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HAZED AND CONFUSED: THE EFFECT OF AIR POLLUTION ON DEMENTIA

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ABSTRACT

We find that long-term exposure to fine-particulate air pollution (PM2.5) degrades health and human capital among older adults by increasing their risk of developing Alzheimer's disease and related dementias. We track U.S. Medicare beneficiaries' cumulative residential exposures to PM2.5 and their health from 2004 through 2013, leveraging within- and between-county quasi-random variation in PM2.5 resulting from the expansion of Clean Air Act regulations. We find that a 1 ìg/m3 increase in decadal PM2.5 increases the probability of a dementia diagnosis by 1.68 percentage points. The effects are as large or larger when we adjust for mortality-based sample selection and additional Tiebout-sorting dynamics. We do not find relationships between decadal PM2.5 and placebo outcomes. Our estimates suggest that the federal regulation led to nearly 182,000 fewer people with dementia in 2013, yielding $214 billion in benefits. Further, PM2.5's effect on dementia persists below the current regulatory thresholds.

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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 million dementia patients spent $277 billion on health care services in 2018, with an additional 18 billion labor hours by unpaid caregivers (Alzheimer's Association 2018). The precise causes of dementia remain unknown. However, recent medical research raises suspicion that long-term exposure to fine-particulate air pollution smaller than 2.5 microns in diameter ($PM_{2.5}$) may contribute to dementia (Block et al. 2012, Underwood 2017). 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 spatially correlated 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 as an equilibrium outcome 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

[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.

from the U.S. Medicare program to develop a longitudinal research design that comprehensively addresses residential sorting. First, we assemble ten years of data on a random sample of millions of Americans over the age of 65 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 2004 through 2013. Then we combine individuals' location histories with $PM_{2.5}$ data from the Environmental Protection Agency (EPA) to measure long-term $PM_{2.5}$ exposure at the individual level, accounting for migration.

Like the prior observational studies, we observe 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 correlations caused by residential sorting, sample selection, errors in measuring pollution exposure, or other sources of omitted variable bias. 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 longitudinal two-stage-least-squares (2SLS) models flexibly control for individual char-

acteristics 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 fixed effects to absorb spatial variation in diagnostic standards, health care quality and access, and latent environmental quality. 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.

We find 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 this 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.

Because our main estimation sample is limited to people who survived to 2013, our finding could diverge from the population-wide effect if unobserved health affecting survival is correlated with unobserved health affecting dementia. To adjust for this classic selection bias, 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. The 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.

Our selection-corrected 2SLS estimate could still diverge from the population average treatment effect due to sample selection and/or residential sorting based on heterogeneous sensitivity to $PM_{2.5}$ in terms of developing dementia. In other words, people who have a higher dementia sensitivity to $PM_{2.5}$ might be more or less likely to survive the decade or more or less likely to live near pollution hot spots. We allow for these types of scenarios by adapting Garen's (1984) correlated random coefficient model to simultaneously address selection and sorting on latent heterogeneity in sensitivity to $PM_{2.5}$ within the selection-corrected 2SLS model. However, we find that this has almost no effect on our estimates, and we cannot reject the hypothesis of no sorting or selection on $PM_{2.5}$ sensitivity.

We implement four sets of additional tests to investigate the validity and robustness of our research design and the mechanisms underlying our findings. 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, supporting our research design. Similarly, we repeat the estimation using dementia at baseline as the outcome. Our estimate is small and relatively precise, but statistically indistinguishable from zero, suggesting 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 dementia. Second, we show that our results are not explained by vascular dementia that may arise due to short-term pollution spikes leading to strokes, but rather

by Alzheimer's disease specifically. Third, we test whether our findings can be explained by shorter durations of exposure. The estimated effects of $PM_{2.5}$ increase in exposure duration and become statistically significant beyond eight years, supporting the medical literature's hypothesis that long-term exposure drives $PM_{2.5}$'s effect on dementia. Fourth, our results persist when we modify our main specification to use different instruments, different measures of dementia such as the use of prescription drugs for the symptoms of Alzheimer's disease, different samples that either include or exclude people who select into managed care plans known as Medicare Advantage, and different approaches to measuring $PM_{2.5}$ exposure including adjusting for the possibility that dementia's onset changes sorting behavior.

Our results suggest that the negative effects of air pollution on health and human capital and the monetary benefits of regulation are substantially larger than previously realized due to its effects on dementia. Incorporating these effects will be important for comprehensively evaluating 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 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 people with dementia in 2013, 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 regulation would yield additional benefits.

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 people 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 developing novel data and methods to overcome this problem, we contribute to the sorting literature by providing the

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

first empirical analysis of long-term pollution exposure to account for migration.

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. 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. The literature has proposed 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). People 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 people 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).

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 people who chose to live near major roadways (e.g., Chen et al. 2017). 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 pollution. However, this study did not

address potential confounding from residential sorting.

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 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.[3] 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 (Aufhammer, Bento, and Lowe 2009).

Prior research has leveraged similar policy changes to evaluate air pollution's

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

effects by assuming that people's 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 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 $PM_{2.5}$.

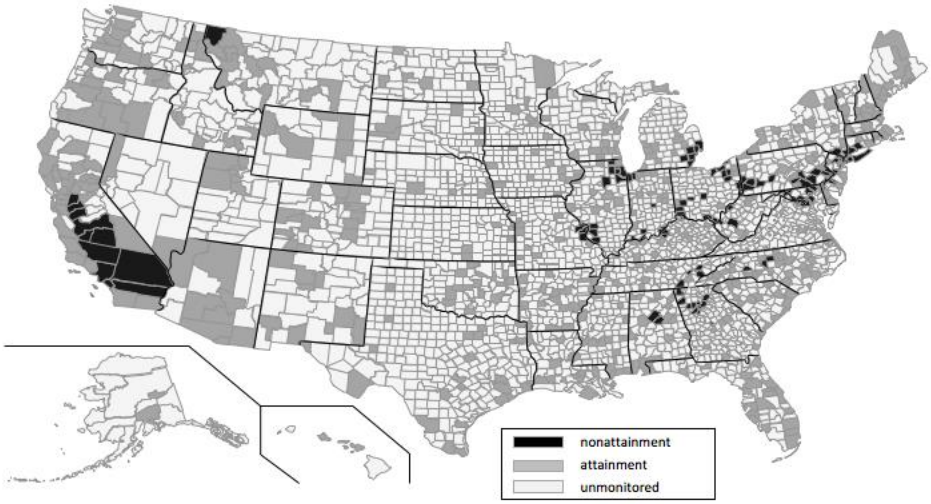
In 1997, the EPA established initial monitoring protocols for $PM_{2.5}$ and set the maximum-allowable annual average concentration at 15.05 $\mu g/m^3$. By 1999, a national network of more than 900 air quality monitors was put into place. Several litigants challenged the new $PM_{2.5}$ standard, but 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.[4]

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

[4] 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).

ensure that nonattainment counties met the 15.05 μg/m^3 standard by 2010.

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



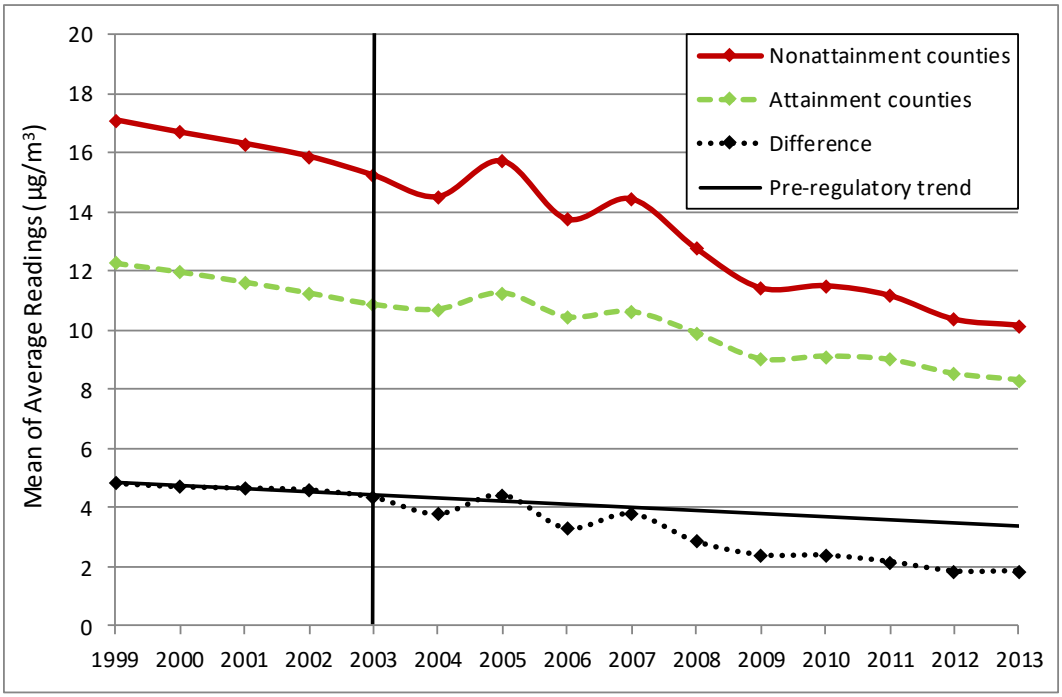
Note: The map shows attainment status in 2005 for US counties that had air quality monitors in place throughout the 2001-2003 evaluation period. There were 132 nonattainment counties located in 21 states and 528 attainment counties in 50 states.

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.[5] 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 μg/m^3

[5] 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. 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.

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, such as an operation, a test, or a visit to the doctor.[6] Alternatively, beneficiaries can choose to enroll in a Medicare Advantage (MA) managed care plan that charges a monthly premium in exchange for limiting annual out-of-pocket expenditures and, often, for providing additional forms of coverage such as dental and vision.[7] We analyze Medicare administrative

[6] 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.

