exposure conditional on baseline exposure. The excluded instruments consist of a fourth-order polynomial function of baseline exposure interacted with nonattainment indicators for the county and nearest monitor, which may or may not be in the same county. In the legend, "A" and "NA" denote attainment and nonattainment. The dotted lines represent 95% confidence bands based on 1,000 bootstrap replications, with clustering by Census block group.

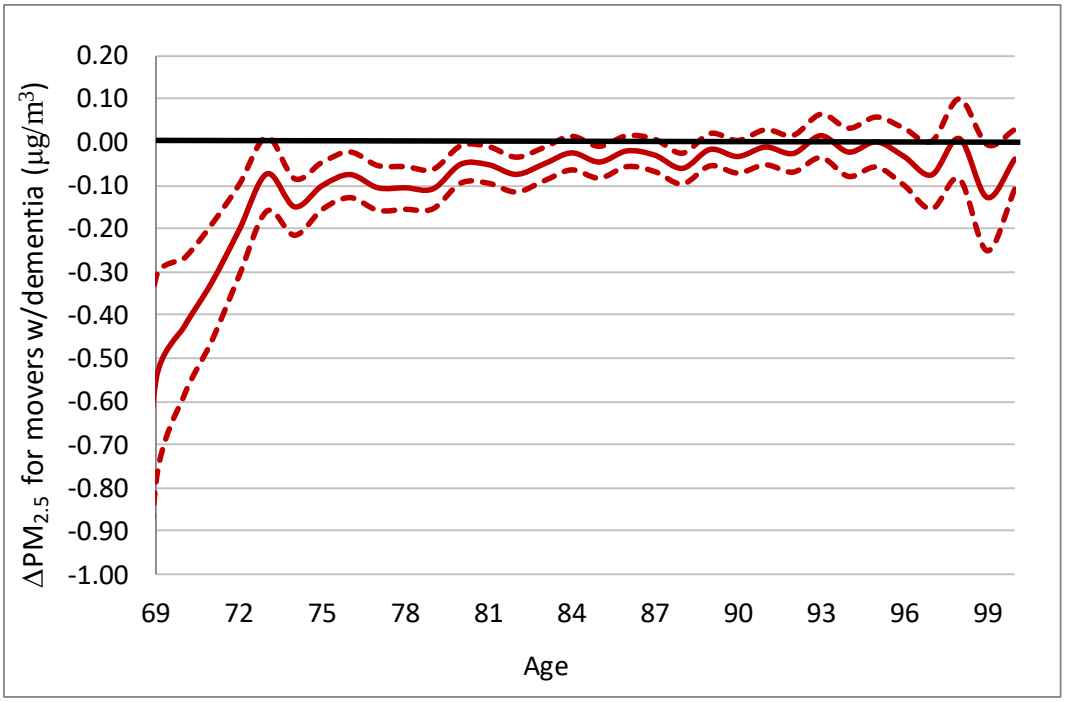
TABLE A5—ESTIMATES INCLUDING OTHER MEASURES OF AIR POLLUTION

	Concentrations		(1)	(2)	(3)	(4)
	mean	standard deviation				
decadal $PM_{2.5}$ (1 μg/m^3)	10.94	1.700	1.666***	1.520*	1.258***	1.657**
			(0.43)	(0.78)	(0.43)	(0.75)
decadal PM_{10} (1 μg/m3)	21.31	4.223		-0.396		-0.711*
				(0.48)		(0.40)
decadal ozone (parts per million)	0.04	0.004		584.195		-33.664
				(403.75)		(329.51)
decadal nitrogen dioxide (parts per billion)	13.21	4.022		0.623		0.327
				(0.65)		(0.39)
decadal sulfur dioxide (parts per billion)	2.51	1.063		-0.712		4.777
				(5.28)		(3.38)
decadal carbon monoxide (parts per million)	0.38	0.069		25.558*		23.541
				(13.26)		(15.27)
p-value on F-stat for PM_{10}, O_3, NO_2, SO_2 and CO				0.1239		0.2662
IV = county x monitor attainment			x	x	x	x
CBSA dummies					x	x
county dummies			x	x		
number of individuals			1,257,232	1,257,232	1,257,232	1,257,232
share with dementia in 2013			22.0	22.0	22.0	22.0

Note: Col (1) repeats the specification from Col (2) of Table IV. It is modified for each remaining column. Col (2) adds other criteria air pollutants. Columns (3) and (4) replace the county dummies with CBSA dummies.

