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HAZED AND CONFUSED: THE EFFECT OF AIR POLLUTION ON DEMENTIA

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ABSTRACT

We test whether long-term exposure to air pollution degrades human capital by causing dementia. We link fifteen years of Medicare records for 6.9 million adults age 65 and older to the EPA's air quality monitoring network and track the evolution of individuals' health, onset of dementia, financial decisions, and cumulative residential exposure to fine-particulate air pollution (PM2.5). Our instrumental variables framework capitalizes on quasi-random variation in pollution exposure due to the EPA's 2005 designation of nonattainment counties for PM2.5. We find that a 1 microgram-per-cubic-meter increase in average decadal exposure (9.1% of the mean) increases the probability of receiving a dementia diagnosis by 1.3 percentage points (6.7% of the mean). This finding is consistent with hypotheses from the medical literature. We conclude that regulation of air pollution has greater benefits than previously known, in part because dementia impairs financial decision making. We estimate that the dementia-related benefits of the EPA's county nonattainment designations exceeded $150 billion. We also find that the effect of PM2.5 on dementia persists below current regulatory thresholds.

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Air pollution is known to impair human capital from the beginning through the middle of the life cycle. Among school age children, daily pollution spikes increase school absences and reduce scores on high stakes exams (Currie et al. 2009, Ebenstein, Lavy and Roth 2016). Among adult workers, daily pollution spikes reduce labor productivity in both manual and cognitive tasks (Graff-Zivin and Neidell 2012; Chang et al. 2016a,b, Archsmith, Heyes, and Saberian 2017). Prolonged exposures to elevated concentrations can have lasting consequences. For instance, Isen, Rossin-Slater, and Walker (2017) find that exposure to higher levels of air pollution during the first year of life causes people to have lower wages and lower labor-force participation during early adulthood. By contrast, little is known about the effects of long-term exposure during adulthood on human capital later in life. Burgeoning medical literature provides reason to suspect that long-term exposure to elevated pollution levels may permanently impair older adults' cognition, especially in the case of particulates smaller than 2.5 microns in diameter, commonly known as "fine particulate matter" or "$PM_{2.5}$". The small size of $PM_{2.5}$ allows it to remain airborne for long periods, to penetrate buildings, to be inhaled easily, and to reach and accumulate within brain tissue. The accumulation of particulates in the brain can cause neuroinflammation, which is associated with symptoms of dementia. While suggestive, the medical evidence implicating air pollution as a cause of dementia is based only on animal studies and small specialized human cohorts (Underwood 2017).

This article represents the first large scale national study of how long-term exposure to air pollution affects dementia. We assemble fifteen years of Medicare records on 6.9 million United States residents over age 65 to track their dementia diagnoses, other illnesses, demographics, residential exposure to air pollution, and financial decisions. These data are well-suited to studying how long-term pollution exposure affects cognitive impairment. Dementia is a syndrome of brain disease that is diagnosed when patients demonstrate multiple symptoms of cognitive impairment. Dementia is prevalent among those age 65 and above, impairing their "memory, thinking, orientation, comprehension, calculation, learning capacity, language and judgment", thereby disrupting their social relationships and limiting their abilities to perform basic activities of daily living such as managing their finances.[1] The most common form of dementia is Alzheimer's disease. About

[1] The World Health Organization's 10th revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-10) 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

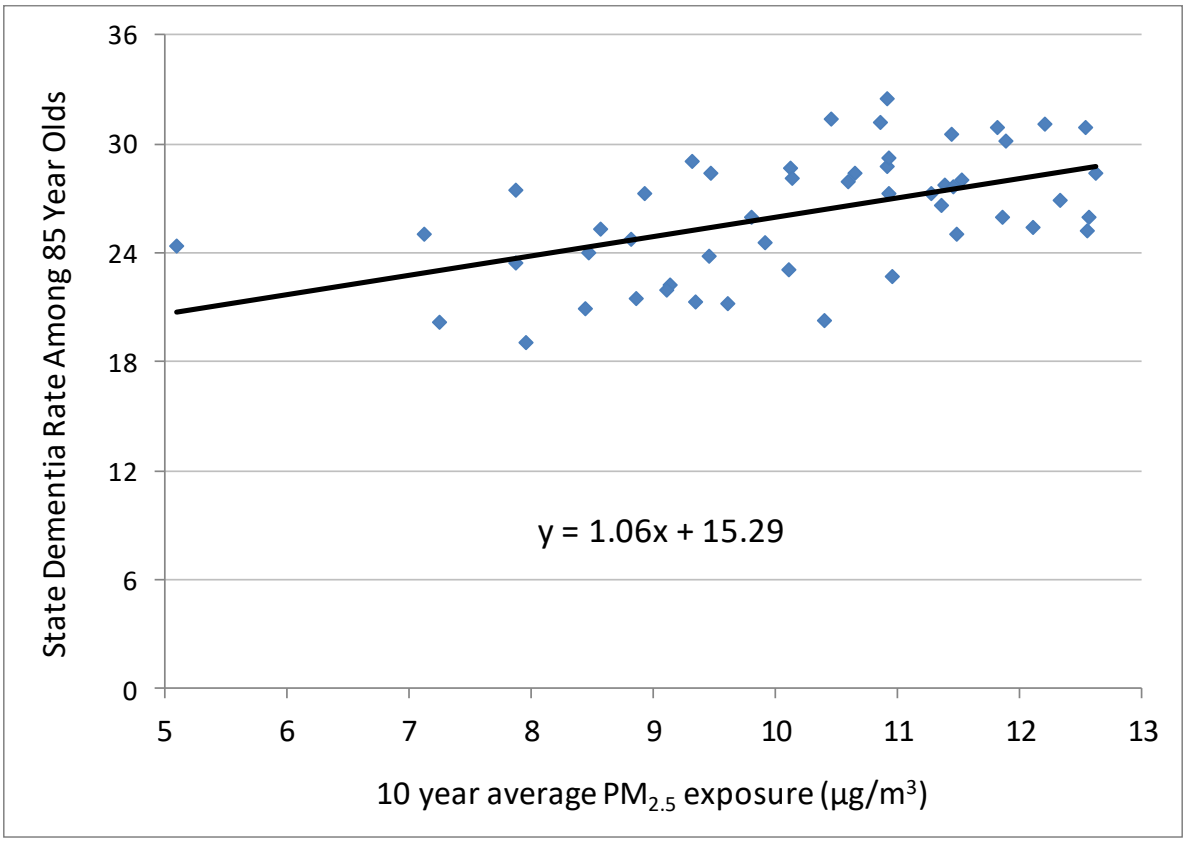
one percent of Americans are diagnosed with Alzheimer's disease and related dementia by age 65, and this share roughly doubles with each additional five years of age with about one third of all Americans over age 85 being afflicted (Querfurth and LaFerla 2010). The Alzheimer's Association (2018) reports that more than 5 million Americans are currently living with the disease.

Dementia's social costs are high and growing. In 2016, it was the fifth leading cause of death worldwide and the sixth leading cause of death in the United States. In addition to causing mortality, dementia lowers people's quality of life and increases medical spending. For instance, the Alzheimer's Association (2018) estimates that US patients will spend $277 billion on health care services in 2018, most of which will be paid by taxpayers through Medicare, and these expenditures exclude the cost of an estimated 18 billion labor hours supplied by unpaid caregivers, typically family and friends of the afflicted individuals. Furthermore, dementia erodes the cognitive skills that people use to make a host of complex and important financial decisions, such as retirement planning and health insurance choices (Lusardi and Mitchell 2014). Agarwal et al. (2009) hypothesized that dementia is a likely source of the observed decline in the quality of financial decisions as people age, but prior research has not tested this directly.

In this study we use Medicare records to determine if and when beneficiaries were first diagnosed with dementia. The data allow us to track each person's precise residential location from 1999 through 2013, including moves. We merge individual migration histories with the US Environmental Protection Agency's (EPA's) national network of air quality monitors to construct person-specific measures of long-term cumulative exposures to $PM_{2.5}$ and five other federally regulated air pollutants (particulate matter smaller than 10 microns (PM_{10}), ozone, carbon monoxide, nitrogen dioxide, and sulfur dioxide). $PM_{2.5}$ and dementia are strongly correlated across geographic areas. Figure I illustrates this correlation by plotting state-level dementia rates among 85-year-old Medicare beneficiaries in 2013 against their average residential exposure to $PM_{2.5}$ from 2004 through 2013. The diagnosis rate varies substantially across states and is strongly positively correlated with $PM_{2.5}$. The trend line indicates that a 1 microgram per cubic meter ($\mu g/m^3$) increase in average exposure between the ages of 76 and 85 is associated with a 1.06 percentage point increase in the dementia rate at age 85. We observe similar positive correlations between $PM_{2.5}$ and dementia at every age in our data (Appendix Figure A1 provides additional examples).

conditions primarily or secondarily affecting the brain."

FIGURE I: SPATIAL CORRELATION BETWEEN $PM_{2.5}$ AND DEMENTIA AT AGE 85 IN 2013



Note: Each data point represents the fraction of 85-year old individuals living in a particular state who had been diagnosed with dementia before the end of 2013, as measured on the vertical axis. The horizontal axis reports their average residential exposure to concentrations of air pollution particulates smaller than 2.5 microns from the beginning of 2004 through the end of 2013. Exposure is measured by spatial interpolation from air quality monitors to residential locations, incorporating any changes due to migration. The median state has 1,080 85-year olds.

We leverage the Medicare data to investigate whether the relationship in Figure I is causal or due to residential sorting, spatial variation in access to health care, anticipatory behavior, errors in measuring pollution exposure, or other potential confounders. First, we develop a nationwide, individual-level model that tracks the onset of dementia over a decade among those who did not have dementia at the start of the decade. Second, we control for a rich set of individual characteristics associated with dementia risk. These include flexible and comprehensive measures of age, race, gender, and pre-existing medical conditions that are associated with increased dementia risk. Third, we address the potential correlation between air pollution and aspects of health and human capital by controlling for individuals' medical expenditures at the start of the decade, the socioeconomic composition of people living in their neighborhoods (defined as Census block groups) and baseline pollution levels in those neighborhoods, while simultaneously employing high-resolution geographic fixed effects for Census core-based statistical areas. Finally, we instrument for decadal

pollution exposure by adapting the instrumental variables design employed in Chay and Greenstone's (2005) county-level analysis to our individual-level model.

In 1997 the EPA strengthened the Clean Air Act regulations to establish a federal standard on maximum-allowable $PM_{2.5}$ concentrations. In 2005, the EPA designated counties as violating this new standard, generating quasi-experimental variation in future pollution levels. These counties contained more than one quarter of all Americans over age 65. We find that people who lived in these "nonattainment" counties at the time they were targeted for enhanced regulation experienced a 1.24 $\mu g/m^3$ larger reduction in average $PM_{2.5}$ exposure over the following decade compared with people who lived in counties that did not violate the EPA standard. While prior studies have used the EPA's county nonattainment designations for particulate matter and ozone to identify effects of air pollution on a variety of health, housing and labor market outcomes (e.g., Chay and Greenstone 2005, Walker 2013, Bento, Freedman, and Lang 2015, Isen, Rossin-Slater, and Walker 2017) our study is the first to do so for the 2005 $PM_{2.5}$ standard, the first to measure long-term (i.e., decadal) air pollution exposure at the individual level, and the first to focus on dementia.

As with studies of air pollution's short-term effects on health (e.g., Schlenker and Walker 2016, Deryugina et al. 2016) we find that instrumenting for exposure increases the point estimates. We find that a 1 $\mu g/m^3$ increase in average $PM_{2.5}$ exposure over a decade (9.1% of the mean) increases the probability of being diagnosed with dementia by 1.3 percentage points (pp) (6.7% of the mean), while the comparable OLS specification yields an estimate of 0.2 pp (1% of the mean). To put our instrumental variables estimate in context, the elevated risk of dementia due to a 1 $\mu g/m^3$ increase in average decadal exposure is equivalent to the elevated risk associated with a female aging from 74 to 77, around two-thirds of the elevated risk associated with having hypertension, around one-third of the elevated risk associated with having diabetes, or one-quarter of the elevated risk associated with having congestive heart failure.

The magnitude of our main instrumental variables estimate persists across a wide variety of alternative specifications. These include employing different methods for calculating pollution exposure, utilizing different sources of variation in pollution exposure, excluding people who move after receiving a dementia diagnosis, and repeating the estimation after aggregating Medicare records to the county level. To test for biases due to residential sorting on unobserved health and other threats to identification, we estimate the same instrumental variables model for other chronic ill-

nesses that share similarities with dementia but are not *a priori* suspected to be caused by air pollution. These placebo tests yield point estimates that are mostly small and statistically indistinguishable from zero, supporting our research design.

Additional results indicate that our findings reflect the degree to which long-term exposure to fine particulate matter increases the risk of Alzheimer's disease. First, we demonstrate that our findings are not explained by other federally regulated air pollutants that are correlated with $PM_{2.5}$. Second, we demonstrate that our findings are not driven by vascular dementia that may arise due to short-term pollution spikes leading to strokes. Finally, we show that about 90% of the effects of $PM_{2.5}$ on all types of dementia are explained by Alzheimer's disease specifically.