[7] 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.

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 1999 and then add random 10% samples of all new beneficiaries each year from 2000 to 2013.[8]

After compiling these data, we extract records for the subset of people 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. First, we drop 2.7 million people 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.[9] Next, we restrict the sample to people 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. Thus, our third exclusion is to drop 0.3 million people who had dementia in 2004 because the disease is currently irreversible, leaving no scope for change.[10] Finally, we drop 0.4 million people 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 compro-

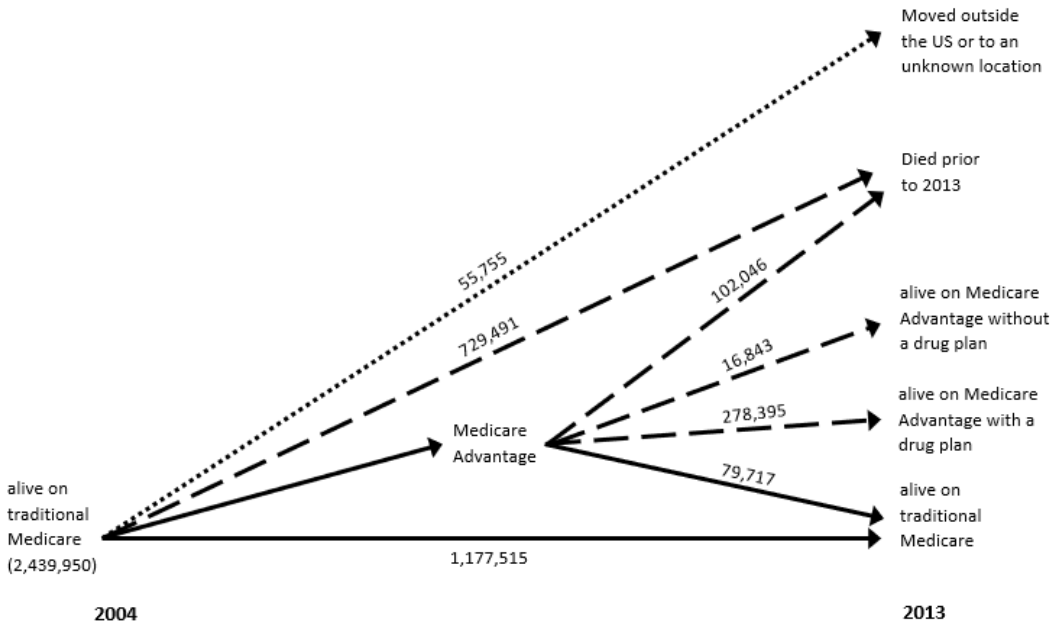
[8] Some people become eligible prior to age 65, for example due to disability, but we exclude them from the data until they turn 65. Due to the provenance of our data, we also include an independent, random 20% sample from the universe of age 65 and over beneficiaries who purchased standalone prescription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

[9] 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.

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

mise 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 people in 2004. Figure III illustrates how between 2004 and 2013, some of these people move outside of the continental US, move out of TM into MA and perhaps back again, or die. Our primary estimation sample is comprised of 1,257,232 people who are alive and enrolled in traditional Medicare in 2013 (1,177,515 people who 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 people we first observe 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

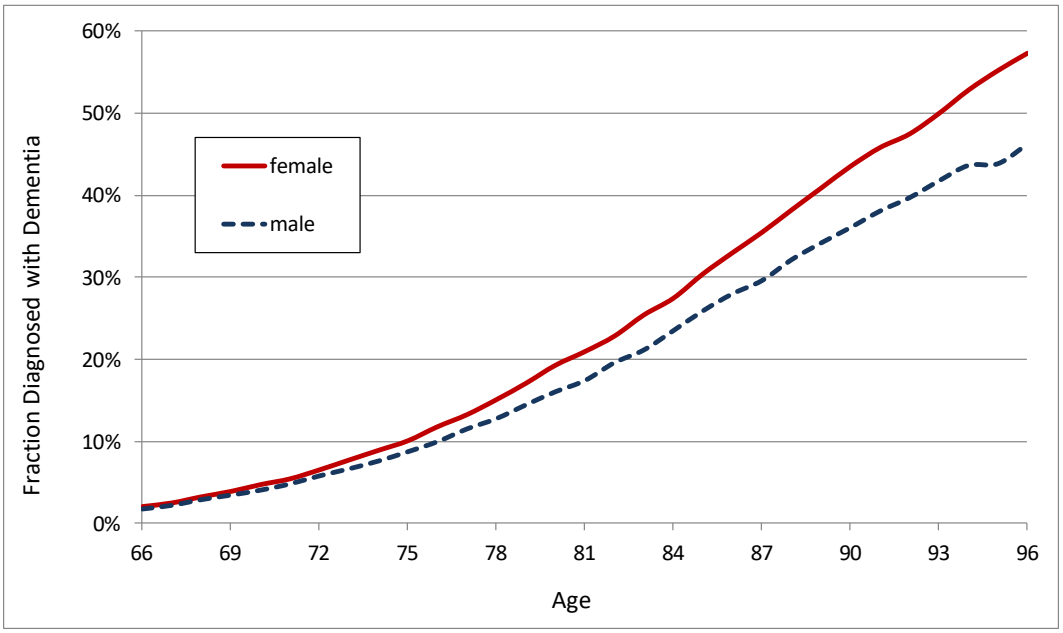
For people in traditional Medicare, CMS's Chronic Conditions Data Warehouse file uses codes on insurance claims to track if and when each person is diagnosed with a range of specific chronic medical conditions. A diagnosis of dementia as officially defined by the World Health Organization (see footnote 1) is based on the presence of multiple symptoms of cognitive impairment that significantly impact daily functioning.[11] 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).[12] Figure IV shows how the fraction of people with dementia varies by age and gender in 2013. Approximately 2% of our sample 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.

In addition to the diagnoses recorded in the Chronic Conditions Data Warehouse file, we observe if and when each person began taking one of the five drugs

[11] This claims-based approach to identifying dementia cases has been well validated (Taylor Jr., Fillenbaum, and Ezell 2002).
[12] 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).

used to treat the symptoms of Alzheimer's disease: donepezil, galantimine, rivastigmine, memantine, and donepezil and memantine in combination. We develop this measure from prescription drug claims data that cover many beneficiaries. Beginning in 2006, Medicare beneficiaries could purchase prescription drug coverage through Medicare, either from standalone prescription drug plans or as part of their coverage from a Medicare Advantage plan. In our sample, 1,098,256 individuals meeting our other criteria also had drug coverage through Medicare, and 12% of them initiated one of these medications between 2006 and 2013. Among 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 diagnoses by 2013.

FIGURE IV: DEMENTIA 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 2018). Most people 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 people living in each individual's 2004 Census block group.[13] 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 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 patterns in our sample are similar to those reported by the Census Bureau for individuals aged 65 and above. Over two thirds of people live in the same ZIP+4 throughout our study period. Of the 31% of people who move at least once, 17% move between counties and 10% move between states. We use this information to measure each person's long-term exposure to air pollution, incorporating changes in pollution experienced as a result of moving.[14]

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

[14] 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.

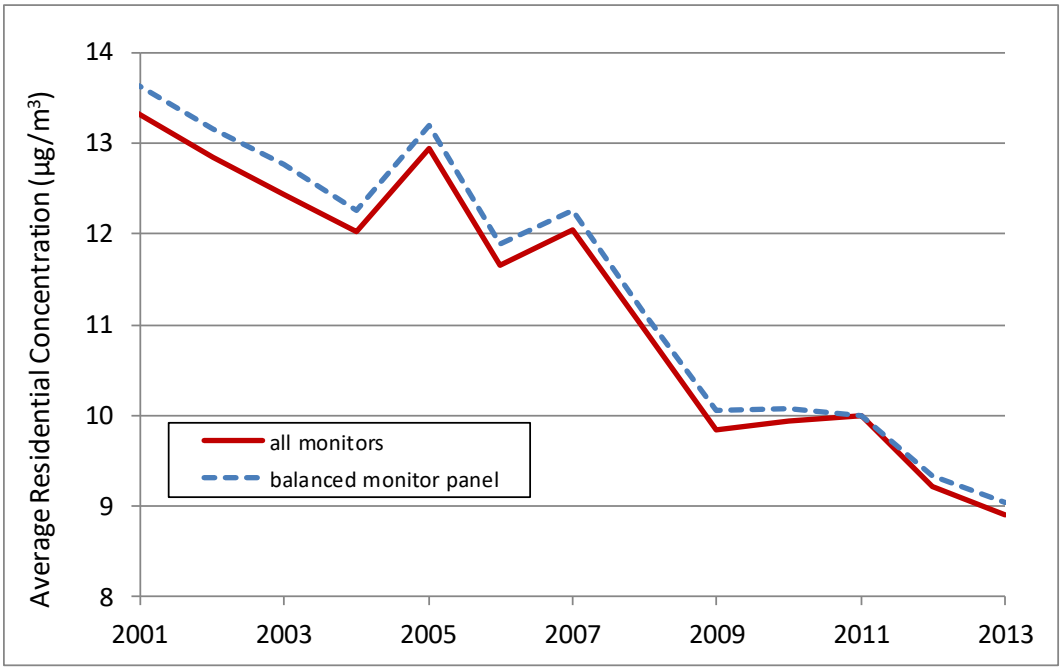
Individuals in our estimation samples live in 2.7 million distinct ZIP+4 Codes between 2004 and 2013. We measure annual $PM_{2.5}$ concentrations at the centroids of each of these areas using data from the EPA's air quality system, consisting of 1,722 monitors over the period 2001 to 2013. To approximate annual average concentrations in each ZIP+4 Code, we use the latitude and longitude coordinates of each monitor along with the coordinates of each ZIP+4.[15] 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.[16] 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 people 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 people 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 older adults' cognition. Like all existing methods for measuring pollution exposure, the constructed histories may embed measurement error because of our inability to fully observe and control for 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

[15] 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.
[16] This method of interpolation, with weights given by the distance raised to a negative exponent, is a predominant method in the environmental economics literature.

sealing, and variation in respiration due to health and physical activity. Our instrumental variables approach also helps to address these sources of measurement error.