The table shows coefficients on $PM_{2.5}$ with and without simultaneously controlling for other federally regulated air pollutants: coarse particulate matter (PM_{10}), ozone (O_3), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and carbon monoxide (CO). The main text provides additional details.

FIGURE A6: ANNUAL AVERAGE CHANGES IN $PM_{2.5}$ BY AGE, MIGRATORY STATUS, AND DEMENTIA

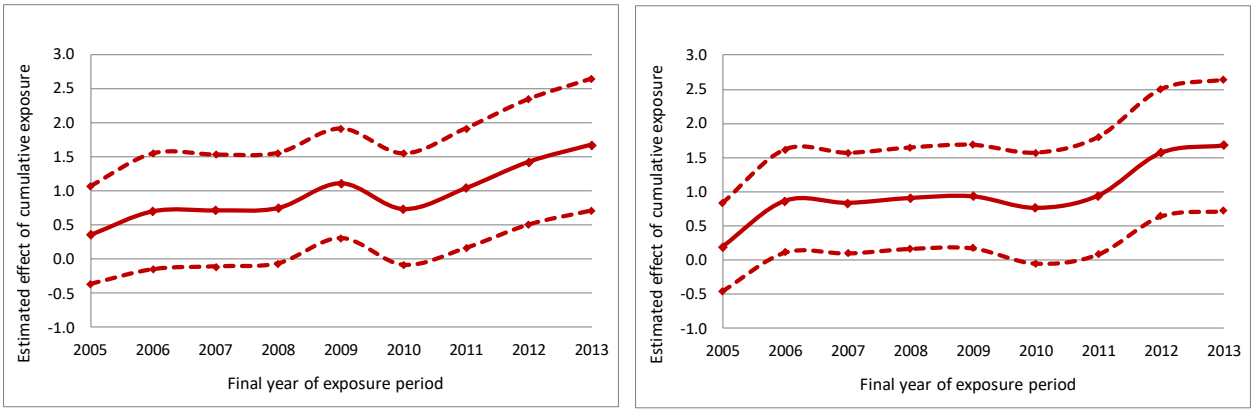


The solid trend line shows that movers with dementia tend to experience relatively larger year-to-year reductions in their $PM_{2.5}$ exposures as a result of moving, compared to non-movers of the same age (who may or may not have dementia). The dashed lines are 95% confidence bands on our estimates for the differentials. More specifically, the figure is constructed from a vector of coefficients, χ, estimated by regressing the year-to-year changes in individuals' $PM_{2.5}$ exposures on indicators for integer age and interactions between indicators for (i) integer age, (ii) whether the individual has dementia, and (iii) whether the year-to-year change in $PM_{2.5}$ exposure straddled a move.

$$\Delta PM25_{it} = PM2.5_{i,t} - PM2.5_{i,t-1} = \varrho + \varsigma\{age_t\} + \chi\{age_t\}\{move_t\}\{dementia_t\} + \vartheta_i.$$

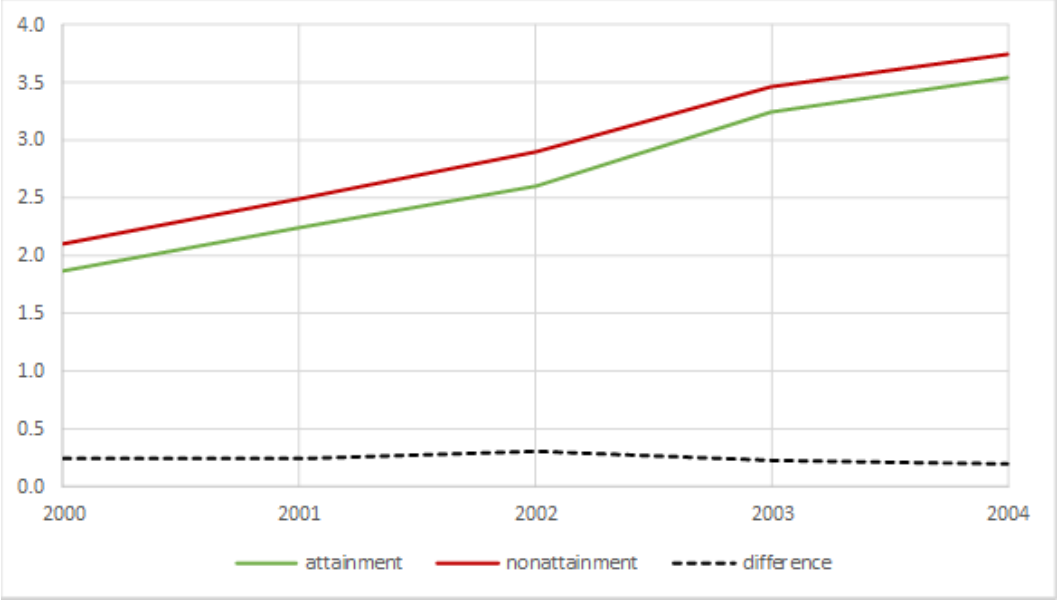
Like our main econometric models, all individuals age 100 and over are grouped into a single age bin at 100. Since the model includes 9 observations per individual and the errors may exhibit autocorrelation the confidence intervals are constructed from robust standard errors clustered at the individual level.