Our results have several implications for policy. We estimate that enforcement of the EPA's 1997 standard on maximum allowable $PM_{2.5}$ concentrations led to air quality improvements in newly regulated counties that averted approximately 140,000 people living with dementia in 2013, yielding $163 billion in benefits under moderate assumptions about the value of the quantity and quality of life. Furthermore, by reducing dementia rates the EPA regulations improved Medicare beneficiaries' financial decisions, as we illustrate in the context of prescription-drug insurance markets. Finally, we find that the effects of $PM_{2.5}$ on dementia persist at levels below the EPA's current regulatory threshold, suggesting that further regulation would yield additional benefits. Overall, our findings indicate that the social costs of air pollution and the benefits of regulation on human cognition are substantially larger than previously known.

I. Background on Known Links between Air Pollution, Human Capital, and Dementia

In addition to constraining the production of human capital among children and young adults, air pollution degrades the stock of human capital by increasing morbidity and mortality (Chay and Greenstone 2003, Currie and Neidell 2005, Schlenker and Walker 2016, Deschenes, Greenstone and Shapiro 2017, and others reviewed in Graff-Zivin and Neidell 2013). Older adults are also vulnerable. For example, Deryungina et al. (2016) used an instrumental variables regression to conclude that a 1 $\mu g/m^3$ increase in $PM_{2.5}$ over a three-day period caused 2.7 life years to be lost per million Medicare beneficiaries.[2] Di et al. (2017) used a Cox proportional hazards model to conclude that a 1 $\mu g/m^3$ increase in annual average $PM_{2.5}$ increased the mortality rate by 0.7%

[2] They also find that this same short-term increase caused 2.3 additional emergency room visits and $15,000 in additional medical expenditures per million beneficiaries.

among Medicare beneficiaries.

Conditional on the stock of human capital, hourly and daily spikes in pollution levels have been found to reduce adults' performances in laboratory tests of cognition as well as their job productivity (Graff-Zivin and Neidell 2013, Chen et al. 2017b). Research has implicated several pollutants, with an emphasis on $PM_{2.5}$ because its small size allows it to penetrate buildings and pollute indoor air (Graff-Zivin and Neidell 2013).

Recent medical studies have proposed multiple pathways by which air pollution, and $PM_{2.5}$ specifically, may cause dementia. First, pollution exposure is linked to increased risk for strokes and subsequent vascular dementia among older adults (Wellenius et al. 2012). Second, postmortem analysis has detected that $PM_{2.5}$ had accumulated in human brains (Maher et al. 2016). People living in more polluted areas, such as near roadways, for long periods tend 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). Third, controlled exposure of mice to air pollution in laboratory experiments results in neuroinflammation and patterns of brain cell damage similar to postmortem analysis of Alzheimer's patients (Block et al. 2012). 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 existing evidence on pathways linking air pollution to dementia is based on non-human mammal studies and specialized human cohorts, such as people who chose to live near major roadways.[3]

II. Data

We use data on Medicare beneficiaries to identify the effects of $PM_{2.5}$ on dementia and of dementia on decision making. All US citizens age 65 and above are eligible for Medicare benefits. The US Centers for Medicare and Medicaid Services (CMS) maintains a national database of beneficiaries' administrative records, including information on their residential address histories, medical claims, demographics, and enrollment decisions for prescription drug insurance plans. We use these records to develop a novel longitudinal database on long-term exposure to air pollution, health outcomes, healthcare utilization, and insurance-plan enrollment.

We start with a random 10% sample from the universe of Medicare beneficiaries who were at

[3] One larger scale study, by Chen et al. (2017a), found that living near major roadways is associated with substantial increases in the incidences of dementia and cognitive impairment. Their study leveraged administrative data to define a population-representative cohort for Ontario, Canada. However, they did not observe individuals' exposures to individual pollutants, baseline health and migration patterns.

least 65 years old in 1999. We then add random 10% samples of all new 65-year-old Medicare beneficiaries each year from 2000 to 2013. Finally, we obtain 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.[4]

After compiling the union of these samples, we perform sample cuts that drop just under half of all individuals. First, we drop people who ever enrolled in Medicare Advantage plans, which replace traditional Medicare with a managed care plan. CMS lacks data on these individuals' dementia diagnoses. Second, we drop people who cannot be matched to a precise residential location at any point during the period 1999 to 2013. This includes addresses that are post-office boxes or incomplete address records. Our sample cuts are unlikely to compromise external validity. Appendix Tables A1 reports summary statistics for our estimation sample and the excluded subsets. The excluded individuals are similar to those in our estimation sample in terms of their average demographics, longevity and, when observable, medical conditions, health expenditures, pollution exposure, and Census block-group demographics.

Our sample consists of 6.9 million individuals whom we observe for 55.4 million person-years. Approximately 44% of these individuals are male and 83% are white. The mean age upon entering our sample is 71. This reflects an average taken over the random sample of Medicare beneficiaries in the first year of our data (1999) and the beneficiaries who enter our panel in subsequent years when they turn 65 and become eligible for Medicare benefits. Once an individual enters our sample, we follow them through the end of 2013 or until they die. Approximately 60% of individuals survive through the end of 2013. For those who die, the mean age at death is 83.

We observe where each person lives each year since entering our sample, their annual medical expenditures, and if and when they are diagnosed with dementia and other chronic illnesses. For the subset who choose to enroll in prescription-drug insurance plans (PDP) through the Medicare Part D markets (1.1 million people), we also observe their annual PDP choice sets, enrollment decisions, prescription drug claims and expenditures on plan premiums, and out-of-pocket costs over the first five years the markets existed (2006-2010). We use this information to construct a series of metrics that have been used in prior literature to assess the quality of financial decisions.

[4] We exclude those receiving low-income subsidies because they are auto-enrolled into plans. This contrasts with individuals in the subsidy ineligible population who must actively select a plan to become insured. For this reason, prior studies of decision making in the Medicare Part D PDP markets have excluded those receiving low-income subsidies.

These metrics are described in Section VI.B.

Finally, we use these data to define sub-samples comprised entirely of individuals for whom we can match decadal pollution exposure to new dementia diagnoses. Specifically, our micro-level analysis focuses on a cohort of approximately 1.8 million individuals who were at least 65 years old in 2004, had not received a dementia diagnosis by the end of 2004, and were still alive in 2013.[5] Nineteen percent of this cohort had been diagnosed with dementia by the end of 2013. Following this cohort allows us to investigate how variation in decadal pollution exposure arising from new environmental regulations affected the probability of a dementia diagnosis conditional on survival.

A. Clinical Measures of Dementia and Known Risk Factors

For each person who receives a dementia diagnosis, we observe the initial diagnosis date in CMS's Chronic Conditions Data Warehouse file. This file tracks if and when each individual is diagnosed with a specific chronic medical condition using insurance claim codes. A diagnosis of dementia (as officially defined in footnote 1) is based on the presence of multiple symptoms of cognitive impairment that significantly impact daily functioning. Examples include memory loss, impaired judgement, loss of spatial awareness, depression, and behavioral changes.

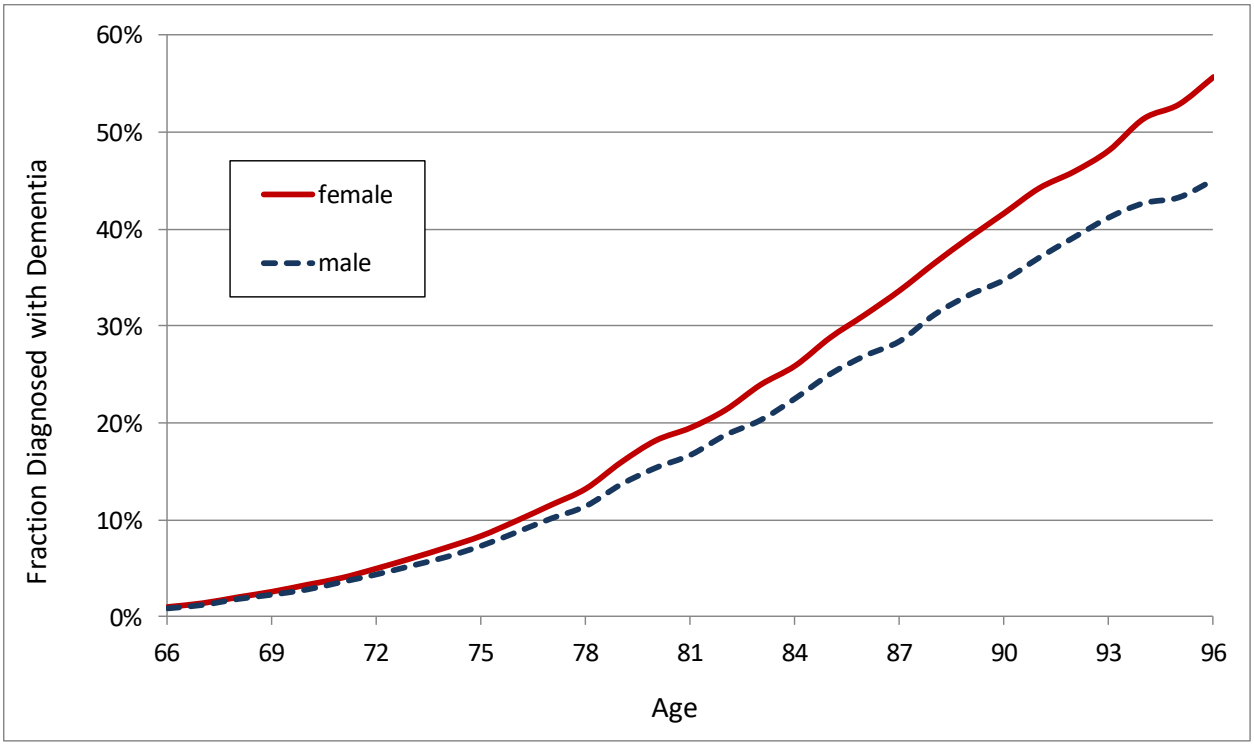
Twenty-three percent of people in our sample receive a dementia diagnosis during our study period. Figure II shows how the fraction of people living with a diagnosis varies with age and gender in 2013. Approximately 1% of our sample receives a diagnosis before the age of 66. Diagnosis rates increase 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.

According to the Alzheimer's Association (2018), physical risk factors for dementia include chronic medical conditions that reduce the flow of blood and oxygen to the brain. Appendix Table A1 shows that most individuals in our data are diagnosed with at least one of these risk factors during our study period: stroke (19%), diabetes (32%), congestive heart failure (36%), ischemic heart disease (48%) and hypertension (71%). Additional behavioral factors believed to reduce the risk of dementia include higher educational attainment, better nutrition and overall physical health,

[5] Table A1 summarizes their individual demographic characteristics, medical diagnoses, Census block group demographics and exposure to air pollutants in 2013.

and a higher degree of social and cognitive engagement. Because we are unable to observe these behaviors at the individual level, we proxy for them using the average characteristics of people living in each individual's Census block group. 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.

FIGURE II: DEMENTIA BY AGE AND GENDER IN 2013



B. Using Residential 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 (also referred to as nine-digit ZIP Codes) for each individual's sequence of home addresses from 1999 to 2013. ZIP+4 Codes are close to street addresses in terms of spatial precision: each code corresponds to a single mail delivery segment such as 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, equating to about one for every four households.

Migration patterns of the individuals in our sample are similar to those reported by the Census

Bureau for individuals aged 65 and above. More than 80% of individuals live in the same ZIP+4 throughout our study period. Of the 18% of people who move between ZIP+4 Codes at least once, 10% move between counties and 5% move between states.[6] We use this information to measure each individual's long-term exposure to air pollution, accounting for migration.[7]

Individuals in our sample live in 9.8 million distinct ZIP+4 Codes between 1999 and 2013. We measure annual air pollution at the centroids of each of these areas, focusing on six criteria pollutants regulated by the Environmental Protection Agency (EPA). In addition to $PM_{2.5}$, these include PM_{10}, ozone, carbon monoxide, nitrogen dioxide and sulfur dioxide. Annual data on ambient pollutant concentrations are drawn from the EPA's air quality system, consisting of an unbalanced panel of 6,679 monitors in operation between 1999 and 2013.[8] To approximate annual average concentrations in each ZIP+4 Code, we use the latitudinal and longitudinal coordinates of each monitor along with the coordinates of each ZIP+4.[9] Specifically, we use the Great Circle algorithm to calculate the surface distance from each ZIP+4 centroid to each monitor.[10] Then, for each centroid-pollutant-year combination, we calculate a weighted average of ambient concentrations recorded at all operating monitors with the weights given by the square of the inverse distance.[11] Thus, as the distance from a ZIP+4 centroid to a monitor increases, the weight assigned to that monitor decreases. Finally, we combine the resulting set of 882 million local pollution readings (9.8 million centroids by 6 pollutants by 15 years) with individuals' residential ZIP+4 histories to construct individual-specific exposure histories.

These exposure histories are the most comprehensive data ever developed to study how air pollution affects cognition. Nevertheless, like all methods for estimating 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

[6] Among those who ever move between ZIP+4 Codes 73% move once during our study period, 19% move twice, 5% move three times and 2% move four or more times.

[7] 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 for administrative purposes. 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.

[8] Appendix Figure A2 maps the locations of monitoring stations for each pollutant. The six criteria pollutants that we study are tracked at between 794 and 2,010 monitoring stations from 1999 to 2013. For example, there were 1,797 monitoring stations for $PM_{2.5}$. The EPA also regulates lead as a criteria air pollutant but had far fewer monitors (477) during our study period and less nationally representative coverage.