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 an unbalanced panel of all monitors in operation each year (solid line) or a balanced panel of 485 monitors that monitored $PM_{2.5}$ continuously from 2001 through 2013 (dashed line). We use this balanced panel in our main econometric analysis to avoid measurement error that could be introduced if new monitors tend to be located in cleaner or dirtier areas (Muller and Rudd 2017, Grainger, Schreiber and Chang 2018) although 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 longitudinal 2SLS model

Let $y_{i,t}$ indicate whether person *i* has dementia in year *t*. As described above, we restrict our primary sample to people who had not received a dementia diagnosis before the end of 2004 and who were still alive in 2013. We define an indicator, $\Delta y_i = y_{i,2013} - y_{i,2004}$, for whether person *i* is newly diagnosed with dementia by the end of 2013. This measure of change in individual cognition is the dependent variable in our linear probability model,

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

The coefficient of interest in equation (1), α, measures the effect of the average concentration of $PM_{2.5}$ at the person's residence over the decade (from 2004 to 2013) on Δy_i.

We control for individual and neighborhood characteristics that may be correlated with both dementia and $PM_{2.5}$. First, we add dummy variables, η_i, for the approximately one thousand core-based statistical areas (CBSAs) in which people live in 2013 to absorb the effects of environmental factors that could be spatially correlated with both pollution and dementia.[17] 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 people who never move during our study period, the CBSA dummies will control for pre-regulatory sorting across CBSAs on the basis

[17] CBSAs 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.

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).[18] These flexible age-by-gender controls absorb the nonlinear trends in dementia rates shown in Figure IV.

The H_i vector characterizes baseline health in 2004. We employ a full-factorial design to control for pre-existing medical conditions known to elevate the risk of dementia, adding dummy variables for each of 32 possible combinations of hypertension, diabetes, congestive heart failure, ischemic heart disease, and stroke.[19] We further control for unobserved 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.[20]

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 person 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

[18] 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.
[19] 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.
[20] Medicare Parts A and B cover virtually all medical services aside from prescription drugs. This includes doctors' services, preventive care, durable medical equipment, hospital outpatient services, laboratory tests, x-rays, hospital inpatient services, nursing facilities, and hospice care.

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 person *i*'s residential location in 2004. This controls for any residual effects of pre-regulatory sorting into more polluted neighborhoods by people 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-differenced model. That is, α is identified by how cumulative $PM_{2.5}$ exposure from 2004 to 2013 affects the probability of a dementia diagnosis, conditional on pre-regulatory concentrations in the individuals' baseline neighborhoods. Because the dependent variable is the diagnosed change in cognition, an individual fixed effect is purged from the econometric model.

Despite the rich set of controls in equation (1), two potential threats to identification remain: measurement error in pollution exposure and omitted variable bias. We address both concerns by instrumenting for decadal exposure. Equation (2) provides the first stage of the 2SLS model:

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

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

Thus in the longitudinal 2SLS models, α is identified by variation in (instrumented) 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 similar 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 Aufhammer, Bento, and Lowe (2009) and Bento, Freedman, and Lang (2015).[21] Third, people who moved between 2004 and 2013 experienced variation in exposure due to their migration paths, similar to the identifying variation in Banzhaf and Walsh (2008).

B. First-stage results

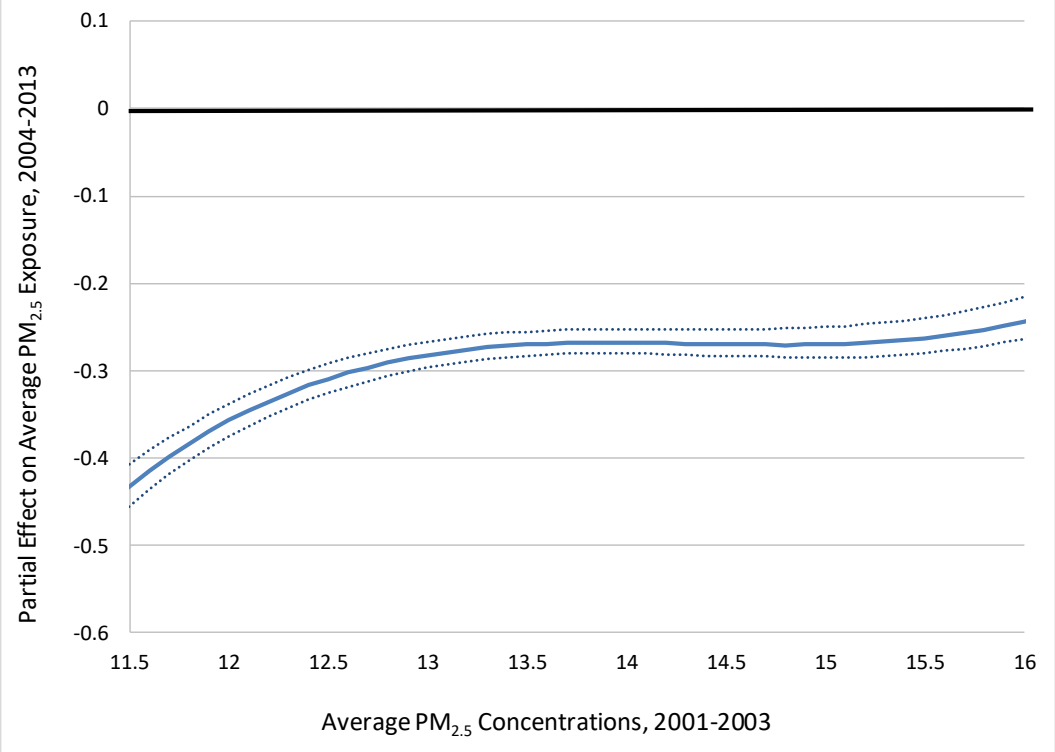
The pollution exposure histories and first-stage estimates reveal that the EPA's $PM_{2.5}$ regulation was followed by four notable changes in exposure. First, average exposures declined for more than 95% of people between 2001-2003 and 2004-2013. Second, the declines were larger for people whose 2004 neighborhoods were more polluted at baseline (2001-2003). Third, conditional on baseline neighborhood pollution, the declines were larger for people whose 2004 neighborhoods were in nonattainment counties. Fourth, the nonattainment county differential declines nonlinearly as we approach the regulatory threshold from below. The last two sources of variation power our econometric model.

Figure VI uses the coefficients on the instruments, π, to illustrate the identifying

[21] 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.

variation in $PM_{2.5}$.[22] Intuitively, the partial effect of nonattainment on post-regulatory $PM_{2.5}$ exposure is negative. The size of the effect declines in baseline concentrations as we approach the regulatory threshold from below. This trend mirrors Auffhammer, Bento and Lowe's (2009) estimate for the partial effect of the EPA's 1990 county nonattainment designations for PM_{10} on subsequent PM_{10} concentrations (see their Figure 4).

FIGURE VI: 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 96% confidence bands constructed from 1,000 bootstrap replications, with clustering on Census block group.

The partial effect of nonattainment on individual $PM_{2.5}$ exposure in Figure VI

[22] The first-stage F statistic is 637, suggesting that any finite sample bias is negligible. Table A2 reports model coefficients.

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 approximate 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 differences-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 counties for the purposes of policy scenarios considered in Section VII. This reduction is slightly larger than in Figure II mainly because of within-county variation in where people live in relation to monitors.

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.[23]

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

[23] 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.

baseline health and $PM_{2.5}$ exposure, demographics and socioeconomic status.

Columns (3) and (4) show the 2SLS analogs to the OLS models in columns (1) and (2). Using instrumental variables increases the estimates for $PM_{2.5}$'s effect and makes the estimates less sensitive to the inclusion of individual covariates.[24] 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.[25] 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.[26]

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

	(1)	(2)	(3)	(4)
decadal $PM_{2.5}$ (1 $\mu g/m^3$)	0.751***	0.209*	1.164***	1.679***
	(0.06)	(0.11)	(0.09)	(0.49)
individual & neighborhood covariates		x		x
specification	OLS	OLS	2SLS	2SLS
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: 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 person's Census block group, and pre-regulatory $PM_{2.5}$ levels at their residence from 2001-2003. Cols (3) and (4) are the 2SLS analogues to Cols (1) and (2), respectively. Coefficients on all other covariates in the first and second stage models in Col (4) are reported in Appendix Table A2. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered by Census block group.

[24] 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.

[25] The first stage results are reported in Table A2. The p-value from a Sargan test of overidentifying restrictions is 0.34, so we fail to reject the joint hypothesis that our instruments are valid and that the model is correctly specified. Fitted probabilities of receiving a dementia diagnosis lie between zero and one for 99.9% of individuals.

[26] 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, a 10 pp increase in the fraction of block group residents with graduate degrees (relative to less than 8th grade education) is associated with a 0.5 pp reduction in the dementia diagnosis probability.

V. Assessing the Effects of Selection and Sorting on the Main Results

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 1 $\mu g/m^3$ increase in $PM_{2.5}$ for one day caused 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 2.4 percentage points, equivalent to 6% of the decadal mortality rate.[27] 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 bias may be an issue.

For example, suppose unobserved health affecting survival is negatively correlated with unobserved health affecting dementia, i.e., sicker people who are more likely to die sooner are also more likely to be diagnosed with dementia if they live. In this case, selection induces negative correlation between the error in equation (1) and our instrumented measure of $PM_{2.5}$. This classic form of selection bias would yield a downward-biased estimate of $PM_{2.5}$'s effect on dementia in the selected sample.[28]

We address this classic form of selection bias using a control-function approach based on Heckman (1979) and Heckman and Robb (1986). 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.