FIGURE A7: SENSITIVITY OF CUMULATIVE EXPOSURE ESTIMATES TO SAMPLE COMPOSITION



The figure on the left is the same as figure VII in the main text. It shows the estimated effect of a 1-μg/m^3 increase in average $PM_{2.5}$ exposure from 2004 through the final year of exposure on the horizontal axis. The sample size decreases from 2.377 million individuals in 2005 to 1.257 million in 2013 due to death and transition to Medicare Advantage. The figure on the right is constructed by repeating the estimation using only the 1.257 million individuals who survived to 2013.

FIGURE A8: TRENDS IN NEW DEMENTIA DIAGNOSIS PRIOR TO 2005 BY COUNTY ATTAINMENT STATUS



Supplemental Appendix B: Additional Background on Models

A Model of a Dementia Production Function

To illustrate how our econometric model for dementia's onset can be linked to a more primitive "production function" for dementia we start by writing dementia as being determined by the lifetime history of $PM_{2.5}$ exposure (from initial year B to year t), all time-varying determinants of dementia, ζ_{it} (which includes both observed and unobserved), and all time-invariant determinants of dementia, ξ_i (which includes both observed factors and unobserved factors such as genetics):

$$y_{it} = \alpha \sum_{s=t-9}^{t} PM_{is}\,(1-\delta)^{t-s} + g(PM_{iB}, PM_{iB+1}, \dots, PM_{it-11}, PM_{it-10}) + \zeta_{it} + \xi_{it}$$

where we specify the functional form for the most recent decade of $PM_{2.5}$ exposure with discount rate δ, and allow all previous exposure to enter flexibly via the function $g(\cdot)$. First-differencing yields:

$$\Delta y_{i2013} \equiv y_{i2013} - y_{i2003}$$

$$= \alpha \sum_{s=2004}^{2013} PM_{is}\,(1-\delta)^{2013-s} - \alpha \sum_{s=1994}^{2003} PM_{is}\,(1-\delta)^{2003-s} + \Delta g_{i2013} + \Delta\zeta_{i2013},$$

where ξ_i has dropped out, $\Delta g_{i,2013} = g(PM_{iB}, \dots, PM_{i2003}) - g(PM_{iB}, \dots, PM_{i1993})$, and $\Delta\zeta_{i,2013} = \zeta_{i2013} - \zeta_{i2003}$. Next, we assume that the discount rate on the most recent decade of exposure is zero, embedding the medical literature's hypothesis that the near-term effect of $PM_{2.5}$ exposure are cumulative (e.g., Underwood 2017, Block et al. 2012), as in: [60]

$$\Delta y_{i2013} = \alpha \sum_{s=2004}^{2013} PM_{is} - \alpha \sum_{s=1994}^{2003} PM_{is} + \Delta g_{i2013} + \Delta\zeta_{i2013}.$$

To illustrate how this primitive specification relates to our empirical model of dementia onset, recall that our main specification is given by:

[60] In principle, one could alternatively aim to estimate the discount rate, δ, via non-linear least squares.