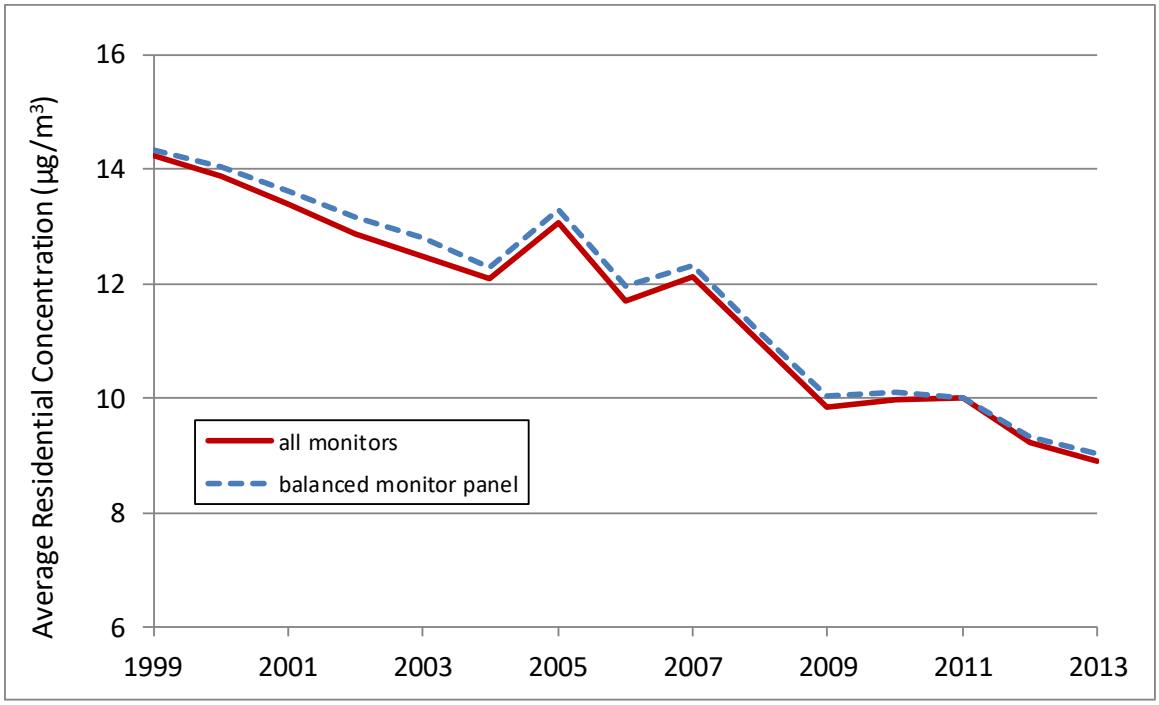
[9] Geographic coordinates of ZIP+4 centroids were purchased from GeoLytics, which created them from the Census Bureau's TIGER/line Shapefiles and US Postal Service records.

[10] In other words, we use the geographic coordinates of both the ZIP+4 Codes and the monitors to calculate the shortest distance between each pair on the surface of the spherical Earth.

[11] This method of interpolation, with weights given by the distance raised to a negative exponent, is the predominant method in the literature going back to Shepherd (1968). Related examples include Currie and Neidell (2005) and Bishop and Timmins (2018).

outside of the home, variation in indoor air penetration rates due to heterogeneity in home sealing, and variation in respiration due to health and physical activity. Like prior studies, we develop instrumental variables to address potential attenuation bias (Chay and Greenstone 2003, Schlenker and Walker 2016).

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



Note: The figures report 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 III shows that annual average residential exposure to $PM_{2.5}$ declined from about 14 $\mu g/m^3$ in 1999 to about 9 $\mu g/m^3$ in 2013. This is true regardless of whether we measure exposure using the unbalanced panel of all monitors in operation each year (solid line) or a balanced panel of monitors that operated 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 state and county officials responded to increased federal regulation of $PM_{2.5}$ during the mid-2000s by strategically locating new monitors in cleaner areas (Muller and Rudd 2017, Grainger, Schreiber and Chang 2018).[12]

[12] Our results are robust to this modeling decision, as shown in Section IV.B.

III. Research Design

Our research design combines the quasi-experimental approach to identification from economic studies of air pollution (e.g., Chay and Greenstone 2005, Walker 2013, Bento, Freedman, and Lang 2015, Isen, Rossin-Slater, and Walker 2017) with the logic of a cohort design similar to those used in medical and epidemiological studies (e.g., Pope et al. 2002, Landen et al. 2006, Di et al. 2017). In particular, we analyze how decadal exposure to air pollution affects the probability of new dementia diagnoses using quasi-random variation in pollution exposure resulting from Clean Air Act regulations.

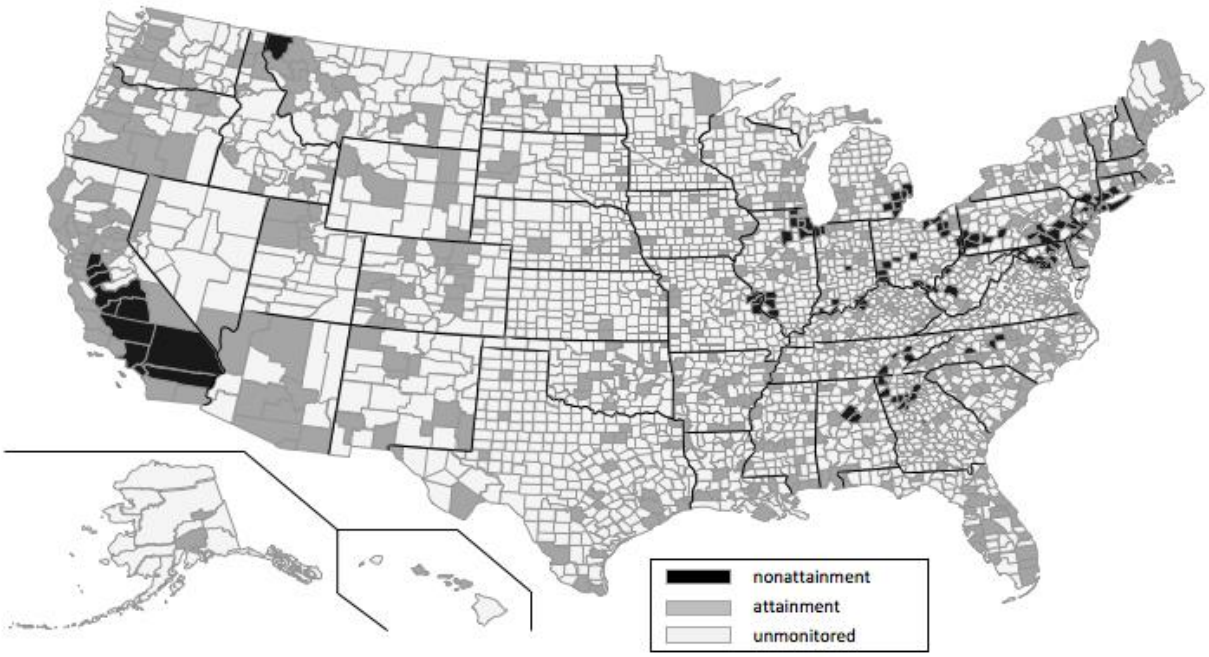
A. Clean Air Act Regulations as a Quasi-Random Source of Variation in $PM_{2.5}$ Exposure

A signature feature of the Clean Air Act is the establishment of national standards for maximum-allowable, county-level concentrations of particulate matter, ozone, carbon monoxide, sulfur dioxide, nitrogen dioxide and lead. Counties that violate these standards are designated as being in "nonattainment" by the EPA. States are then responsible for developing implementation plans to ensure that nonattainment counties reduce concentrations enough to meet the national standards. States that fail to bring their counties into attainment risk losing their federal highway funds and may face additional federal penalties, thereby spurring local regulation.

Among the regulated pollutants, particulate matter is believed to have the most pernicious effects on human health at commonly observed concentrations (US EPA 2011). Beginning in 1971, the EPA regulated total suspended particulates (TSP). In light of evidence that health effects were being 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. Enactment of each new standard was followed with new nonattainment designations at the county level. These nonattainment designations caused the regulated counties to have relatively large reductions in particulate matter. Importantly, households, workers, and firms would have been unlikely to have anticipated these reductions when making prior location decisions. Following this logic, Chay and Greenstone (2005) and Isen, Rossin-Slater, and Walker (2017) use county attainment status for TSP as an instrument for changes in TSP concentrations, while Walker (2013) and Bento, Freedman, and Lang (2015) develop instruments based on attainment status for PM_{10}. In this paper, we exploit the most recent target of

the EPA's county attainment designations: the 1997 $PM_{2.5}$ standard.[13]

FIGURE IV: 2005 COUNTY 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. Almost all of the unmonitored counties were defined by the EPA as "unclassifiable" because they lacked the air quality monitor data needed to determine attainment status.

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. After several years of litigation failed to overturn this new standard, the EPA made initial county nonattainment designations in 2005 based on monitor readings from 2001 to 2003.[14] 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. Remaining counties that lacked the monitoring data needed to make a clear designation were generally defined as "unclassifiable" and not subjected to additional regulation (US EPA 2005).[15] The map in Figure IV shows the locations

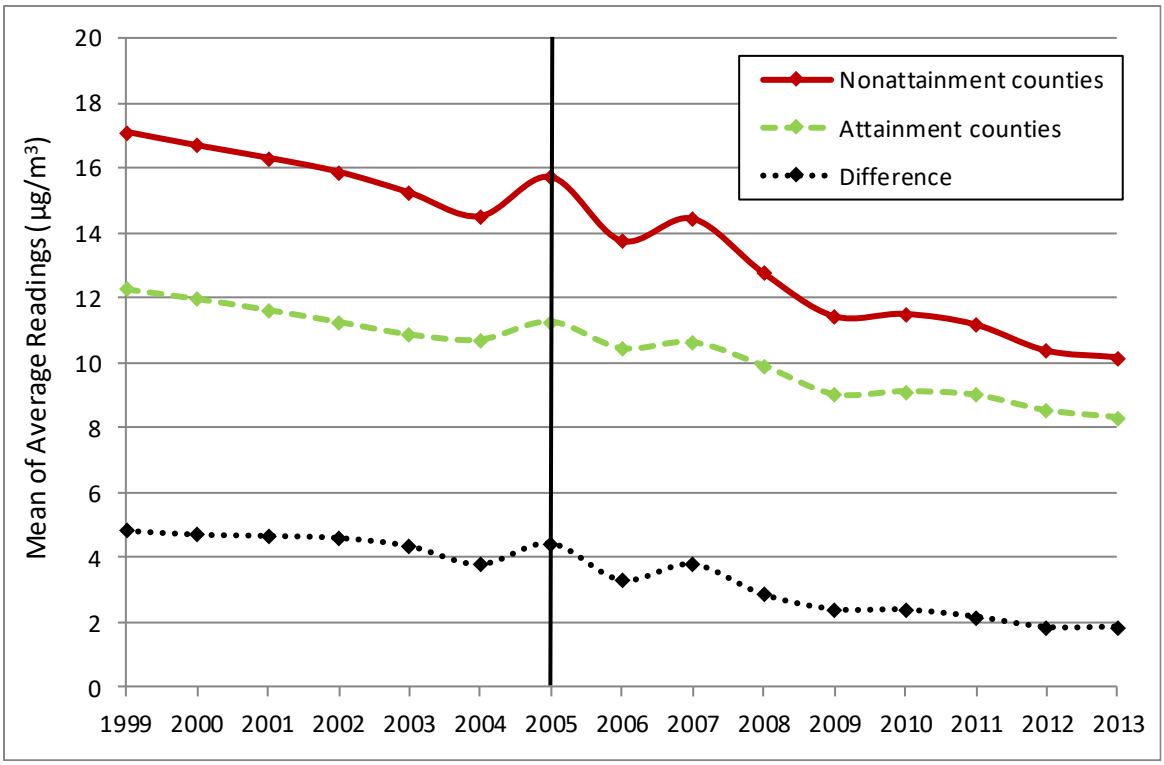
[13] We have kept our review of the institutional details brief because the rationale for using county nonattainment status as an instrument for changes in air pollution because it has been thoroughly explained by prior studies. Readers seeking additional background on federal regulation of air pollutants should see the discussions in Kahn (1997), Chay and Greenstone (2005), Walker (2013) and US EPA (2005).

[14] 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).

[15] Exceptions to this rule occurred for unmonitored counties that were believed to contribute to violations in nearby monitored counties. In such

of attainment and nonattainment counties with air quality monitors. States were directed to ensure that nonattainment counties met the 15.05 μg/m^3 standard by 2010.[16]

FIGURE V: $PM_{2.5}$ CONCENTRATIONS BY COUNTY ATTAINMENT STATUS IN 2005



Note: The figure reports annual average concentrations of particulates smaller than 2.5 microns ($PM_{2.5}$). Measurements are taken from air quality monitors in counties designated in 2005 as attainment or nonattainment with the federal standard. Each data point in the nonattainment line is a simple average over monitors in nonattainment counties that were in continuous operation from 2001-2013. The attainment county line is defined similarly. The bottom line shows the difference between the nonattainment and attainment lines. 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. Corresponding general population shares were 43% (attainment) and 28% (nonattainment).