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

[27] Table A3 reports results from mortality models that parallel the specifications used in Table I.
[28] 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.

We define M_i to include indicators for 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 impact decadal survival, are assumed to be unrelated to latent features of health that affect the probability of a dementia diagnosis. This exclusion restriction is supported by the medical literature on dementia (Driver et al. 2012, Ganguli 2015).[29] We then use the survival function residuals, $\hat{v}_i$, to define an additional control variable that we include in equations (1) and (2). Given the well-documented equivalence of 2SLS and control-function estimation in linear models, we estimate the following control-function equation,

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

$\hat{v}_i$ 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 level of the block group.

B. Selection and sorting based on sensitivity to $PM_{2.5}$

Thus far we have assumed a common coefficient on $PM_{2.5}$. We now extend the control-function model to specify heterogeneous coefficients on $PM_{2.5}$ and allow the unobserved heterogeneity in these sensitivities to be correlated with survival and/or residential location choice.

First we allow latent health determining survival to be correlated with latent

[29] We provide further evidence in support of the exclusion restrictions below when we discuss results from placebo models that include individuals' cancer diagnoses as explanatory variables and are found to have no effect on dementia. Thus, our exclusion restrictions are supported by exceptionally rich data on individuals' medical diagnoses as well as medical literature and placebo tests. This allows us to sharpen our estimator relative to bounding methods that do not require exclusion restrictions (Lee 2009).

heterogeneity in cognitive sensitivity to $PM_{2.5}$. The motivating concern is that our survivor sample may be comprised of people who were either more vulnerable or more resilient to pollution. We test this concern by implementing a control-function approach motivated by Garen (1984).[30] This 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. Assuming the heterogeneity in sensitivity is linearly related to latent health, $\bar{\alpha}$ can be identified by extending (4) to include an interaction between the survival-equation residual and decadal $PM_{2.5}$ exposure following Garen (1984) and Wooldridge (2015).[31]

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

$$\text{where}\quad \tilde{\epsilon}_i = \epsilon_i - \phi_1\hat{v}_i - \phi_2\hat{\varepsilon}_i - \psi_1\hat{v}_i\sum_{t=2004}^{2013}\frac{PM2.5_{i,t}}{10}.$$

Equation (5) also provides a formal test of sensitivity-based selection. We can recover an estimate of the average sensitivity among survivors: $\bar{\alpha} + \psi_1 E[v_i|S_i = 1]$. Therefore, $\psi_1 E[v_i|S_i = 1]$ measures the degree to which the average sensitivity among survivors differs from the population-wide average; if this term is greater than (less than) zero, the survivors are, on average, more (less) sensitive than the population as a whole.

A separate concern is that sensitivity could affect residential sorting, e.g., people 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 the local average treatment effect among a non-random subset of the population. We modify (5) to address this concern by

[30] See Heckman and Vyltacil (1998), Card (2001) and Wooldridge (2003) for discussions of this and similar approaches.
[31] 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).

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 parallels the Garen-type 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.

Finally, we 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. In other words, we generalize (5) to include both interaction terms: $\hat{v}_i \sum_{t=2004}^{2013} \frac{PM2.5_{i,t}}{10}$ and $\hat{\varepsilon}_i \sum_{t=2004}^{2013} \frac{PM2.5_{i,t}}{10}$, nesting the prior two specifications.[32]

C. Results

The first column in Table II reports the estimate for α using equation (4) to augment our main 2SLS model to control for selection on survival. The coefficient, 2.33 pp, is larger than the 2SLS estimate from Table I. This is consistent with classic selection bias caused by latent health factors that make people who are more likely to survive the decade also less likely to develop dementia.[33]

The second column reports results using equation (5) to control for survival-based selection on heterogeneity in $PM_{2.5}$ sensitivity. The coefficient is effectively unchanged from the first column. Likewise, our estimate of ψ_1 is close to zero, suggesting that heterogeneity in $PM_{2.5}$ sensitivity is not correlated with survival.[34]

[32] The final two specifications also assume that the expected value of the parameter governing heterogeneity is linearly 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.
[33] In our estimation of the survival function, equation (3), the instruments are jointly significant at the 99% level and individually significant at the 99% level with the exception of prostate cancer, as may be seen in Appendix Table A4.
[34] The point estimate implies that those who survived were slightly less sensitive than those who died. The average sensitivity among survivors is calculated as $\bar{\alpha} + \hat{\psi}_1 E[\hat{v}_i | S_i = 1] = 2.339 - 0.016 * 0.3009 = 2.334$, which is trivially lower than the sensitivity of the population as a whole.

The third column presents results from the alternate version of equation (5) that allows people to sort across neighborhoods based on heterogeneity in α_i. The estimate for mean sensitivity is effectively unchanged from the prior two columns and the 95% confidence interval for the coefficient on ψ_2 includes zero, suggesting that people were not sorting based on cognitive sensitivity to $PM_{2.5}$. This may be because people do not know their cognitive sensitivity to $PM_{2.5}$ or because the heterogeneity is minimal.

TABLE II—ESTIMATES ACCOUNTING FOR SELECTION AND SORTING

	(1)	(2)	(3)	(4)
decadal $PM_{2.5}$ (1 $\mu g/m^3$)	2.334***	2.339***	2.328***	2.332***
	(0.51)	(0.51)	(0.51)	(0.51)
Corrects for selection based on survival	x	x	x	x
α_i allowed to vary with survival		x		
α_i allowed to vary with attainment			x	
α_i allowed to vary with survival and attainment				x
survival control, ϕ_1	-27.264***	-27.082***	-27.256***	-27.080***
	(0.17)	(0.93)	(0.17)	(0.93)
attainment control, ϕ_2	-2.236***	-2.328***	-2.569***	-2.569***
	(0.53)	(0.51)	(0.77)	(0.77)
survival control * decadal $PM_{2.5}$, ψ_1		-0.016		-0.016
		(0.08)		(0.08)
attainment control * decadal $PM_{2.5}$, ψ_2			0.030	0.030
			(0.05)	(0.05)
number of people in survival regression	2,384,195	2,384,195	2,384,195	2,384,195
number of people in dementia regression	1,257,232	1,257,232	1,257,232	1,257,232
share with dementia in 2013	22	22	22	22
share who survive through 2013	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. Col (1) controls for selection on mortality. Col (2) extends Col (1) to allows people to differ in their vulnerability to $PM_{2.5}$, with vulnerability being potentially correlated with latent factors affecting survival. Col (3) extends Col (1) to allow people to differ in their vulnerability to $PM_{2.5}$, with vulnerability being potentially correlated with latent factors affecting residential sorting on air pollution. Col (4) nests the models in the first three columns to control for all three mechanisms simultaneously. 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.

Finally, the last column presents results from the most general specification that

allows heterogeneity in α_i to be simultaneously related to the survival-based selection process and the residential sorting process. As before, we find 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 2.33 percentage points.

Overall, Table II suggests that, if anything, classic selection on survival causes our main estimates to be attenuated relative to the population average treatment effect. However, these results do not appear to be influenced by differential sensitivities to $PM_{2.5}$, either through selection or sorting.

VI. Additional Sensitivity Analysis

A. Alternative measures of dementia

Table III shows results from models that first repeat the estimation after adding people who self-selected into Medicare Advantage plans 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 expand the sample to include people who exited traditional Medicare at some point after 2004 to enroll in a Medicare Advantage plan that included prescription drug coverage at some point from 2006 to 2013. This expands the sample by 278,395 people (accounting for 94% of the sample who switched to MA and survived through 2013 (Figure III)). Because we do not observe claims-based diagnoses for this sample but can observe their prescription drug claims from 2006 onward, we redefine the dementia measure 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 estimate, indicating that our main estimate is not biased by selection into Medicare Advantage.

TABLE III—ESTIMATES USING ALTERNATIVE MEASURES OF DEMENTIA

	(1)	(2)	(3)	(4)	(5)
decadal $PM_{2.5}$ (1 μg/m^3)	1.679*** (0.49)	1.692*** (0.46)	0.611 (0.38)	1.068*** (0.39)	1.696*** (0.48)
dependent variable	claim-based diagnosis	claim-based diagnosis or drug	claim-based diagnosis without Alzheimer's	claim-based diagnosis with Alzheimer's	claim-based diagnosis
dependent variable mean	22	21	12	10	22
Number of individuals	1,257,232	1,535,746	1,257,232	1,257,232	1,257,232
% in traditional Medicare in 2013	100	82	100	100	100
% in Medicare Advantage in 2013	1	19	1	1	1

Note: Col (1) repeats the main specification from Table I. Col (2) extends the sample to include people who switched to a Medicare Advantage plan with prescription drug coverage at some point between 2006 and 2013, while redefining the dependent variable to be 100 for people who are diagnosed with dementia and/or take prescription drugs for Alzheimer's disease. Col (3) is the same as (1) but defines the dependent variable as dementia cases without an Alzheimer's diagnosis and Col (4) defines it as Alzheimer's disease specifically. Col (5) is the same as (1) but adds an indicator for whether people had a stroke by 2013. Summing the percentages of people enrolled in traditional Medicare and Medicare Advantage rounds to just over 100% because a small fraction of people 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.