$$\Delta y_{i2013} = \alpha \sum_{s=2004}^{2013} PM_{is} + f(PM_{i2001}, PM_{i2002}, PM_{i2003}) + \beta X_i + \gamma H_i + \theta W_i + \eta_{c(i)} + \epsilon_i,$$

where we proxy for $\alpha \sum_{s=1994}^{2003} PM_{is}$, $\Delta g_{i,2013}$, and $\Delta \zeta_{i,2013}$ with $f\left(PM_{i,2001}, PM_{i,2002,} PM_{i,2003}\right)$, $\beta X_i, \gamma H_i, \theta W_i$, and $\eta_{c(i)}$. This implies that, by definition, our econometric error is given by:

$$\varepsilon_i = -\alpha \sum_{s=1994}^{2003} PM_{is} + \Delta g_{i2013} + \Delta \zeta_{i2013} - f(PM_{i2001}, PM_{i2002}, PM_{i2003}) - \beta X_i - \gamma H_i - \theta W_i - \eta_{c(i)}.$$

Our key identifying assumption is therefore that cov(ϵ_i, Z_i) = 0 where Z_i are the instruments and ϵ_i contains all attributes that affect dementia onset conditional on $f\left(PM_{i,2001}, PM_{i,2002,} PM_{i,2003}\right)$, βX_i, γH_i, θW_i, and $\eta_{c(i)}$. Note that in this specification, if $g_{it} = \alpha \sum_{s=B}^{t-10} PM_{is}$, then $-\alpha \sum_{s=1994}^{2003} PM_{is} + \Delta g_{i,2013} = 0$.

Thus, in words, the identifying assumption required for consistent estimation in our main specification is that, after we condition on our controls, individuals are not systematically sorting themselves into neighborhoods located in different counties (within the same CBSA) that differ in their likelihood of being designated nonattainment in the future, based on unobserved time-varying factors that are correlated with the likelihood of a new dementia diagnosis. Note that we condition on an extensive set of controls given by (1) the CBSAs where individuals had chosen to live at the start of 2004; (2) their observed individual demographics; (3) their observed measures of individual health from 2001-2003; (4) the observed measures of socioeconomic status among the individuals living in their residential Census block groups in 2004; and (5) their baseline $PM_{2.5}$ exposure from 2001-2003. It is worth noting that analogs to this assumption 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). Further, the strength of our identifying assumption is weakened relative to most prior studies in this literature by the way we leverage the panel structure of CMS administrative data to additionally purge separable time-constant individual-specific unobserved characteristics that may affect health outcomes.

Additional Details on Bounds

This derivation follows Lee (2009) and is modified for our application.

For notational simplicity, we let *Y* denote the binary outcome of interest, i.e., a new diagnosis of dementia, and let *X* denote the continuous explanatory variable of interest, i.e., instrumented decadal exposure to $PM_{2.5}$, and suppress all other variables. *S* is the binary variable denoting survival. We consider changes in the expected value of *Y* for a marginal increase in *X*, where the increase is denoted *h*.

We are interested in the causal effect of *X* on *Y*, holding selection on survival constant. We denote this causal effect α_X. For any given *X*, this is defined as the change in the expected value of *Y* among those who would survive under both *X* and *X*+*h*, i.e., inframarginal individuals,

$$\alpha_X = \lim_{h \to 0} \frac{E[Y|X+h, S(X+h)=1] - E[Y|X, S(X+h)=1]}{h}.$$

Without additional assumptions, one cannot directly recover α_X from the data. However, one can recover the total effect of *X* on *Y*, which we denote as Δ_X.

$$\Delta_X = \lim_{h \to 0} \frac{E[Y|X+h, S(X+h)=1] - E[Y|X, S(X)=1]}{h}. \tag{B1}$$

This captures the fact that those exposed to *X*+*h* will have a different survival rate compared with those exposed to just *X*. Denoting the share of marginal individuals as ρ_{Xh}, allows us to rewrite the second term in the numerator of equation (B1) as,

$$E[Y|X, S(X)=1] = \rho_{Xh} E[Y|X, S(X)=1, S(X+h)=0] + (1-\rho_{Xh}) E[Y|X, S(X)=1, S(X+h)=1]. \tag{B2}$$

Gathering terms, this allows us to write the numerator in equation (B1) as,

$$E[Y|X+h, S(X+h)=1] - E[Y|X, S(X)=1, S(X+h)=1] - \rho_{Xh}(E[Y|X, S(X)=1, S(X+h)=0] - E[Y|X, S(X)=1, S(X+h)=1]).$$