Figure V provides initial evidence that nonattainment designations caused the newly regulated counties to improve air quality. The solid and dashed lines show the trends in annual average $PM_{2.5}$ readings for nonattainment and attainment counties.[17] Prior to 2005, $PM_{2.5}$ concentrations were trending downward similarly in both attainment and nonattainment counties. The dotted line shows

cases, the unmonitored counties were classified as nonattainment (US EPA 2005). 76 counties in our data fit this description.

[16] The regulation allowed for potential extensions of up to 5 years at the discretion of the EPA administrator. Guidelines for state implementation plans were released in 2007 and new source review standards were released in 2008. The EPA also indirectly regulated particulate matter through the Clean Air Interstate Rule, enacted in March 2005 to mitigate interstate transport of $PM_{2.5}$ precursors, and through new regulations on emissions from mobile sources.

[17] The figure is based on a balanced panel of 488 monitors in operation continuously from 2001-2013. Appendix Figure A3 shows that the figure looks virtually identical if we reconstruct it from an unbalanced panel of all operating monitors that satisfied EPA's monitoring standards for regulatory decisions, had at least 10 readings per year, and did not exclude events such as forest fires (between 787 and 1,106 per year). It also looks virtually identical if we reconstruct it from the balanced panel of 393 monitors that were in continuous operation from 1999 through 2013.

that the difference between the two trend lines was fairly stable from 1999 to 2005.[18] After the nonattainment designations were made, $PM_{2.5}$ concentrations declined at a noticeably faster rate in nonattainment counties. The difference between the average monitor readings in nonattainment and attainment counties dropped by more than half between 2005 and 2013. Figure V mirrors the analysis in Chay and Greenstone (2005) of the 1975 attainment designations for TSP (see Figure 2 in that paper), suggesting that little has changed in the way that local regulators respond to federal incentives for reducing air pollution. Similar to that study, we use county attainment status as an instrumental variable for changes in particular matter concentrations over the subsequent decade.[19]

B. Main Econometric Model

Let $D_{i,t}$ be an indicator for whether person *i* is diagnosed with dementia before the end of year *t*. We restrict our Medicare sample to the cohort who were at least 65 years old in 2004, who had not received a dementia diagnosis at that point, $D_{i,2004} = 0 \; \forall \; i$, and who were still alive in 2013. We define an indicator, $y_i = D_{i,2013} - D_{i,2004}$, describing whether person i receives a dementia diagnosis by the end of 2013. This y_i indicator is the dependent variable in our linear probability model,

$$(1) \quad y_i = \alpha \sum_{s=2004}^{2013} \frac{PM2.5_{i,s}}{10} + \eta_{i,2013} + \beta X_{i,2013} + \gamma H_{i,2004} + \theta W_{i,2004} + f\left(\sum_{s=2001}^{2003} \frac{PM2.5_{i,s}}{3} \right) + \epsilon_i.$$

The coefficient of interest in Equation (1), α, measures the effect of 10-year average residential exposure to $PM_{2.5}$. This cumulative exposure measure is derived from each person's residential location history so it incorporates changes in pollution experienced as a result of moving.

We draw on administrative Medicare records to control for individual and neighborhood characteristics that may be correlated with both dementia and $PM_{2.5}$. First, we add dummy variables, $\eta_{i,2013}$, for the approximately one thousand Core Business 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.[20] Examples include extreme temperatures, the presence of lead

[18] The slight drop in 2004 could reflect spatiotemporal variation in weather and economic activity or a preemptive response to anticipated future regulation. In any case, our econometric models of decadal exposure define 2004 as the first year of the exposure decade, while controlling for observable pre-decadal exposure.

[19] Chay and Greenstone's preferred version of this instrument was an indicator for mid-decade attainment status. They preferred this partly because using mid-decade attainment status limited the scope for unobserved spatial sorting by households. By contrast, we observe migration at the individual level.

[20] We create a state-specific dummy variable for people living in rural areas outside CBSAs.

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.[21]

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 $X_{i,2013}$ vector includes indicators for race and gender-specific indicators for integer age at the end of 2013 (from 74 through 99).[22] These flexible age-by-gender controls absorb the nonlinear trends in dementia rates shown in Figure II.

The $H_{i,2004}$ 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.[23] 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.[24]

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, $W_{i,2004}$, 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 fractions of residents in each of seven educational-attainment bins. These neighborhood-level measures also serve to control for within-CBSA heterogeneity in other neighborhood amenities known to attract wealthier households with higher education (Kuminoff, Smith and Timmins 2013).

[21] Additionally, for the majority of people who never move during our study period, the CBSA dummies will control for any pre-period sorting across CBSAs on the basis of latent characteristics that may serve as risk factors for dementia (Finkelstein, Gentzkow and Williams 2016).
[22] 74 is the minimum age in 2013 because 65 is the minimum age at the start of the decade. 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.
[23] 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.
[24] Medicare Parts A and B cover virtually all medical services aside from prescription drugs. This includes doctors' services, preventive care, durable medical equipment, hospital out-patient services, laboratory tests, x-rays, hospital in-patient services, nursing facilities, and hospice care.

Finally, we add a fourth-order polynomial function, $f(\cdot)$, in baseline $PM_{2.5}$ exposure. We calculate this baseline exposure using annual averages over the EPA's monitoring window from 2001 through 2003 at person i's residential location in 2004. This controls for any residual effects of pre-sorting into more polluted neighborhoods by people who are more likely to receive a future dementia diagnosis. Controlling for baseline exposure also modifies the interpretation of α in equation (1) to be similar to a first-differenced model. That is, α measures how the change in cumulative $PM_{2.5}$ exposure from 2004 to 2013 affects the probability of being newly diagnosed with dementia. Measuring the dependent variable as a diagnosed change in cognition also purges an individual fixed effect from the econometric model.

Despite the rich set of controls in Equation (1), two threats to identification remain. Measurement error in pollution exposure is our primary concern. A secondary concern is omitted variable bias, though our controls are chosen to substantially mitigate this concern. We address both of these issues by instrumenting for decadal pollution exposure with an indicator for county attainment status, as shown in Equation (2).

$$(2) \quad \sum_{s=2004}^{2013} \frac{PM2.5_{i,s}}{10} = \pi Z_i + \eta_{i,2013} + \sigma X_{i,2013} + \tau H_{i,2004} + \omega W_{i,2004} + f\left(\sum_{s=2001}^{2003} \frac{PM2.5_{i,s}}{3}\right) + \varepsilon_i.$$

The instrument takes a value of one for individuals who resided in counties that were designated as nonattainment in 2005. The Z_i vector interacts this instrument with the polynomial function of baseline pollution exposure that enters the second stage model to allow regulatory responses to vary with distance from the attainment threshold.

Thus, α is identified by variation in decadal exposure to $PM_{2.5}$ experienced by people of the same age, race, and gender who, at the start of the decade, had received the same medical diagnoses for dementia risk factors, had the same amount 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. Holding all of these factors fixed, some people lived in counties that were newly regulated as a result of the EPA's $PM_{2.5}$ standard while others did not; those living in newly regulated counties were exposed to less $PM_{2.5}$ on average over the next 10 years (shown in Section IV.A). This identification framework provides a micro-level research design similar to the aggregate, county-level research design developed in Chay and Greenstone (2005).

C. Econometric Model with Aggregated Data

As a robustness check on our micro-level specification and a methodological comparison to prior literature, we aggregate Medicare administrative records to the county level and model changes in county-level dementia rates. Similar to Chay and Greenstone (2005), we estimate a first-differenced model using 2SLS with an indicator for county-level nonattainment status as the instrument. Our second-stage model regresses county-level changes in dementia rates on changes in their residents' cumulative exposure to $PM_{2.5}$.

$$(3)\quad \Delta y_j = \alpha \Delta \text{PM2.5}_j + \delta_j + \beta \Delta X_j + \gamma_1 H_{j,2004} + \gamma_2 \Delta H_j + \theta \Delta W_j + \varphi \Delta C_j + \Delta \epsilon_j.$$

The outcome in (3) is the change between 2004 and 2013 in the fraction of people living with a dementia diagnosis in county *j* and the focal variable, $\Delta PM2.5_j$, denotes the decadal change in the average county resident's cumulative exposure to $PM_{2.5}$ relative to the baseline period (2001-2003).[25]

$$(4)\quad \Delta \text{PM2.5}_j = \sum_{t=2004}^{2013} \frac{PM2.5_{j,t}}{10} - \sum_{s=2001}^{2003} \frac{PM2.5_{j,s}}{3}.$$

Formulating the model in changes purges time-invariant latent characteristics of counties that may lead to persistently higher dementia rates such as pre-period pollution levels, climate, and supply of retirement communities or long-term care facilities that may attract migrants who are at higher risk of dementia.

The remaining covariates parallel our micro-level specification in that they control for time-varying features of counties that may be correlated with changes in pollution and dementia. They include dummy variables for the nine Census divisions (δ_j) to absorb regional trends in diagnosis rates, changes in the distribution of people living in county *j* in terms of integer age, gender, and race (ΔX_j), the fraction of people diagnosed with each morbidity risk for dementia at the beginning of the decade (hypertension, diabetes, congestive heart failure, ischemic heart disease and stroke), the average resident's 2004 medical expenditures ($H_{j,2004}$) along with changes in each of those

[25] The 2001-2003 monitoring period is a natural choice for measuring baseline exposure because, as we explain above, it was used to determine a county's attainment status. Our model controls for cumulative lifetime pollution exposure prior to 2001 experienced by the average resident of each county via the county fixed effects purged by measuring the dependent variable in differences.

variables due to turnover in county j's population (ΔH_j), and changes in the demographic composition of people living in the average resident's Census block group (ΔW_j). Finally, we proxy for changes in access to medical care (ΔC_j) using changes in hospital beds per capita, changes in medical doctors per capita, changes in the fraction of residents enrolled in Medicare Advantage (e.g., to account for the possibility that those at lower risk of dementia are more likely to select out of our sample and into Medicare Advantage), and changes in what Medicare pays physicians as captured by the Geographic Practice Cost Indices (GPCIs).[26]

IV. Results

A. Results from the Main Econometric Model

i. Second Stage Results

Table I summarizes our main results. The dementia indicator is multiplied by 100 so that the linear probability model coefficients on $PM_{2.5}$ may be interpreted as percentage point (pp) changes in the probability of receiving a dementia diagnosis. Standard errors are robust to heteroscedasticity and clustered at the Census block group level to allow for spatial correlation in diagnoses. Column (1) shows the result from a univariate OLS regression. A 1 $\mu g/m^3$ increase in average $PM_{2.5}$ exposure from 2004 through 2013 is associated with a 0.46 pp increase in the probability of receiving a dementia diagnosis by the end of 2013. About 80% of this association persists in column (2) after we add a flexible function of baseline neighborhood $PM_{2.5}$ concentrations from 2001-2003. The $PM_{2.5}$ coefficient declines slightly further to 0.33 pp in column (3) when we add all of the observed measures of baseline health, demographics and socioeconomic status. Narrowing our focus to variation in exposure among people who lived in the same Core Business Statistical Area in 2013 further reduces the coefficient to 0.26 pp in column (4). These 982 CBSA dummy variables alone absorb 83.5% of the variation in individuals' decadal $PM_{2.5}$ exposures.

The national sample used to estimate the models in columns (1)-(4) includes people who lived in counties that lacked air pollution monitors at the time nonattainment designations were made. Spatially interpolating their pollution exposures relies exclusively on information from other coun-

[26] Medicare's three GPCIs track spatiotemporal variation in the wages of professional workers, malpractice insurance costs, and practice cost and expense and CMS uses this information to adjust levels at which Medicare reimburses doctors for specific procedures which, in turn, could affect the rates at which local doctors treat Medicare patients.

ties, which may increase measurement error due to the greater distance between people's residences and the monitors. This could pose a threat 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. Repeating estimation of the model in column (4) using the smaller sample of 1.2 million people reduces our OLS estimate for the $PM_{2.5}$ coefficient in column (5) to 0.21 pp. To assess the sensitivity of these results to our functional form assumptions, we estimate the probit analogue to the model in column (5). The average marginal effect from the probit model is virtually identical to that from the linear probability model (0.21 pp, p=0.043).

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

	(1)	(2)	(3)	(4)	(5)	(6)
Decadal $PM_{2.5}$ (1 μg/m^3)	0.459***	0.372***	0.333***	0.255***	0.210**	1.288***
	(0.02)	(0.05)	(0.05)	(0.09)	(0.11)	(0.46)
4th order polynomial in 2001-2003 exposure		x	x	x	x	x
individual & neighborhood covariates			x	x	x	x
CBSA dummies				x	x	x
monitored county sample					x	x
2SLS: county attainment status IV						x
Kleibergen-Paap rk Wald F-statistic						658
Number of individuals	1,851,175	1,851,175	1,851,175	1,851,175	1,256,440	1,256,440
Share with dementia in 2013	18.9	18.9	18.9	18.9	19.2	19.2

Note: The dependent variable equals 100 if an individual was diagnosed with dementia prior to the end of 2013 and 0 otherwise. Pollution exposures are based on 10-year averages corresponding to the individual's residential address history. The estimation sample is limited to individuals alive in 2013 for whom we continuously observe pollution exposure at their home address between 2001 and 2013. Col (1) is a univariate OLS regression. Col (2) adds a 4th order polynomial function of baseline residential exposure from 2001-2003. Col (3) adds covariates for baseline health in 2004, individual demographics and mean demographics for the person's 2004 Census block group. Col (4) adds dummies for the 982 Core Business Statistical Areas where people lived in 2013. Col (5) limits the sample to people who were living in counties with air pollution monitors that were designated as "attainment" or "nonattainment" by the EPA in 2005. Col (6) reports results from 2SLS estimation of the model in Col (5), instrumenting for decadal $PM_{2.5}$ exposure using an indicator for county non-attainment status interacted with the 4th order polynomial function of baseline exposure. Coefficients on all other covariates in the first and second stage models are reported in Appendix Table A2. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered by initial Census block group.