Columns (3) and (4) repeat the estimation of the model in (1) 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. Therefore, 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 stroke. Our results suggest that the probability of being diagnosed with dementia is 19.1 pp higher for those who had a stroke (95% CI = [18.8,19.3]). However, controlling for this has virtually no effect on the $PM_{2.5}$ coefficient, as shown in column (5). This reinforces the conclusion 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 IV 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 county nonattainment indicator according to whether the average $PM_{2.5}$ concentration from 2001 to 2003 at the air quality monitor closest to a person's residence exceeded the federal standard. This generates three indicators that vary within counties: (i) nonattainment county with nearest monitor exceeding the standard, (ii) nonattainment county without nearest monitor exceeding the standard, and (iii) attainment county with nearest monitor exceeding the standard. As in our main specification, each indicator is interacted with the polynomial function of baseline exposure. This yields an estimate of 1.67 pp, nearly the same as our main estimate in column (1).[35]

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

[35] Appendix Figure A5 shows the estimated partial effects of each interaction. We find patterns consistent with strategic regulatory targeting. Our estimates suggest that county nonattainment designations led to slightly larger reductions in long-term exposures for people living closest to nonattainment monitors at baseline exposure levels below 11.7 $\mu g/m^3$. Moreover, we find that nonattainment designations produced slight increases in $PM_{2.5}$ for people in attainment counties living near nonattainment monitors. 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).

using the unbalanced panel reduces the instrument's power to explain decadal $PM_{2.5}$ exposures in the first stage and yields a smaller second-stage estimate of 1.26 pp.[36]

TABLE IV—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
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	22.0	22.0	22.0	22.0	22.0	22.0	22.0

Note: Col (1) repeats our main result that is modified for each remaining column. Col (2) stratifies the nonattainment county instrument according to whether the monitor closest to a person's residence was in attainment while replacing CBSA dummies with county dummies. Col (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. Col (4) measures pollution at the coarser 5-digit ZIP code level. Col (5) replaces the 4th 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. Col (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.

In column (4) we measure $PM_{2.5}$ at the centroids of peoples' 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 people 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 μg/m^3 increments) and add an indicator variable for each bin. This again produces a similar

[36] The first-stage F-statistic is 394 for the unbalanced panel compared to 637 for the balanced panel.

$PM_{2.5}$ 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 a person's first post-diagnosis move. For example, if a person 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—the opposite of what would be implied by reverse causality. This is because movers with dementia tend to move to less polluted areas.[37] 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 not impart an upward bias on our estimates.

C. 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 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., exposures 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

[37] We show exposure conditional on migration status and dementia diagnosis in Appendix Figure A6.

estimation sample includes 2.4 million people. As we move from 2005 to 2013, the sample diminishes due to death and switching into Medicare Advantage.

FIGURE VII: ESTIMATED EFFECTS OF $PM_{2.5}$ by EXPOSURE DURATION

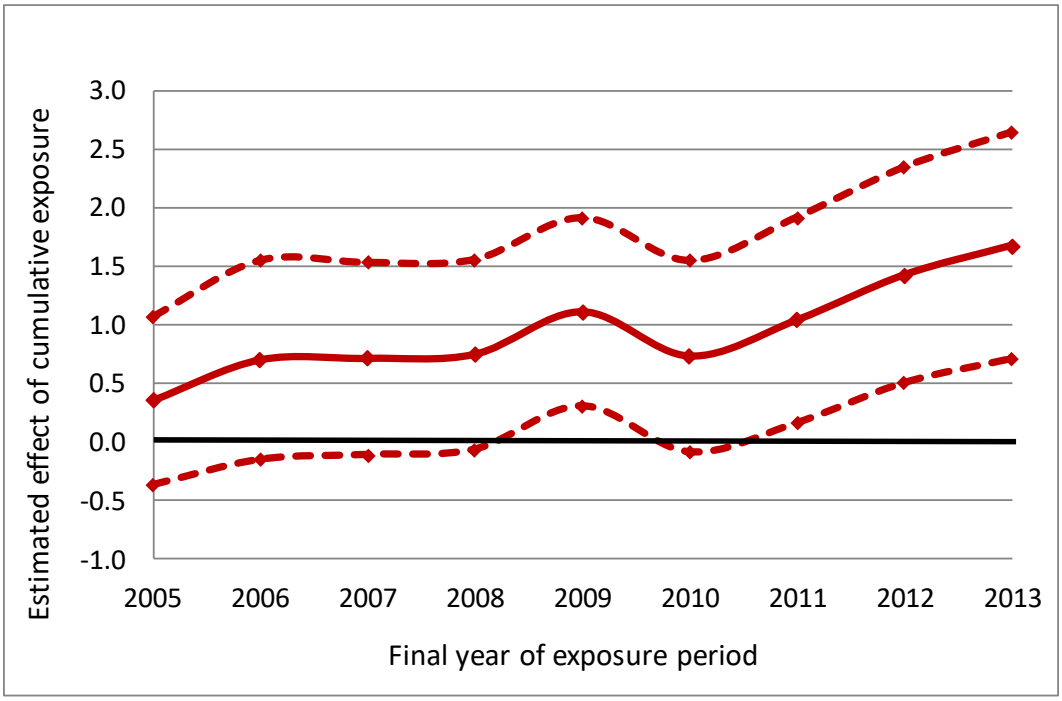


Figure VII shows our estimates for the effects of 1 $\mu g/m^3$ increases in average residential concentrations from 2004 to the interval endpoints shown on the horizontal axis, along with 95% confidence intervals. The estimates increase steadily with exposure duration and remain statistically distinguishable 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 people 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 Medicare Advantage.

D. Placebo tests

Finally, we estimate a series of 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 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-American and Hispanic groups relative to non-Hispanic white groups.[38]

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

	Dementia in 2013	Glaucoma	Fibro-myalgia	Breast cancer	Prostate cancer	Peripheral vascular disease	Dementia in 2004
decadal $PM_{2.5}$	1.679***	-1.026*	-0.465	-0.077	-0.189	0.581	-0.065
($1\ \mu g/m^3$)	(0.49)	(0.53)	(0.53)	(0.21)	(0.23)	(0.60)	(0.25)
number of people	1,257,232	1,065,603	1,182,076	1,248,239	1,249,959	1,186,008	2,673,519
share with outcome	22	17	18	3	4	27	11

Note: the first column repeats our main result for comparison. The next five columns report results using the same model but replacing dementia with each of the placebos. The last column estimates the same model using dementia in 2004 as the outcome. Asterisks indicate statistical significance at the 10%, 5%, and 1% levels based on robust standard errors clustered by Census block group.

Finding large, positive, and statistically significant effects of $PM_{2.5}$ on these

[38] 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%).

placebo morbidities would signal that our 2SLS research design may be compromised. Table V 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.[39]

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 close to zero and estimated relatively precisely. We conclude that placebo models support our research design.

VII. Interpretation and Policy Implications

A. Assessing the magnitude of the $PM_{2.5}$ effects

Our main point estimate (Table I, column (4)) suggests that a 1 $\mu g/m^3$ increase in 10-year average residential concentrations of $PM_{2.5}$ from 2004 to 2013 increased 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 may be understood as a moderate change in exposure.

Table VI compares our $PM_{2.5}$ result to the coefficients we estimated on other dementia risk factors that were included as covariates in the model. For instance,

[39] Our criteria for selecting placebos 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 with pollution, 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). We leave a comprehensive analysis of $PM_{2.5}$ on morbidity to future research.

our estimate for the effect of a 1 μg/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 and not diagnosed with any of the other health risk factors (0.8 pp). Our $PM_{2.5}$ estimate is somewhat smaller than risks associated with pre-existing diagnoses of the other chronic conditions individually, which range from a 2.1 pp increase for ischemic heart disease only to a 8.0 pp increase for stroke only. Someone diagnosed with all five conditions by 2004 had a 20.6 pp higher probability of being diagnosed with dementia by the end of 2013. 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 VI. COMPARING RELATIVE RISKS FOR $PM_{2.5}$ AND OTHER 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, Col(4). Appendix Table A2 reports the full set of model coefficients.

B. Benefits of the EPA's 1997 $PM_{2.5}$ regulation from dementia cases avoided

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.[40] 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.

We estimate the regulation's effect on annual average $PM_{2.5}$ exposure from 2004 to 2013 for people 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. Multiplying this by the Census Bureau's estimate for the size of the 75-and-over population in 2013 in counties that were nonattainment in 2005 (8.7 million) implies that the $PM_{2.5}$ regulation reduced the number of dementia cases by approximately 182,000.

Because we are unaware of any revealed preference estimate of the value of reducing dementia risk, we approximate the benefit of cases avoided by using prior estimates for the value of a quality-adjusted life year (QALY), in conjunction with prior estimates for dementia's impacts on quality of life and the Medicare data for an estimate of dementia's effects on life expectancy. Appendix B describes our calculations in detail. We first use our data to calculate two statistics: the average effect of a dementia diagnosis on life expectancy (-6.1 years) and the average post-diagnosis survival period (2.7 years). Then we use age- and morbidity-specific QALY weights from a review of the health economics literature to translate dementia's effects on morbidity and mortality into a measure of lost QALYs. This results in a central estimate of 5.9 QALYs lost per dementia case, with a range from 5.5 to

[40] 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 cardiovascular and lung cancer deaths but not deaths due to other causes such as dementia.

6.4 reflecting upper and lower bounds on the severity of symptoms. Finally, we assign a value per QALY. A conventional but arbitrary value is $100,000. Empirical studies typically report much higher values. For example, Aldy and Viscusi (2007) estimated a value of $300,000 for those age 65 and above. We consider a range of values with $200,000 as the midpoint, a lower bound of $100,000 and an upper bound of $300,000. The midpoint 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. Multiplying this by our estimate of the number of cases avoided implies that the $PM_{2.5}$ regulation yielded benefits of $214 billion for the cohort of people 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.