We assume monotonicity, such that *S* is weakly decreasing in *X*. This implies that $S(X+h)=1 \rightarrow S(X)=1$, and that,

$$E[Y|X, S(X)=1, S(X+h)=0] = E[Y|X, S(X+h)=1].$$

This allows us to write,

(B3) $\Delta_X = \alpha_X + \lim_{h \to 0} \frac{\rho_{Xh}}{h} (E[Y|X, S(X) = 1, S(X+h) = 1] - E[Y|X, S(X) = 1, S(X+h) = 0])$.

This equation shows that the difference in expected Y under the marginal change in X is comprised of two terms. The first term reflects the effect of the marginal increase in X on the expected value of Y for inframarginal individuals. The second term reflects the underlying difference in the expected Y between inframarginal and marginal individuals, scaled by ρ_{Xh}. In other words, the total effect is comprised of a causal effect of X on expected Y and a compositional effect. As α_X is the object of interest, we rearrange equation (B3) to get,

(B4) $\alpha_X = \Delta_X + \lim_{h \to 0} \frac{\rho_{Xh}}{h} (E[Y|X, S(X) = 1, S(X+h) = 0] - E[Y|X, S(X) = 1, S(X+h) = 1])$.

While Δ_X and ρ_{Xh} are recoverable from the data, the remaining two conditional expectations in equation (B4) remain unknown. However, we can recover $E[Y|X, S(X) = 1]$ from the data, allowing us to construct lower and upper bounds for the difference in the two unknown conditional expectations, as $E[Y|X, S(X) = 1, S(X+h) = 0]$ is naturally bounded between 0 and 100 in our application. This allows us to construct bounds for α_X. Specifically, for the lower bound, we set $E[Y|X, S(X) = 1, S(X+h) = 0]$ to 0 and solve for $E[Y|X, S(X) = 1, S(X+h) = 1]$ using,

$$E[Y|X, S(X) = 1, S(X+h) = 1] = \left(\frac{E[Y|X, S(X) = 1] - \rho_{Xh} E[Y|X, S(X) = 1, S(X+h) = 0]}{1 - \rho_{Xh}} \right)$$

which follows from equation (B2). We then use these values, along with Δ_X and ρ_{Xh}, in equation (B4) to recover the lower bound for α_X. The upper bound is constructed analogously by setting $E[Y|X, S(X) = 1, S(X+h) = 0]$ 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 recover the share of marginal individuals in the data and use this to inform the bounds.

Returning to our application in Section V of the main text. In the linear-probability model, Δ is constant (by definition), so we calculate bounds for α using Δ and measures of $E[Y|X, S(X) = 1]$ and ρ_{Xh} calculated at the mean value of X. In our flexible probit model, we estimate a value of Δ_X for each individual and calculate bounds using individual-specific values of α_X using individual-specific values of Δ_X, $E[Y|X, S(X) = 1]$, and ρ_{Xh}. Note that for simplicity in the main text, we write equation (B4) as,

$$\alpha = \Delta + \rho * (P_y^A - P_y^B).$$

Supplemental Appendix C: Additional Background on Policy Calculations

The EPA's benefit-cost analysis of the CAA excludes the benefits of dementia cases avoided (US EPA 2011). Dementia is not counted among the set of morbidities attributed to air pollution, nor is it included among the channels through which air pollution is assumed to increase mortality. The EPA's mortality estimates are calibrated to the results of cohort studies by Pope et al. (2002) and Landen et al (2006), both of which found that $PM_{2.5}$ increased all-cause mortality via cardio-vascular and lung cancer deaths but not deaths due to other causes such as dementia.

We take a first step toward filling this gap by using our estimates to approximate the value of dementia cases avoided in 2013 in nonattainment counties due to the 1997 $PM_{2.5}$ regulation. Because we are unaware of any revealed preference estimate of the value of reducing dementia risk, our approach relies on estimates of the years of life lost due to dementia, the length of time living with dementia and quality of life lost due to having dementia, and a value of a life year among individuals age 75 and over with and without dementia.