Column (6) reports our main 2SLS result. It modifies the OLS specification from column (5) by using county nonattainment to instrument for decadal exposure. The second-stage coefficient on $PM_{2.5}$ is about six times larger than the corresponding OLS estimate. It implies that a 1 μg/m^3 increase in average $PM_{2.5}$ exposure from 2004 through 2013 increased the probability of receiving a dementia diagnosis by the end of 2013 by 1.29 pp, a 6.7% increase relative to the dementia rate

in our sample.[27] Coefficients on the remaining covariates are reported in Appendix Table A2.[28]

ii. First Stage Results

Our 2SLS estimate is identified by the conditional variation in individuals' decadal $PM_{2.5}$ exposures that arises from county nonattainment designations. Figure VI illustrates this variation graphically. The solid line is constructed by using our first-stage coefficients on the excluded instruments to predict how county nonattainment designations affected decadal exposure conditional on pre-decadal exposure, CBSA dummies, and the other covariates.[29] The dotted lines denote 95% confidence bands on our predictions. For instance, consider people who sorted themselves into ZIP+4 locations that had average $PM_{2.5}$ concentrations of 13 $\mu g/m^3$ from 2001 to 2003. Within this group, average exposure from 2004 to 2013 is predicted to be approximately 0.3 $\mu g/m^3$ lower for the subset living in counties that received nonattainment designations. This is close to our estimate for the median partial effect among all people living in nonattainment counties (-0.27 $\mu g/m^3$).

The partial effect function for $PM_{2.5}$ is negative below the attainment threshold, as expected. Its monotonic shape indicates that local regulatory actions led to relatively larger $PM_{2.5}$ reductions in nonattainment county neighborhoods with lower baseline concentrations. This makes sense: as local concentrations rise within an attainment county so does the likelihood of future regulation. The prospect of future regulation provides an incentive for local regulators to take preemptive actions, reducing the magnitude of differences in future $PM_{2.5}$ reductions between attainment and nonattainment counties. Relatively polluted neighborhoods within attainment counties may also be more likely to benefit from spillover effects of regulation in nearby nonattainment counties. The residual variation in $PM_{2.5}$ shown in Figure VI arises from two mechanisms. First, some CBSAs include attainment and nonattainment counties with overlapping distributions of baseline

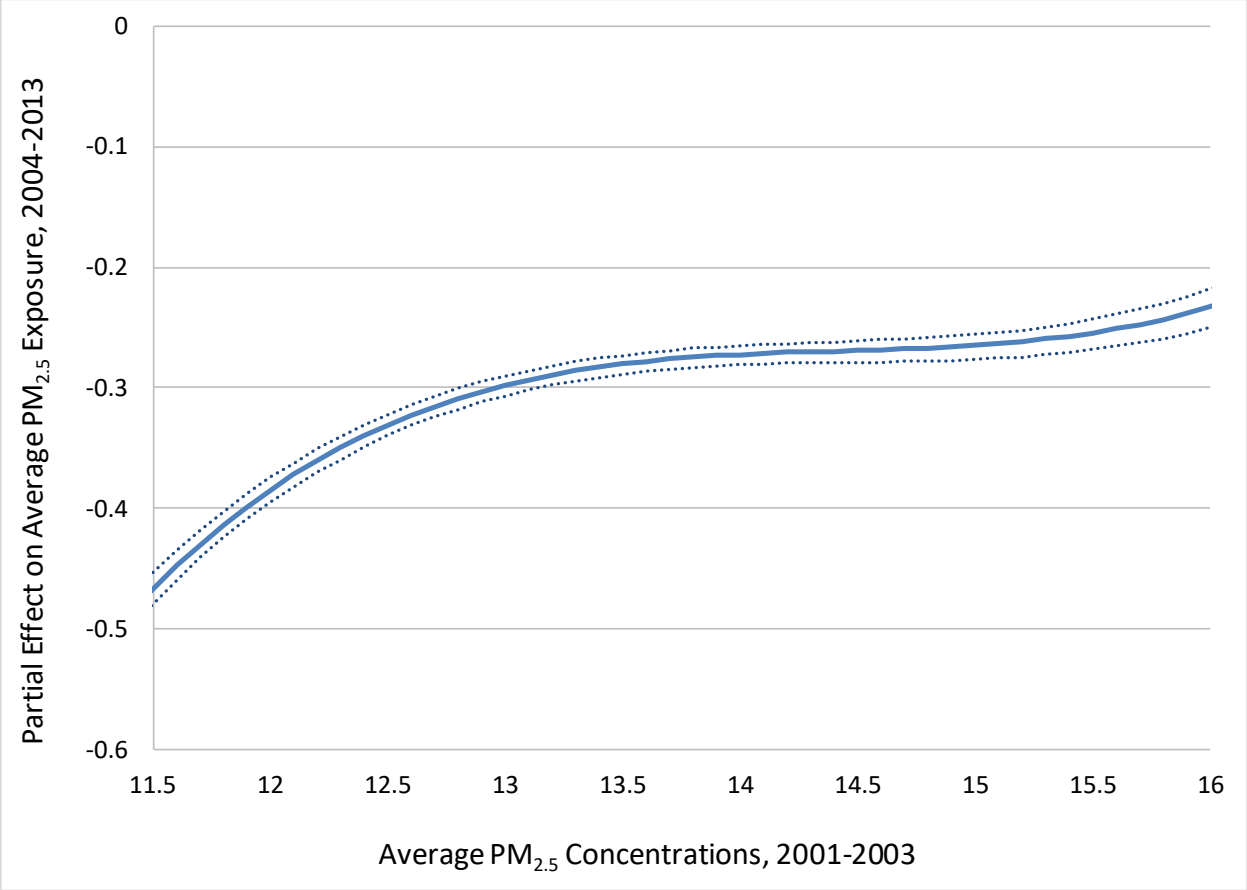
[27] The p-value on Hansen's J-statistic is 0.454, so we fail to reject the hypothesis that our instruments are valid and the model is correctly specified. Fitted probabilities of receiving a dementia diagnosis from the IV model lie between zero and one for 95.25% of individuals. They are less than zero for 4.74% of individuals and greater than one for 3 individuals. In comparison, fitted probabilities for the OLS model in column 5 are less than zero for 4.56% of individuals and greater than one for 3 individuals.

[28] For example, Table A2 shows that after we condition on the age and gender effects already seen in Figure II, as well as on the other controls and the baseline and instrumented $PM_{2.5}$ measures, diagnosis rates tend to be higher for African-Americans (+3.4%) and Hispanics (+3.1%) relative to Asians (+1.9%) and Whites (+1.7%), with "other race" as the omitted category. Diagnosis rates also decline by about 1.3% 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% increase in the fraction of block group residents with graduate degrees (relative to less than 8^{th} grade education) is associated with a 0.4% reduction in the dementia diagnosis probability. Section V provides a more detailed comparison between the estimated effect of $PM_{2.5}$ and estimated effects of age and pre-existing medical conditions.

[29] The Kleibergen-Paap rk Wald F statistic is 657.6, allowing us to strongly reject the hypothesis that our instruments are weak. In comparison, the critical value for the Stock-Yogo test of 5% maximal IV bias is 18.37.

$PM_{2.5}$ concentrations.[30] Nonattainment designations within these CBSAs create variation in decadal $PM_{2.5}$ exposures among the subset who had the same residential ZIP+4 codes from 2004 through 2013 and had pre-sorted themselves into neighborhoods that had the same baseline $PM_{2.5}$ concentrations. Intuitively, we can compare people in relatively clean areas of nonattainment counties with those in relatively dirty areas of attainment counties in the same CBSA. Second, the subset who changed their residential ZIP+4 codes at some point from 2004 to 2013 experienced variation in exposure from their migration paths.

FIGURE VI: PARTIAL EFFECT OF COUNTY NONATTAINMENT DESIGNATION ON $PM_{2.5}$ EXPOSURE



Note: The figure shows the average effect of the 2005 nonattainment designation on the average conditional change in decadal PM2.5 concentration levels. These estimates are derived from the first stage of the 2SLS model as shown in Appendix Table A2.

[30] Appendix Figure A4 illustrates the within-CBSA variation in county nonattainment status conditional on baseline $PM_{2.5}$ concentrations, using New York and Chicago as examples.

We interpret the partial effect function in Figure VI as a lower bound on the impact of the EPA's regulation on people who started the decade in nonattainment counties. Because the function conditions on baseline $PM_{2.5}$ concentrations, CBSA dummies, and other covariates, the partial effects exclude any reductions in exposure that can be explained by a linear function of those conditioning variables. This is important because the regulation was designed to lead local air quality managers to reduce pollution more in more polluted areas. We can approximate the regulation's full effect by a differences-in-differences regression of the change in average $PM_{2.5}$ exposure on county attainment status. We find that average $PM_{2.5}$ exposure declined by 1.24 $\mu g/m^3$ more for people in nonattainment counties, above and beyond a reduction of 1.80 $\mu g/m^3$ shared between attainment and nonattainment counties.

B. Robustness

Table II summarizes results from alternative 2SLS specifications. Column (1) repeats our main result from Table I for comparison, columns (2)-(5) summarize sensitivity to changing features of the research design, and columns (6)-(9) test hypotheses about the mechanisms underlying our findings. In column (2) we extend the nonattainment instrument to utilize within-county variation in monitor readings, similar to Bento, Freedman and Lang (2015). Specifically, we stratify the county nonattainment indicator according to whether the air quality monitor closest to a person's residence violated the federal regulatory standard. This generates three indicators: (i) nonattainment county with nearest monitor exceeding the threshold, (ii) nonattainment county without nearest monitor exceeding the threshold, and (iii) attainment county with nearest monitor exceeding the threshold. As in our main specification, each indicator is interacted with the polynomial function of baseline exposure. This strategy is based on Auffhammer, Bento and Lowe (2009) who found that county regulators responded to nonattainment designations for PM_{10} by strategically targeting areas close to nonattainment monitors for more aggressive action, yielding larger reductions in particulates near nonattainment monitors relative to attainment areas in the same counties. An advantage of extending the set of instruments to include information on monitor attainment is that it allows us to utilize identifying variation in $PM_{2.5}$ exposure that comes from within-county conditional variation in long-term exposure among those who never moved. Column (2) shows that doing so yields a 1.03 pp (5.4% of the mean) estimate for the effect of a 1 $\mu g/m^3$ increase in

$PM_{2.5}$ on the probability of a dementia diagnosis by the end of the decade.[31]

TABLE II—ROBUSTNESS CHECKS ON THE EFFECT OF $PM_{2.5}$ ON DEMENTIA

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Decadal $PM_{2.5}$ (1 μg/m^3)	1.288***	1.027**	1.290***	1.016**	1.362***	1.901***	1.357**	0.14	1.148***
	(0.46)	(0.41)	(0.41)	(0.43)	(0.46)	(0.39)	(0.62)	(0.36)	(0.34)
unbalanced monitor panel				x					
spline function of baseline $PM_{2.5}$					x				
other pollutants included							x		
Dementia with Alzheimer's diagnosis									x
Dementia without Alzheimer's diagnosis								x	
exclude post-dementia movers						x			
CBSA dummies	x	x		x	x	x		x	x
county dummies			x				x		
2005 attainment instruments									
county	x			x	x	x		x	x
county x monitor		x	x				x		
Kleibergen-Paap rk Wald F-statistic	658	455	511	367	142	653	74	658	658
Number of individuals	1,256,440	1,256,440	1,256,440	1,256,440	1,256,440	1,195,022	1,256,440	1,256,440	1,256,440
Share with dementia in 2013	19.2	19.2	19.2	19.2	19.2	15.0	19.2	19.2	19.2

Note: For reference, Col (1) repeats the results from our main specification (Table 1, Col 6) 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. Col (3) uses the same instruments as Col (2) but replaces the CBSA dummies with dummy variables for counties. Col (4) replaces our preferred measure of decadal pollution exposure (based on a balanced panel of continuously operating monitors) with data from an unbalanced panel of all monitors in operation each year. 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) tests for reverse causality by excluding everyone who moved after receiving a dementia diagnosis. Col (7) adds other criteria pollutants: PM_{10}, ozone, carbon monoxide, nitrogen dioxide, and sulfur dioxide. They are all treated as endogenous. Their coefficients are reported in Appendix Table A3. Col (8) defines the dependent variable as dementia cases without Alzheimer's disease while Col (9) defines it as Alzheimer's disease specifically. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered by initial Census block group.

Another advantage of the county-by-monitor instruments is that they allow us to tighten our spatial controls for omitted variables. We can replace the CBSA dummies with dummy variables for counties while retaining variation in long-term exposure among those who never moved. Column (3) shows that adding county dummies produces an estimate that is virtually identical to column (1).