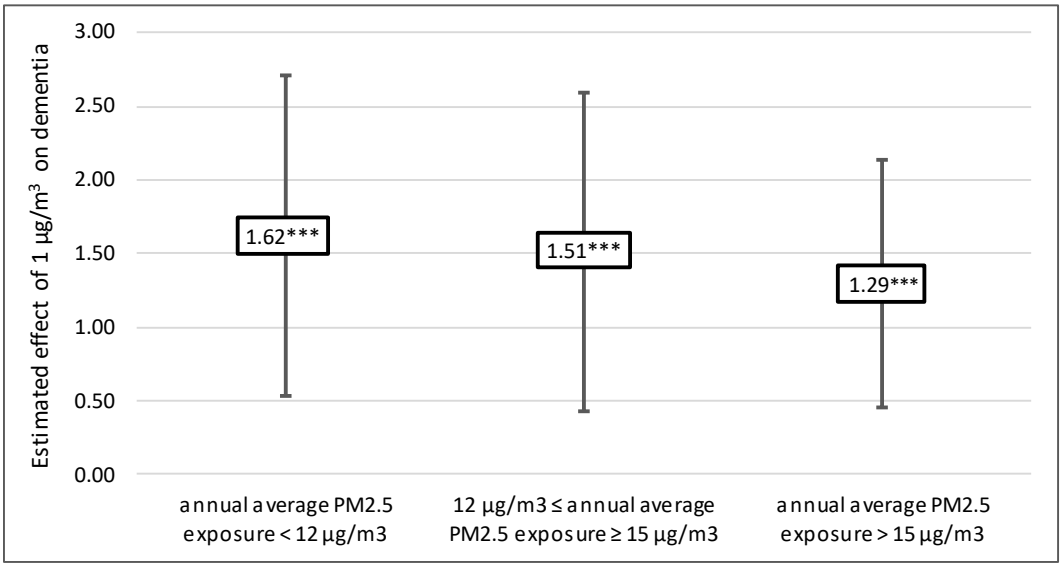
We interpret these estimates as likely lower bounds on the benefits of the EPA's 1997 $PM_{2.5}$ standard for several reasons. First, we exclude any benefits that accrued to people in attainment counties, for example due to spatial spillover of $PM_{2.5}$ reductions. We also exclude health benefits for people 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 people who were over 65 and survived to the end of the decade.

C. Assessing the effect of lowering the regulatory threshold on dementia

We conclude our policy analysis by considering the EPA's 2012 lowering of the federal cap on maximum allowable annual average $PM_{2.5}$ concentrations from 15 $\mu g/m^3$ to 12 $\mu g/m^3$. While not enough time has passed to assess how lowering the cap affected dementia rates via lower exposure over the subsequent decade, we can investigate whether $PM_{2.5}$'s effects on dementia would diminish at lower levels of exposure. We do so by repeating estimation of 2SLS models after interacting our

measure of annual average decadal exposure with dummies for whether exposures exceeded the 1997 threshold (above 15 μg/m^3), fell between the 1997 and 2012 thresholds (12 to 15 μg/m^3), or fell below the 2012 threshold (12 μg/m^3). We use the instruments defined by interacting county attainment status with nearest monitor attainment (Table IV, column 2) to separately identify $PM_{2.5}$'s effects over each range of exposure.

FIGURE IX: EFFECTS OF $PM_{2.5}$ ON DEMENTIA BY LEVELS OF EXPOSURE



Note: Asterisks indicate statistical significance at the 1% level based on robust standard errors clustered by Census block group.

The results in Figure IX indicate that the marginal effects of $PM_{2.5}$ on dementia are weakly decreasing in $PM_{2.5}$ concentrations. This is consistent with prior evidence that $PM_{2.5}$ has larger marginal effects on mortality at lower concentrations in general (Pope et al. 2015, Li et al. 2019) and at concentrations below 12 μg/m^3 specifically (Di et al. 2017). While confidence intervals are too wide to conclusively determine that marginal effects are larger at lower exposure levels, they also show that our estimated positive effects are unlikely to be due to sampling error if the effects do not exist among the populations within each exposure range. These

findings indicate that the 2012 policy change is likely to continue to improve health by reducing dementia, as would further reductions of the threshold.

VIII. Discussion and Conclusions

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. We find that the effects are driven by Alzheimer's disease rather than by vascular dementia resulting from strokes triggered by short-term pollution spikes. Furthermore, our results show that $PM_{2.5}$'s effect on dementia is driven by cumulative exposure and that this effect is not explained by 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, income, and preferences for neighborhood amenities (Banzhaf and Walsh 2008, Bayer, Ferreira and McMillan 2007, Bayer, Keohane and Timmins 2009, Kahn and Walsh 2015).

Dementia's global social costs continue to grow with the aging of populations in 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 and that further regulation would be likely to yield additional health benefits. Our estimates for the monetary benefits of cases avoided ($214 billion) are sufficiently large to suggest that dementia-related benefits may matter for future benefit-cost analyses of air quality regulations.

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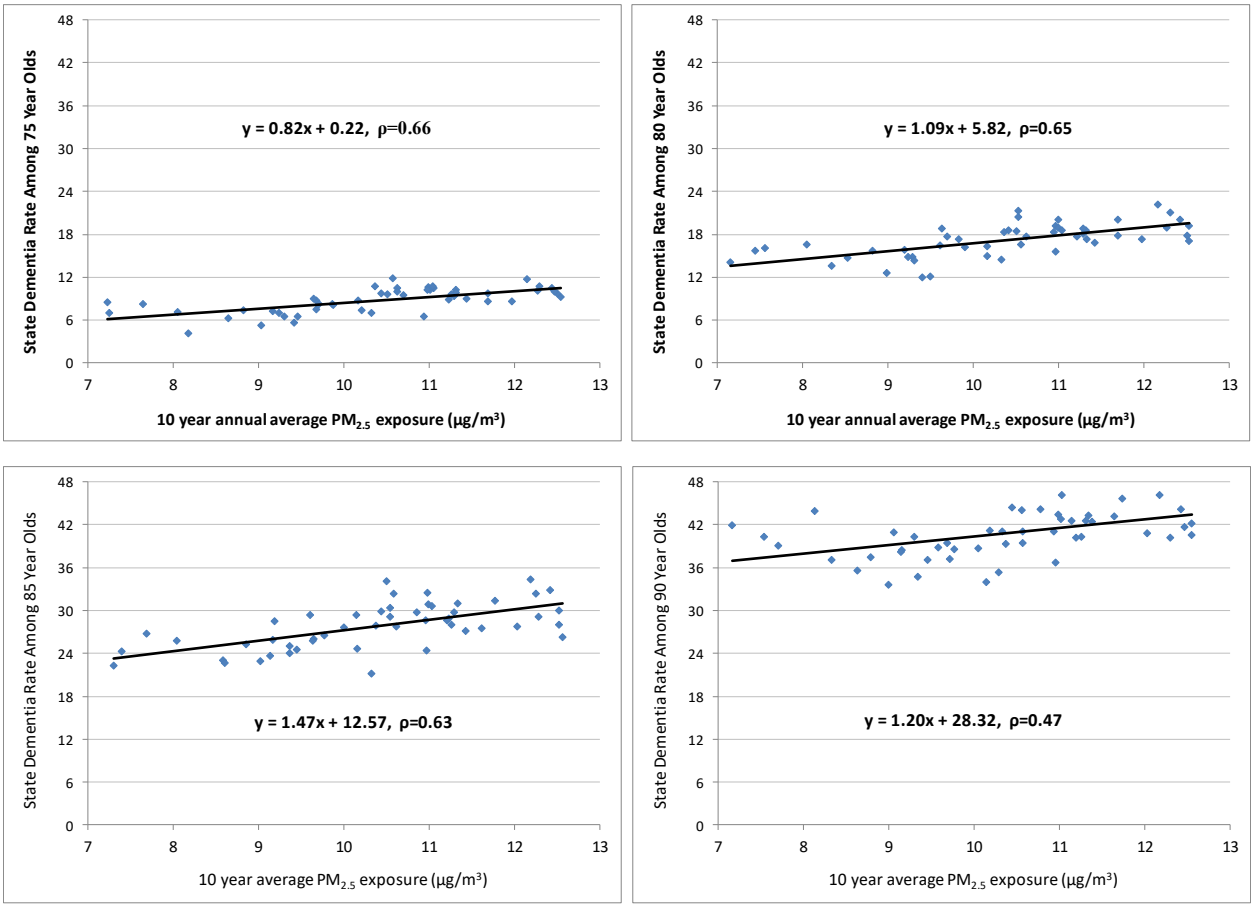
Supplemental Material: For Online Publication Only