We do not know of any published estimates of the effects of dementia on life expectancy. To approximate this, we use the Medicare data to compare the average age at death of those who died with dementia against the average age at death of those who died without dementia. This yields a difference of 6.1 years (80.2 versus 86.3). Because the Medicare population is not typically in full health even apart from dementia, each year of life lost does not represent a full quality-adjusted life year (QALY). Using estimates from Ara and Brazier (2011), we estimate that the average health-state utility value (or "QALY weight") among this population is 0.8. These values together imply that a dementia diagnosis on average leads to 4.88 QALYs lost due to mortality, because each year without dementia for this population is equivalent to 80 percent of a year in full health.

To estimate the lost QALYs due to lower quality of life while living with dementia, we combine the median QALY weights for mild, moderate and severe Alzheimer's disease and related dementia from Kasai and Maguro (2013) with the transition rates between severity levels from Spackman et al. (2012). We rely on these prior estimates because we cannot directly observe dementia severity with the Medicare data. We combine them with estimates from the Medicare data for the probability of survival to the end of each year following a dementia diagnosis. These estimates are provided in the table below. From Spackman et al. (2012), among those who remain living with dementia, an estimated 77% of mild cases transition each year to moderate, and 50%

of moderate transition to severe. Kasai and Maguro (2013) estimated the health-state utility value for each level to range from 0.52–0.73 in mild cases, 0.30–0.53 in moderate cases, and 0.12–0.49 in severe cases. Combining the midpoints of these ranges with the transition rates and survival rates and again assuming a utility value of 0.8 apart from dementia yields an estimated loss of 1.0 QALY per dementia case due to morbidity. This ranges from 0.6 QALYs using the high end of the health state utility value range to 1.5 using the low end. By combining this with the loss from mortality we find a central estimate of 5.9 QALYs lost per dementia case, with a range from 5.5 to 6.4 QALYs.

TABLE B1—MORTALITY RATES BY YEARS SINCE DEMENTIA DIAGNOSIS

Years since Dementia Diagnosis	Percent Dying	Cumulative Percent Dead
0	23.38	23.38
1	19.89	43.28
2	14.17	57.45
3	11.32	68.76
4	8.82	77.58
5	6.72	84.3
6	5.02	89.32
7	3.58	92.9
8	2.57	95.46
9	1.77	97.24
10	1.17	98.4
11	0.76	99.17
12	0.46	99.63
13	0.25	99.89
14	0.11	100

We use a range of estimates for the value of a statistical life year in full health, from $100,000 to $300,000, with a central estimate of $200,000. The lower bound is a common benchmark, the upper bound is from Aldy and Viscusi (2007). Previously, Hirth et al. (2000) found a wide range of estimates, with the central estimates between $114,000 and $196,000 in 2018 dollars. The mid-point estimates of the QALYs lost per dementia diagnosis and the value of a QALY imply a value per statistical case of dementia avoided of approximately $1.2 million.

We combine these statistics with our estimate that the regulation's effect on annual average $PM_{2.5}$ exposure from 2004 to 2013 for individuals age 75 and above in nonattainment counties using our difference-in-difference estimate of -1.24 $\mu g/m^3$. Multiplying this reduction by our main estimate for the effect of a 1-$\mu g/m^3$ increase in decadal exposure on the probability of a dementia

diagnosis (1.68 pp) implies that the regulation reduced the dementia rate by 2.1 pp. We multiply this by the Census Bureau's estimate that 8.7 million individuals age 75 and above lived in in counties in 2013 that were officially designated as nonattainment in 2005. This implies that the $PM_{2.5}$ regulation led to approximately 182,000 fewer cases of dementia among this population in 2013. At $1.2 million per case, the $PM_{2.5}$ regulation yielded benefits of $214 billion for the cohort of individuals age 75 and above in nonattainment counties. Using our lower bound estimates for the lost QALYs per diagnosis and the value of a QALY yields a benefit of $100 billion, while using the upper bound indicates a benefit of $349 billion.

We interpret these estimates as likely lower bounds on the benefits of the EPA's $PM_{2.5}$ standard for several reasons. First, we exclude any benefits that accrued to individuals in attainment counties, for example that might occur due to spatial spillover of $PM_{2.5}$ reductions. We also exclude health benefits for individuals who were under age 65 at the start of the decade, benefits for those who died during the decade, and any health benefits other than reduced dementia rates for individuals who were over 65 and survived to the end of the decade.

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