Column (4) replaces our "balanced monitor panel" measure of $PM_{2.5}$ exposure with a measure constructed from an unbalanced panel of all monitors in operation each year (between 787 and

[31] Appendix Figure A5 shows that after we condition on baseline exposure, CBSA dummies, health and demographics we find some exposure patterns consistent with strategic regulatory targeting for $PM_{2.5}$. Our first-stage 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 μg/m^3. Moreover, we find that nonattainment designations produced the smallest declines in $PM_{2.5}$ for people in attainment counties living near nonattainment monitors. This pattern could result from strategic regulatory actions diverting pollution from areas near nonattainment monitors to areas in adjacent attainment counties.

1,106 monitors per year). Using the unbalanced panel may improve efficiency by using all available ground-level information on pollutant concentrations, but it also has potential to introduce measurement error (Muller and Rudd 2017, Grainger, Schreiber and Chang 2018). Consistent with measurement error, we find that using the unbalanced panel reduces the instrument's power to explain decadal $PM_{2.5}$ exposures and yields a smaller second-stage estimate. These reductions are small, however, revealing that our conclusions are not driven by which monitors are used to measure pollution exposure.

Column (5) replaces the fourth order polynomial function of baseline (2001-2003) residential $PM_{2.5}$ concentrations with a more flexible "spline" function. We first partition neighborhoods into 72 bins by baseline concentrations, using a bin width of 0.33 $\mu g/m^3$, and then we add an indicator variable for each bin. This produces a slightly larger $PM_{2.5}$ coefficient (1.36 pp) with virtually no change in statistical precision.

Our findings in columns (1)-(5) could be confounded by reverse causality if a dementia diagnosis causes patients to move to more polluted areas (e.g., if assisted living facilities tend to be located in more polluted urban areas). We test this hypothesis by repeating estimation of the model in (1) after dropping everyone who moved after being diagnosed with dementia. Column (6) shows that dropping movers with dementia increases our estimate to 1.90 pp. This result reflects the fact that movers with dementia tend to move to cleaner areas (shown in Appendix Figure A6) and provides evidence against reverse causality due to residential sorting on health.

Our $PM_{2.5}$ coefficients could capture partial effects of other federally regulated air pollutants that are co-generated as a byproduct of power generation, transportation and manufacturing activities that are constrained by EPA regulation. To test this hypothesis, we extend the model in (1) to include measures of decadal exposure to PM_{10}, ozone, sulfur dioxide, nitrogen dioxide, and carbon monoxide. We treat all of these air pollutants as endogenous, using the instruments from column (2). The resulting $PM_{2.5}$ coefficient in column (7) is slightly larger than column (1) whereas the other pollutants' coefficients vary in sign and are all statistically indistinguishable from zero at the 10% level (Appendix Table A3). Hence, we cannot reject the hypothesis that dementia rates are unaffected by elevated long-term exposures to particulates larger than 2.5 microns that are captured by measures of PM_{10}, ozone, sulfur dioxide, nitrogen dioxide, and carbon monoxide.

Finally, we repeat estimation of the model in (1) after stratifying the dependent variable to

measure dementia cases with and without an associated diagnosis of Alzheimer's disease specifically.[32] Our point estimates in columns (8) and (9) suggest that Alzheimer's disease accounts for about 90% of the all-cause 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 disease and other forms of dementia without an autopsy or extensive brain imaging. Therefore, as a further test of which types of dementia drive our results, we repeat estimation of the model in (1) after adding a dummy for whether the individual had a stroke by the end of 2013. Strokes cause vascular dementia, the second most common form of dementia behind Alzheimer's disease, and may be caused by short-term spikes in air pollution. Hence, the stroke variable absorbs any effects of $PM_{2.5}$ on dementia that occur due to stroke. Our results suggest that the probability of being diagnosed with dementia is 18.74 pp higher for those who had a stroke (95% CI = [18.50, 18.97]). However, controlling for this has virtually no effect on the $PM_{2.5}$ coefficient, 1.299 ($p<0.01$). This reinforces the conclusion that long-term exposure to $PM_{2.5}$ increases the risk of Alzheimer's disease.

C. Results from the Model with Aggregated Data

Table III summarizes the results from a second set of robustness checks in which we aggregate Medicare records to the county level, using a county nonattainment indicator as the instrument for the decadal change in $PM_{2.5}$ exposure in the spirit of Chay and Greenstone (2005). The covariates parallel our micro-data specification. They include dummy variables for Census divisions, changes in county Medicare populations in terms of the integer age distribution, gender, race and health, changes in access to medical care including Medicare payment levels, changes in the demographic composition of the average Medicare beneficiary's Census block group, beginning-of-decade medical expenditures, and beginning-of-decade fractions of people diagnosed with each morbidity risk for dementia. We calculate means for these variables in 2004 and 2013 along with annual average $PM_{2.5}$ exposures using all of the individuals we observe living in each county in each year. This is an unbalanced panel of 6.9 million people, with 3.6 to 4.3 million people per year. All specifications use robust standard errors.

[32] 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".

TABLE III—FIRST DIFFERENCE ESTIMATES OF THE EFFECT OF $PM_{2.5}$ ON DEMENTIA

	(1)	(2)	(3)	(4)	(5)
$\Delta PM_{2.5}$ (1 ug/m^3)	0.417***	0.558**	1.099*	1.017*	0.351**
	(0.142)	(0.265)	(0.626)	(0.545)	(0.152)
F statistic on attainment IV	43	34	11	16	36
large county sample		x			
matching sample			x		
threshold sample				x	
full sample (including "unclassifiable" counties)					x
mean dementia rate (2013)	11.3	11.3	11.5	11.2	11.2
number of individuals (2013)	2,978,573	2,950,862	2,229,286	1,486,396	4,303,352
number of counties	646	553	378	343	3,061

Note: The dependent variable is the change from 2004 to 2013 in the share of living people in a county diagnosed with dementia. This difference is regressed on $\Delta PM_{2.5}$, defined by the difference between average exposure from 2004 to 2013 and average exposure from 2001 to 2003 calculated for all the people observed living in each county in each year. The instrument for $\Delta PM_{2.5}$ is an indicator for nonattainment designation in 2005. All specifications include dummy variables for Census divisions and covariates describing changes in county Medicare populations in terms of the integer age distribution, gender, race and health, changes in access to medical care, changes in the demographic composition of the average Medicare beneficiary's Census block group, beginning-of-decade medical expenditures, and beginning-of-decade fractions of people diagnosed with each morbidity risk for dementia. Col (1) estimates the model by weighted least squares for the 646 "attainment" and "nonattainment" counties with air quality monitors, using county populations as weights. Col (2) estimates the model by OLS for the subset of counties for which we observe at least 500 people each year. Col (3) repeats WLS estimation for 378 counties that had annual average $PM_{2.5}$ concentrations between 12 and 16 $\mu g/m^3$ during the baseline period from 2001-2003. Col (4) repeats WLS estimation for a sample of attainment counties in a window below the regulatory threshold (11.05 to 15.05 $\mu g/m^3$) and nonattainment counties in a window above the threshold (15.05 to 19.05 $\mu g/m^3$). Col (5) repeats WLS estimation using data for all counties, including those without air pollution monitors. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels based on robust standard errors.

Column (1) reports results for 646 counties that had air quality monitors in place throughout the 2001-2003 period EPA used as the basis for making nonattainment designations.[33] These counties tend to be larger as the EPA tends to place monitors in more populous places. In 2013, they contained 69% of the 4.3 million people in our data. We estimate the model by weighted least squares (WLS), weighting each county by the number of people in the sample to make the coefficients nationally representative.[34] The 2SLS estimate suggests that a 1 $\mu g/m^3$ decrease in average exposure to $PM_{2.5}$ between 2004 and 2013 caused county-level dementia rates to decrease by about 0.42 pp, a 3.7% decrease relative to the population-weighted county dementia rate in 2013. Note that these statistics are smaller than the corresponding figures from our individual-level model using microdata (1.3 pp or 6.7% decrease relative to the mean). This occurs because aggregating to the county level weakens the relationship between the cognitive health of a county's current

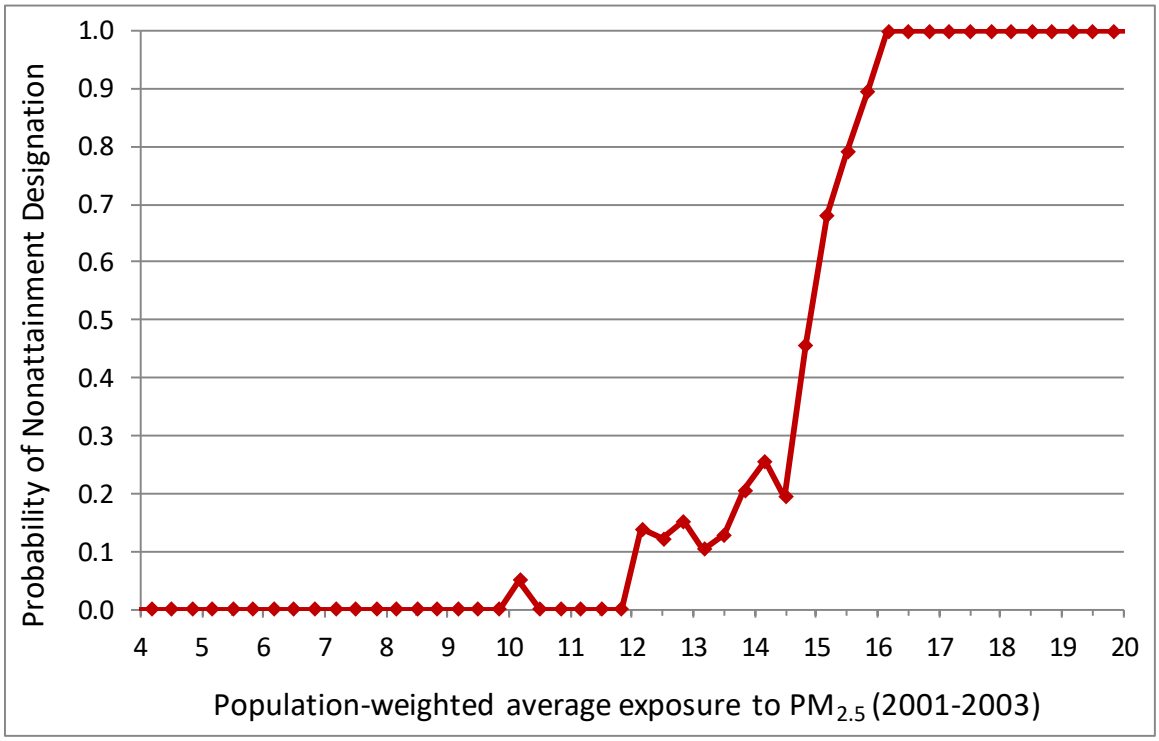
[33] The micro data models in columns 5 and 6 of Table 1 and throughout Table II are estimated using the individuals who lived in these 646 counties at the time nonattainment designations were made.

[34] We weight by the minimum of the county sample sizes in 2004 and 2013. Weighting by the county sample size also improves statistical precision by reducing the weight placed on small, rural counties for which changes in dementia rates are less precisely estimated.

population and past changes in its average resident's $PM_{2.5}$ exposure due to population turnover from migration, death, and aging into Medicare. While this aggregation bias does not change our qualitative conclusions, avoiding it by using micro data increases our point estimates substantially.

Column (2) repeats the estimation using an unweighted regression that ignores differences in county populations. As an alternative to weighting by population, we address measurement error in dementia rates in small counties by limiting the sample to the 553 monitored counties that had at least 500 people in 2013. This increases the point estimate to 0.56 pp.

FIGURE VII: 2005 COUNTY ATTAINMENT STATUS BY BASELINE $PM_{2.5}$ EXPOSURE



Note: The figure displays the fraction of the 646 counties with air quality monitors that EPA designated as nonattainment in 2005 within 0.333 microgram per cubic meter bins for a county's baseline exposure. Baseline county exposures are calculated using our inverse distance-squared measure for the people we observe living in each county between 2001 and 2003. EPA used this 3-year interval to define the 2005 nonattainment status. The points denote bin midpoints.

Next, we re-estimate the WLS model for 378 counties that had average exposures between 12 and 16 $\mu g/m^3$ during 2001-2003. Figure VII shows that there is variation in county attainment status conditional on baseline exposure throughout this range. To construct the figure, we group the 646 counties from column (1) into 0.33 $\mu g/m^3$ bins based on average exposure in 2001-2003. The vertical axis shows the fraction of counties in each bin that the EPA designated as nonattainment. Only one county with concentrations below 12 $\mu g/m^3$ was designated as nonattainment;

every county with concentrations above 16 $\mu g/m^3$ was designated as nonattainment; and as a county's average concentrations increased from 12 to 16 $\mu g/m^3$, so too did its probability of receiving a nonattainment designation. Most of the nonattainment counties with baseline concentrations between 12 and 16 $\mu g/m^3$ had average exposures below the regulatory threshold but were classified as nonattainment because (i) they contained "hot spots" that violated the standard or (ii) they were believed by EPA to contribute to violations in neighboring counties. Focusing on this subset allows us to relax the exogeneity assumption on the nonattainment indicator outside of a narrow range of baseline exposure. Column (3) shows that this increases our point estimate to just over 1 pp.