SUPPLEMENTAL APPENDIX A: ADDITIONAL TABLES AND FIGURES

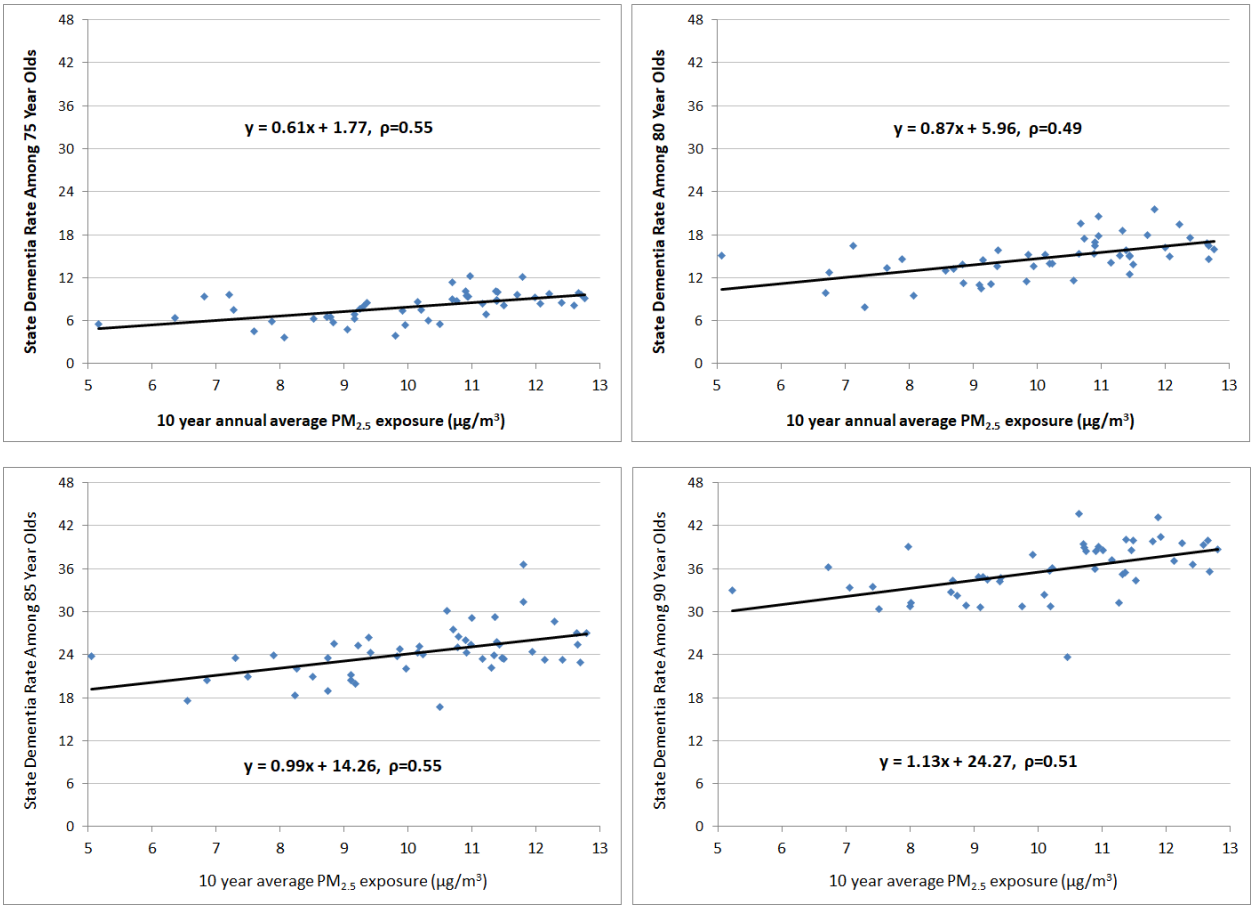
FIGURE A1: ASSOCIATION BETWEEN $PM_{2.5}$ AND DEMENTIA AMONG MEDICARE ENROLLEES, 2013



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.

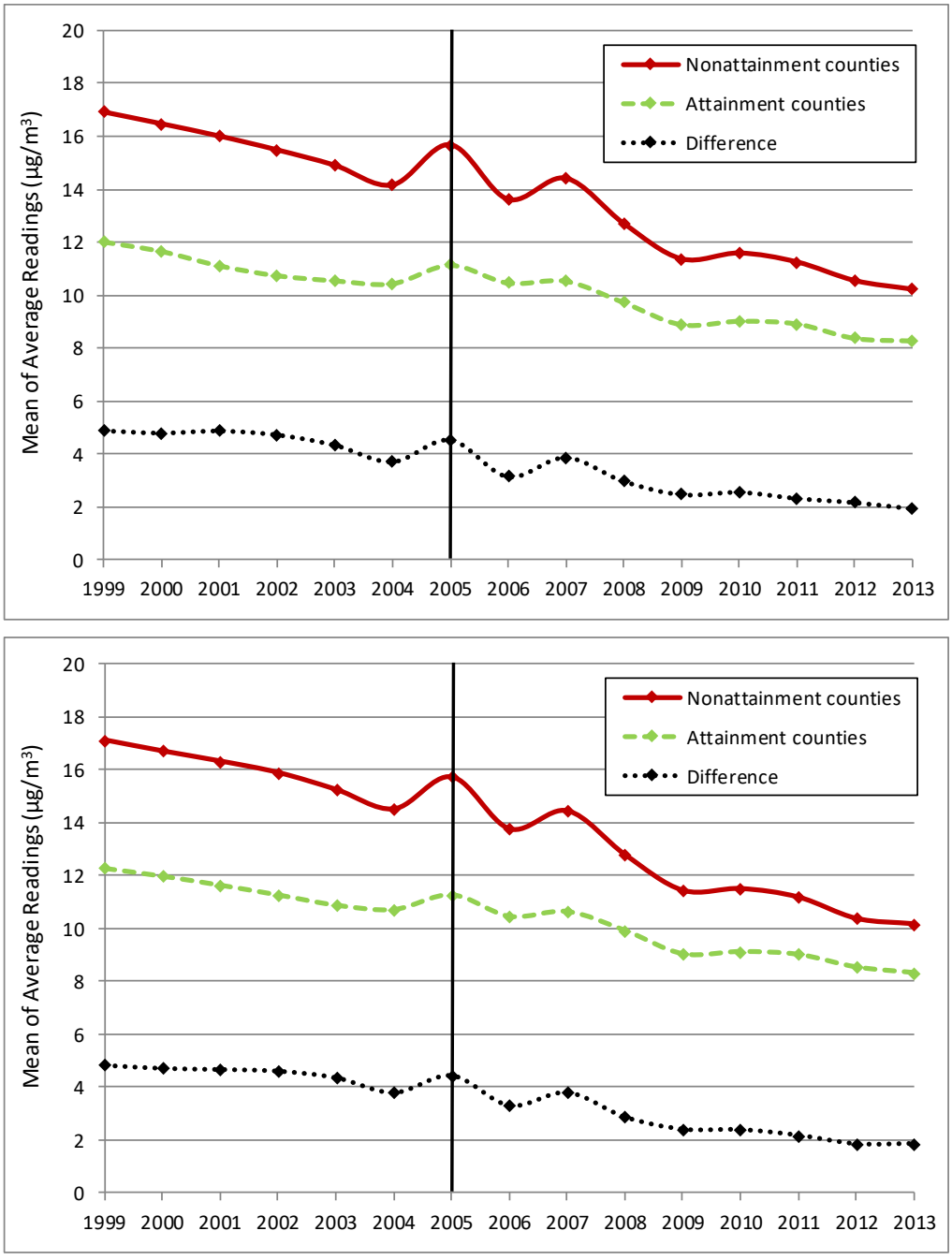
FIGURE A1 (CONTINUED):

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 people included in our main estimation sample. Differences between Figures A1 and A2 are mainly due to dropping people 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

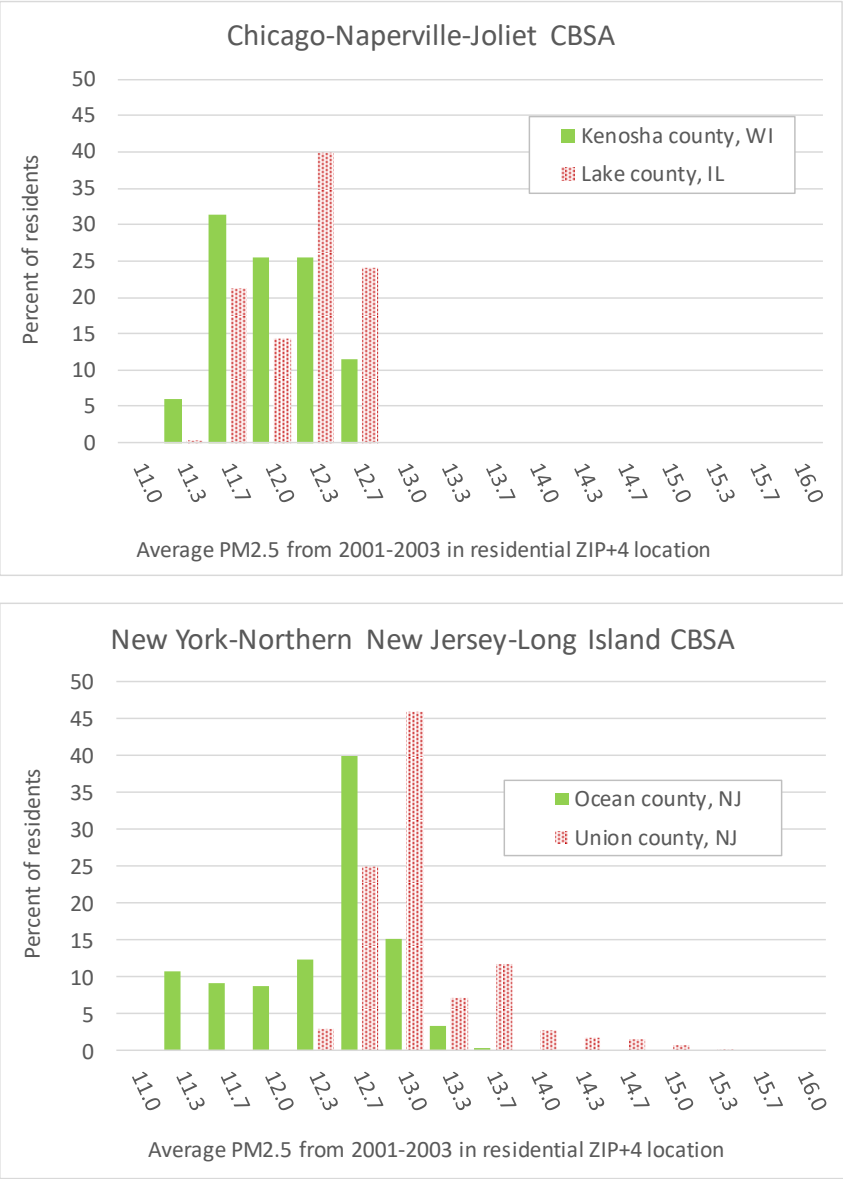
	(1)	(2)	(3)	(4)	(5)	(6)
	Main estimation sample: 2004 - 2013 survivors	Full estimation sample: traditional Medicare enrollees in 2004	EXCLUDED lived in county without pollution monitors	EXCLUDED enrolled in Medicare Advantage in 2004	EXCLUDED had dementia in 2004	EXCLUDED missing data or moved in 2004
# 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 people 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 people who were in TM in 2004 but died or switched to Medicare Advantage (MA) before 2013. Column (3) describes people 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 people 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 people 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 people 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 people 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 people 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

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 IN 2013

	(1)	(2)	(3)	(4)
decadal $PM_{2.5}$ (1 μg/m^3)	0.537***	0.365***	0.734***	2.369***
	(0.06)	(0.09)	(0.09)	(0.45)
individual & neighborhood covariates		x		x
specification	OLS	OLS	2SLS	2SLS
number of individuals	2,384,195	2,384,195	2,384,195	2,384,195
share who survive through 2013	60.5	60.5	60.5	60.5

Note: The dependent variable equals 100 if an individual died before the end of 2013. 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 person's Census block group, and pre-regulatory $PM_{2.5}$ levels at their residence from 2001-2003. Cols (3) and (4) are the 2SLS analogues to Cols (1) and (2), respectively. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered by initial Census block group.