Column (4) shows that we obtain a similar estimate when we implement a version of the model in column (1) in which we limit the sample to attainment counties in a narrow window below the regulatory threshold (11.05 to 15.05 $\mu g/m^3$) and nonattainment counties in a narrow window above the threshold (15.05 to 19.05 $\mu g/m^3$). The coefficients in columns (3) and (4) remain statistically different from zero at the 10% level despite the small sample sizes and extensive covariates.

Finally, column (5) shows that our main result from column (1) is also robust to adding data describing all remaining counties, including those defined by the EPA to be unclassifiable.[35] In summary, as we compare column (1) to columns (2)-(5), reducing the sample size and changing the sample geography causes the point estimates to fluctuate amid wider confidence intervals. Yet the coefficients are uniformly consistent with our micro-data models in that they all indicate that higher exposures to $PM_{2.5}$ from 2004 to 2013 caused higher dementia rates.

D. Placebo Tests

Our research design mitigates potential biases from residential sorting, spatial heterogeneity in health care, omitted variables, and measurement error in pollution exposure. Anticipatory behavior poses another potential threat to identification. For instance, if people at a lower risk of receiving a future dementia diagnosis due to latent factors (e.g., genetics, childhood pollution exposure) not fully controlled by our measures of baseline health moved to neighborhoods prior to our study period that they correctly anticipated would experience relatively large future improvements in amenities due to EPA regulation then our IV estimates could be biased upward. If this mechanism

[35] There are 3,142 counties and county equivalents in the United States. The 81 that are excluded are among the least populous rural counties. They are missing because Census block group variables or Medicare Advantage enrollment information are suppressed to avoid identifying individuals or because our sample does not include multiple people living in the county every year.

were confounding our results, however, then we would expect to see relatively large effects for pollutants that are readily observable to people because they are relatively large in size (PM_{10}), they contribute to urban smog (ozone), and they are generated by point sources such as freeways (carbon monoxide) and factories and coal-fired power plants (nitrogen dioxide, sulfur dioxide) that may be viewed as negative amenities. The fact that we do not find such effects in Appendix Table A3 provides evidence against confounding.

As an additional test of anticipatory behavior and other unspecified threats to identification, we re-estimate the micro-level and county-level 2SLS models shown in the first columns of Tables I and II for six chronic medical conditions that are not known to be caused by air pollution, to the best of our knowledge.[36] These include glaucoma, fibromyalgia, breast cancer, prostate cancer, viral hepatitis, and peripheral vascular disease. We selected these placebo conditions because they share similarities with dementia. 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; viral hepatitis is correlated with measures of socioeconomic status; and peripheral vascular disease is associated with reduced blood circulation.

TABLE IV: EFFECTS OF $PM_{2.5}$ ON PLACEBO MEASURES OF MORBIDITY

	Dementia	Glaucoma	Fibro-myalgia	Breast Cancer	Prostate Cancer	Viral Hepatitis	Peripheral Vascular Disease
$PM_{2.5}$ (1 μg/m^3) [micro data]	1.288***	-0.851*	-0.676	0.047	-0.367	0.115	0.52
	(0.46)	(0.47)	(0.50)	(0.23)	(0.24)	(0.08)	(0.53)
$\Delta PM_{2.5}$ (1 ug/m^3) [county data]	0.417***	-0.18	0.243	-0.046	-0.028	-0.003	0.188
	(0.142)	(0.15)	(0.19)	(0.04)	(0.05)	(0.03)	(0.20)
Prevalence among 75-year olds							
2004	7.9	14.0	8.5	3.8	4.7	0.7	12.4
2013	9.1	17.1	15.2	4.4	5.1	1.2	16.7

Note: The first four rows report point estimates and standard errors from repeating our main micro-level and county-level specifications (shown in the first column of tables II and III) except the outcome is one of six placebo morbidities. The last two rows report the fraction of people in our data who were diagnosed with each morbidity by 2004 and 2013. The first column repeats our findings for dementia for convenience. Asterisks indicate statistical significance at the 10%, 5%, and 1% levels based on robust standard errors clustered at the Census block group level. The text and footnotes to Tables II and III provide additional details about model specification.

[36] This criterion rules out cardiopulmonary conditions along with many other common medical conditions that one might not intuitively associate with air pollution. For example, the medical literature has linked osteoporosis to air pollution via cadmium contained in $PM_{2.5}$.

Finding large, positive, and statistically significant effects of $PM_{2.5}$ on these placebo morbidities would signal that our research design may be compromised. Table IV shows that this is not the case. We fail to reject the null hypothesis of no effect at the 10% level in 2SLS specifications for all but one of the twelve placebo models. This rejection rate is roughly consistent with a true zero effect, and the one statistically significant result (for glaucoma in the micro-level model) has the "wrong" sign. Most of the point estimates are small relative to our findings for dementia. Thus, these placebo tests provide support for our research design.

V. Interpretation

Our main point estimate (Table II, column (1)) suggests that a 1 $\mu g/m^3$ increase in 10-year average $PM_{2.5}$ exposure from 2004 to 2013 increased the probability of receiving a dementia diagnosis by 1.3 pp. This is equivalent to a 6.7% 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 viewed as a moderate change in exposure, albeit a change smaller than the 1.24 $\mu g/m^3$ average reduction due to a county being designated as nonattainment in 2005.

Table V compares our main $PM_{2.5}$ result to other risk factors for dementia in terms of percentage-point changes in the probability of receiving a diagnosis by the end of the decade. For instance, our 1.3 pp estimate for the effect of a 1 $\mu g/m^3$ increase in decadal $PM_{2.5}$ is equivalent to the increase in risk associated with a female aging from 74 to 77 and about one quarter of the increase in risk associated with a female aging from 74 to 80 (5.7 pp). Likewise, our $PM_{2.5}$ estimate is somewhat smaller than the increase in 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 (1.8 pp). We estimate much larger increases in risk associated with pre-existing diagnoses of ischemic heart disease only (3.0 pp), diabetes only (4.2 pp), congestive heart failure only (5.1 pp), and a stroke only (8.8 pp). Someone diagnosed with all five conditions by 2004 had a 21.8 pp higher probability of being diagnosed with dementia by the end of 2013.

TABLE V. RISK OF DEMENTIA FROM $PM_{2.5}$ RELATIVE TO RISKS ASSOCIATED WITH PRE-EXISTING CHRONIC ILLNESSES

Risk Factor	Percentage point increase in dementia diagnosis probability	95% confidence interval	
decadal $PM_{2.5}$ (1 μg/m^3)	1.3	0.4	2.2
Aging from 74 to 77 (women)	1.3	1.0	1.6
hypertension	1.8	1.6	2.0
ischemic heart disease	3.0	2.7	3.4
diabetes	4.2	3.7	4.7
congestive heart failure	5.1	3.9	6.3
aging from 74 to 80 (women)	5.7	5.2	6.1
stroke	8.8	7.6	9.9
All five chronic conditions	21.8	20.7	23.0

Note: The table reports point estimates and 95% confidence intervals for dementia risk factors based on our main micro-level specification from the last column of Table I. Appendix Table A2 reports the full set of results.

VI. Policy Implications

A. Dementia Cases Avoided from the EPA's 1997 $PM_{2.5}$ Regulation

The EPA's benefit-cost analysis of the Clean Air Act 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.[37] We take a first step toward filling this knowledge gap by using our estimates to approximate the value of dementia cases avoided 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 μg/m^3 from Section IV.A.ii.[38] Multiplying this reduction by our main estimate for the effect of a 1 μg/m^3 increase in decadal exposure on the probability of a dementia diagnosis (1.29 pp)

[37] 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. In their Table 3, Landen et al. report a hazard mortality rate ratio of 1.16 for all-cause mortality from a 10 μg/m^3 increase in $PM_{2.5}$ over their entire follow-up period, compared to 1.28 for cardiovascular deaths, 1.08 for respiratory deaths, 1.27 for lung cancer deaths, and 1.02 for all other causes, from which they conclude that "There was no association (p=0.71) with other causes of death". Similarly, Pope et al. report a mortality risk ratio of 1.01 (CI = 0.95, 1.06) for deaths from causes other than cardiopulmonary and lung cancer in their Table 2, compared to 1.06 (1.02, 1.11) for all-cause mortality, 1.09 (1.03, 1.16) for cardiopulmonary, and 1.14 (1.04, 1.23) for lung cancer.

[38] We obtain a similar estimate of -1.05 μg/m^3 from the first-stage of the county level model in Table III, column (1)..

implies that the regulation reduced the dementia rate by 1.6 pp. Multiplying this by the Census Bureau's estimate for the size of the 75-and-over population in nonattainment counties in 2013 (8.7 million) implies that the $PM_{2.5}$ regulation reduced the number of dementia cases by approximately 140,000, with a range from 42,000 to 235,000 cases based on our 95% confidence interval for the $PM_{2.5}$ coefficient.

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), together with the Medicare data for estimates of dementia's effects on life expectancy and prior estimates for dementia's impacts on quality of life. Appendix B describes our calculations in detail. We first use our data to calculate two key 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 life years lost per dementia case, with a range from 5.5 to 6.4 years reflecting Kasai and Meguro's (2013) adjustments for upper and lower bounds on the severity of symptoms during the survival period. 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 in 2007, equating to $365,000 in 2018 dollars. Previously, Hirth et al. (2000) found a wide range of estimates, with the central estimates between $114,000 and $196,000 in 2018 dollars. 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 implies a value per statistical case of dementia avoided of approximately $1.2 million, whereas the lower bound implies a value close to $0.6 million.

Multiplying our central estimates for the value of a life year, quality adjusted life years lost per case, and the number of cases avoided implies that the $PM_{2.5}$ regulation yielded benefits of $163 billion for the cohort of people age 75 and above in nonattainment counties. Figure VIII illustrates how this estimate changes if we instead use the endpoints of our ranges for each statistic. Under every scenario the benefits exceed $48 billion. As a further sensitivity check, we repeat our calculations at range midpoints after replacing our difference-in-difference estimate for the full effect of nonattainment designations on $PM_{2.5}$ exposure (-1.24 $\mu g/m^3$) with a lower bound based on our

central estimate for the partial effect of nonattainment designations from Figure VI (-0.27 $\mu g/m^3$).[39] This yields a benefit estimate of $36 billion.

FIGURE VIII: ESTIMATED BENEFITS OF THE 1997 $PM_{2.5}$ STANDARD DUE TO REDUCED DEMENTIA

Note: The sensitivity analysis varies a single variable across the denoted range while holding the other two constant at their midpoints.

We interpret these estimates as likely lower bounds on the benefits of the EPA's 1997 $PM_{2.5}$ standard because we exclude several other types of benefits. We exclude any benefits that accrued to people who started the decade in attainment counties, for example due to spatial spillover of $PM_{2.5}$ reductions or anticipatory responses by regulators. We also exclude any benefits for people who were under age 65 at the start of the decade, benefits that accrued to people who died during the decade, and any health benefits, such as lower mortality from cardiovascular disease and lung cancer, other than reduced dementia rates for people who were over 65 and survived to the end of the decade. We also exclude the taxpayer savings from lower Medicare expenditures on dementia and the value of time for unpaid caregivers of dementia patients. Last, we exclude the benefits of maintaining the cognitive skills that older adults need to successfully engage with markets. While we leave a full accounting of these benefits to future research, we leverage Medicare administrative records to provide the first evidence on how dementia affects an important financial decision: choice of a prescription drug insurance plan (PDP) through the markets created under Medicare Part D.

[39]In principle, our difference-in-difference estimate for the full effect of nonattainment designations on pollution levels could be understated or overstated. The latter could occur if the great recession or other macroeconomic forces were correlated with county nonattainment designations and pollution levels. Such effects are likely to be purged from our partial effect estimate by the CBSA dummies. They could be understated if nonattainment designations led to indirect air quality improvements in attainment counties , compressing the difference in pollution changes between attainment and nonattainment counties. For instance, nonattainment designations could have reduced emission spillover from nonattainment counties to attainment counties.

B. Dementia's Effects on Financial Decision Making

The Medicare PDP markets provide an ideal setting to assess the extent to which dementia may affect individuals' financial decisions. In 2006, Medicare established regional markets in which participants may choose from competing private insurers selling federally subsidized plans that differ in cost, quality and risk protection. The default for new Medicare beneficiaries is to be uninsured, but from 2006 to 2010 between 64% and 72% of individuals choose to enroll in a PDP.[40] After an individual chooses a plan, they are automatically re-enrolled in the same plan for each subsequent year unless they switch plans during the annual open enrollment period. On average, each individual chooses from approximately 50 different plans and spends approximately 6% of their annual household income on premiums and out-of-pocket drug costs. Importantly, the cost of these drugs varies by over $1,000 on average across each individual's available plans.[41] Hence, PDP enrollees make a financially important decision under uncertainty in the presence of a default assignment rule. This makes PDP enrollment similar to other complex financial decisions made by older adults, such as management of retirement savings, enrollment in a general health insurance plan, and estate planning.