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 μg/m^3 increase in average $PM_{2.5}$ exposure from 2004 through 2013 increased the probability of a death by the end of 2013 by 2.37 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.

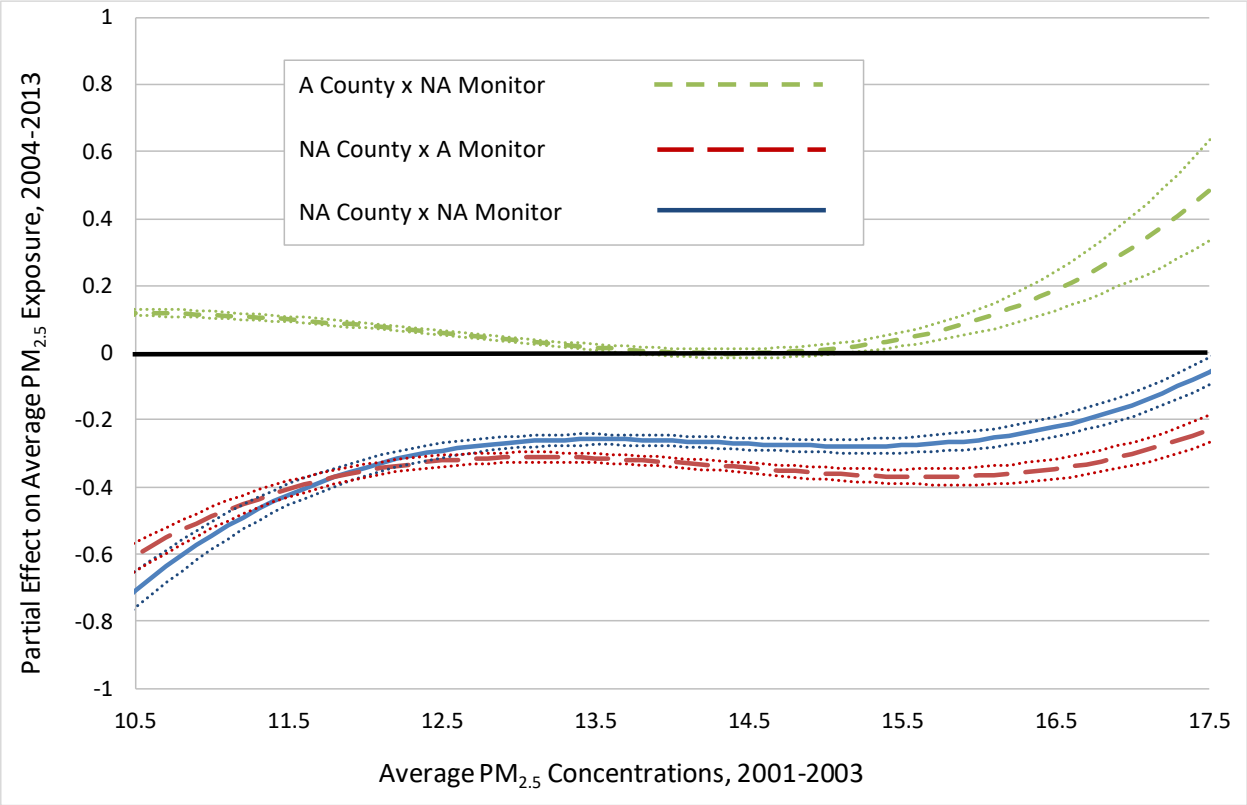
TABLE A4—COEFFICIENTS ON CANCER INSTRUMENTS IN THE SURVIVAL REGRESSION

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.

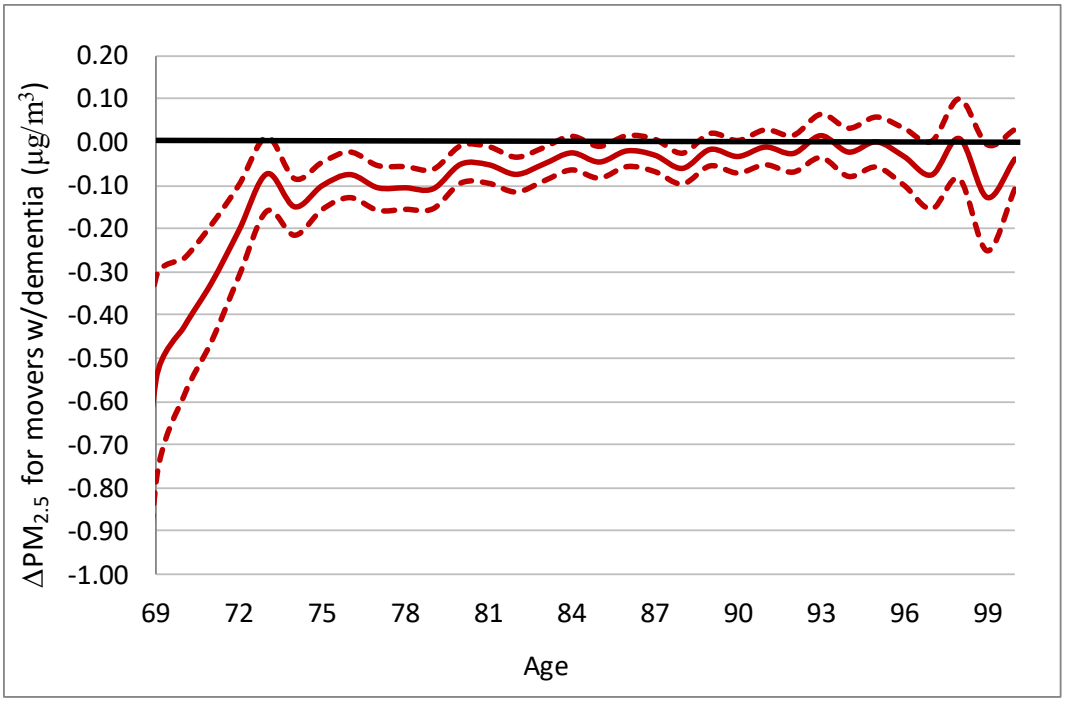
The table shows coefficients on the instruments from the survival regression. The dependent variable is scaled to enable the coefficients to be interpreted as percentage point changes in the probability of survival.

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 4^{th} 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 96% confidence bands based on 1,000 bootstrap replications, with clustering by Census block group.

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

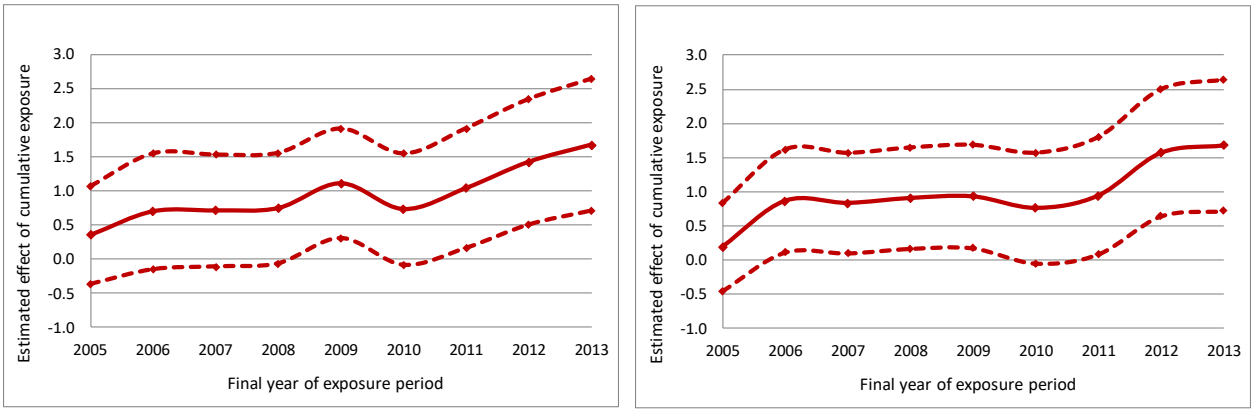


The solid trend line shows that movers with dementia tend to experience relatively larger year-to-year reductions in their $PM_{2.5}$ exposures as a result of moving, compared to non-movers of the same age (who may or may not have dementia). The dashed lines are 95% confidence bands on our estimates 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 person 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 person 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 $\mu g/m^3$ 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 people 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 people who survived to 2013.

Supplemental Appendix B: Additional Background on Policy Calculations

This appendix provides additional details regarding our estimate of the effect of dementia on people's quality-adjusted life years (QALYs). Alzheimer's disease and related dementias reduce QALYs through mortality and morbidity. We are unaware 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). Due to the health of the Medicare population even apart from dementia, each year of life lost does not represent a full 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. Together, these values imply that a dementia diagnosis on average leads to 4.88 QALYs lost due to mortality.

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 rate 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. Combining this with the loss from mortality results in a central estimate of 5.9 QALYs lost per dementia case, with a range from 5.5 to 6.4 QALYs.

We use a range of estimates for the value of a statistical life year, 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.

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

References

Ara, Roberta and John E. Brazier. 2011. "Using Health State Utility Values from the General Population to Approximate Baselines in Decision Analytic Models when Condition-Specific Data are Not Available." *Value in Health,* 14: 539-545.

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