A large empirical literature has used the Medicare PDP markets to judge older adults' abilities to make complex financial decisions (Keane and Thorp 2016 provide a review). The literature starts from the observation that enrollees can reduce their expenditures on prescribed drugs by comparing plan formularies and switching plans when their expected drug needs change.[42] With this in mind, the literature has used three types of metrics to assess decision making outcomes:

1. **Potential savings**, defined as the amount of money an individual spent on drugs in their chosen plan minus the cost of those same drugs under the cheapest plan available (e.g., Heiss, McFadden and Winter 2010, Ketcham et al. 2012).[43]

[40] Some of those who chose not enroll in a PDP obtained prescription drug coverage from a Medicare Advantage managed health care plan and the rest obtained insurance from an employer or other sources or chose to forego insurance for prescription drugs.
[41] Among the participants in these markets not receiving low-income subsidies, surveys indicate that 27% receive help making their enrollment decisions (most commonly spouses and daughters), and another 11% rely on someone else altogether (Ketcham, Kuminoff and Powers 2016). As in previous evaluations of consumer decision making in Part D, we exclude people who receive low income subsidies because they are auto-enrolled into low cost plans and because their cost sharing structure mitigates many of the differences across plans' financial characteristics.
[42] There are several plan finder websites that enrollees can use to compare plans based on the cost of purchasing a particular bundle of drugs, as well as a 1-800 number that enrollees can call in order to have Medicare operators help them compare available plans. These plan finder tools are described in detail in Kling et al. (2012) and Ketcham, Kuminoff and Powers (2016).
[43] We follow prior studies in assuming that consumers have unbiased expectations of their drug needs for the upcoming year at the time they make enrollment decisions. As a robustness check, we also follow prior studies and repeat the estimation under the assumption that consumers are myopic in the sense that they expect their drug use in year *t* to be identical to their drug use in year *t-1*. Appendix Table A4 shows that this has little impact on our results.

2. **Inertia**, defined as whether an individual was passively reenrolled in their default plan because they did not actively switch to a different one during the annual open enrollment period (e.g., Kling et al. 2012, Ho, Hogan and Scott Morton 2015, Ketcham, Lucarelli and Powers 2015).

3. **Dominated choices**, meaning the individual's chosen plan was off the efficient frontier in attribute space when PDPs are characterized by some combination of quality characteristics and moments of an individual's distribution of potential expenditures (e.g., Ketcham, Kuminoff and Powers 2016 and Keane et al. 2018).[44]

We analyze how dementia affects these three outcomes, using data and variable definitions that are consistent with prior studies in the Medicare Part D literature.[45] Specifically, we estimate

$$(5)\quad q_{it} = \alpha y_{it} + \beta X_{it} + \theta W_{it} + \gamma H_{it} + \eta_i + \tau_{it} + \epsilon_{it},$$

where q_{it} is a decision making outcome for person *i* in year *t*. The covariates parallel those in our model of individual dementia diagnoses in equation (1), with three differences. First, y_{it} includes two variables of interest, an indicator for whether person *i* received a dementia diagnosis by year *t* and an indicator for whether a diagnosis was received by the end of 2013 but not by *t*. The second term allows us to assess the role of subclinical cognitive decline. Second, we control for diagnoses of dementia risk factors (hypertension, diabetes, congestive heart failure, ischemic heart disease, stroke) by the year that decisions are made because these diagnoses may affect expected drug needs. Finally, we add state-by-year dummies (τ_{it}) to absorb all of the spatial and temporal heterogeneity in the structure of PDP choice sets (e.g., variation in the number of plans, the number of brands, and plan characteristics—all of which are fixed within a state and year). These dummies force the identification to come from variation in how different individuals choose among the same PDP options. Our data span the first five years the markets operated: 2006-2010.

[44] For example, let c_{ijt} represent consumer *i*'s total expenditures under plan *j* in year *t*. It equals the premium for that plan plus the total out of pocket cost of drugs used by the consumer. If utility depends on plan quality, q_{jt}, and the first two moments of the distribution of potential expenditures, then a weakly risk averse and fully informed consumer whose preference ordering is complete, transitive and monotonic will not choose a plan *j* during year *t* if that plan is dominated by another plan, *k*, in the sense that $E(c_{ikt}) < E(c_{ijt})$, $var(c_{ikt}) < var(c_{ijt})$, and $q_{jt} < q_{kt}$.

[45] We define potential savings using the cost calculator developed by Ketcham, Lucarelli, and Powers (2015). The calculator uses data on the universe of prescriptions filled for each person in each year. It holds those prescriptions fixed across all plans available to the person, using plan formularies and cost structures from CMS to calculate the counterfactual cost of the person's chosen bundle of drugs under every alternative plan in the person's choice set. We also use the calculator to implement a standard cohort-based approach to defining the variance of each person's potential expenditures for each plan in each year. To define the person-plan-year specific variance, we use our full sample and assign each individual to 1 of 1000 cells defined by the deciles to which she belonged in the national distributions of the prior year's total drug spending, days' supply of branded drugs, and days' supply of generic drugs. Then we calculate each plan's variance from the distribution of costs from the cost calculator that arises from the distribution of drugs used by everyone in consumer *i*'s cell and region. If we lack a person's prior year's prescriptions (e.g., the year they first enter the market) we predict them based on health and individual-specific future prescriptions.

Table VI shows that dementia has large negative effects on all of the financial outcomes. All else constant, having dementia increases annual potential saving by $37 (11% of the sample mean) relative to those who were not diagnosed with dementia by the end of 2013. Dementia also reduces the probability of switching plans by 0.4 pp (4% of the sample mean), and increases the probability of choosing dominated plans by 3 to 5 pp (4-12%). The middle column shows that dementia patients are 3 pp more likely to choose a plan that had weakly higher costs and less risk protection (based on the variance of expenditures under a distribution of possible health states). The last two columns show that dementia's negative effect persists when we also condition on measures of plan quality (star ratings, insurance company dummies) that could proxy for better customer service and lower transaction costs of obtaining drugs.

TABLE VI: EFFECTS OF DEMENTIA ON PRESCRIPTION DRUG PLAN CHOICES

	Potential savings ($)	Probability of actively switching out of default plan	Probability chosen plan is dominated in:		
			cost, variance	cost, variance, star rating	cost, variance, insurer
dementia	36.66***	-0.37***	3.10***	4.91***	2.92***
	(1.00)	(0.07)	(0.08)	(0.10)	(0.12)
future dementia	10.01***	-0.59***	1.04***	1.42***	0.80***
	(1.20)	(0.07)	(0.08)	(0.09)	(0.11)
mean of dependent variable	346	10	72	52	25
sample size	3,445,118	2,575,534	3,445,118	3,445,118	2,218,189

Note: Each column reports coefficients estimates from models regressing decision making outcomes on indicators for whether the beneficiary is diagnosed with Alzheimer's disease and related dementias at the time of their enrollment decision (dementia) or after their enrollment decision but before the end of 2013 (future dementia) in comparison to beneficiaries not diagnosed with dementia by the end of the 2013. All models pool data from 2006 to 2010 and include residential CBSA dummies, state-by- year dummies, integer age-by-gender dummies, covariates describing individual health and Medicare expenditures prior to entering Medicare Part D, and covariates describing individual and neighborhood demographics. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels based on robust standard errors clustered at the Census block group level.

Our results also provide the first market-based evidence that subclinical impairments influence financial outcomes, as hypothesized by Agarwal et al. (2009). Relative to those who were not diagnosed with dementia by the end of 2013, financial outcomes are worse among those who became diagnosed with dementia by the end of our study period but were not currently diagnosed at the time of their Part D plan choices.

Despite widespread concern about older adults' financial literacy (Lusardi and Mitchell 2014), this is the first evidence that dementia patients have systematically worse outcomes from actual

financial decisions.[46] Dementia may impair these decisions by eroding patients' cognitive skills and complicating intrahousehold bargaining.[47] If our findings for Medicare Part D are representative of older adults' decision making more broadly, then reducing the prevalence of dementia by regulating $PM_{2.5}$ may lead to substantial gains to consumer surplus through better financial decisions.

C. Dementia Risk and the EPA's Regulatory Threshold

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 $\mu g/m^3$), fell between the 1997 and 2012 thresholds (12 to 15 $\mu g/m^3$), or fell below the 2012 threshold (12 $\mu g/m^3$).

The first column of Table VII corresponds to our main 2SLS specification (Table I, column (6)). The next three columns repeat estimation after replacing the CBSA dummies with county dummies and/or replacing the 4^{th} order polynomial function of baseline exposure with dummies for 72 baseline exposure bins. Regardless of econometric specification, the point estimates indicate that the marginal effects 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) and at concentrations below 12 $\mu g/m^3$ specifically (Di et al. 2017). While confidence intervals on the point estimates are too wide to conclusively determine that marginal effects are larger at lower exposure levels, we can rule out that the effects diminish below 12 $\mu g/m^3$ with a high degree of confidence. These findings indicate that the 2012 policy change is likely to

[46] Prior studies have relied exclusively on small-scale interview-based measures of financial literacy and found that dementia impairs "financial capacity" in such contexts. For example, Ketcham, Kuminoff, and Powers (2016) find that Medicare beneficiaries diagnosed with dementia are less likely to provide the correct answer to a survey question about a key institutional feature of prescription drug insurance markets that they are participating in. Agarwal et al. (2009) is the closest precedent, finding that suboptimal credit card decisions become more prevalent with age and surmising that dementia may play a role.

[47] Dementia may negatively affect financial decision making through several pathways. First, dementia may increase the cost of cognitive processing required to make decisions, or even render them impossible without assistance from others, which may introduce agency problems. Second, the marginal utility of consumption may be health-state dependent and declining in chronic conditions such as dementia (Finkelstein, Luttmer and Notowidigdo 2013). Third, prior research has identified discount rates as a key source of heterogeneity in financial decision making and chronic condition diagnoses increase discount rates (Huffman, Maurer, and Mitchell 2016, Oster, Shoulson, and Dorsey 2013). Fourth, because dementia reduces life expectancy, it may reduce investments in financial skills (Lusardi and Mitchell 2014) or in health capital, leading to additional chronic conditions that increase the complexity of decisions about health insurance (Fang et al. 2007).

continue to improve health by reducing dementia, as would further reductions of the threshold.

TABLE VII: EFFECTS OF $PM_{2.5}$ RELATIVE TO REGULATORY THRESHOLDS

Annual average	(1)	(2)	(3)	(4)
Annual average $PM_{2.5}$ exposure < 12 μg/m^3	1.845**	1.575***	2.144***	1.257***
	(0.78)	(0.49)	(0.62)	(0.46)
12 μg/m3 ≤ annual average $PM_{2.5}$ exposure ≥ 15 μg/m^3	1.761**	1.591***	1.971***	1.208***
	(0.77)	(0.49)	(0.61)	(0.45)
Annual average $PM_{2.5}$ exposure > 15 μg/m^3	1.350***	1.303***	1.548***	0.835**
	(0.46)	(0.40)	(0.47)	(0.40)
control for baseline exposure				
4th order polynomial	x	x		
spline: 0.33 μg bin dummies			x	x
2005 attainment instruments				
county	x		x	
county x monitor		x		x
Number of individuals	1,256,440	1,256,440	1,256,440	1,256,440
Share with dementia in 2013	19.2	19.2	19.2	19.2

Note: Each column represents results from a micro model identical to one in Tables I and II except that the models here allow the marginal effects of decadal exposure to differ across $PM_{2.5}$ levels. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels based on robust standard errors clustered at the Census block group level.

VII. Discussion and Conclusions

Our findings provide the first large scale evidence to support the hypothesis from medical research that long-term exposure to air pollution causes dementia among older adults, reducing the quantity and quality of their lives and impairing their financial decisions. We find that air pollution's effect on dementia is driven by fine particulate matter, not by other federally regulated air pollutants, and the effects are driven by Alzheimer's disease rather than vascular dementia resulting from strokes. These results build on prior knowledge that short-term and annualized measures of $PM_{2.5}$ exposure cause mortality (Deryugina et al. 2016, Di et al. 2017) by adding the insight that long-term exposures to $PM_{2.5}$ also reduces people's quantity and quality of life due to dementia specifically.

Dementia's global social costs continue to grow as populations in many counties age, 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: regulating air pollution. 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 dementia cases avoided ($163 billion) are sufficiently large to suggest that dementia-related benefits may matter for future benefit-cost analyses of federal air quality regulations.

There are several potential directions for future research. First, evaluations of policies targeting dementia could be improved by developing direct estimates for household-level willingness to pay to reduce the statistical risk of dementia. Second, while prescription drug insurance markets are often used to evaluate older adults' abilities to make complex financial decisions, it would be informative to test the generalizability of behavior in these markets by matching dementia diagnoses to other high-stakes financial choices such as investment of retirement savings, use of credit, housing transactions, and estate planning. Third, our findings raise several questions about potential heterogeneity in $PM_{2.5}$'s effects on dementia. For instance, as compositional data on air pollution improve it may be possible to determine whether the effects of $PM_{2.5}$ are driven by particulates from certain production processes such as automobiles, power plants, or industrial manufacturing. Research should also consider whether cognitive decline from long-term exposure can be reversed by moving to cleaner areas or mitigated by investing in human capital earlier in life such as through schooling. Policies targeting air pollution may also have implications for equity. Our data show that African-American and Hispanic individuals are about twice as likely to acquire dementia, as are people who live in areas with lower income and less education. Our results suggest that differences in neighborhood air quality may contribute to these socioeconomic disparities in disease burden. Finally, researchers could develop a life cycle model that integrates the evidence on the effects of particulate matter on human capital during the early, middle, and now late stages of life to gain insights about the dynamics of pollution, migration, health and human capital production.

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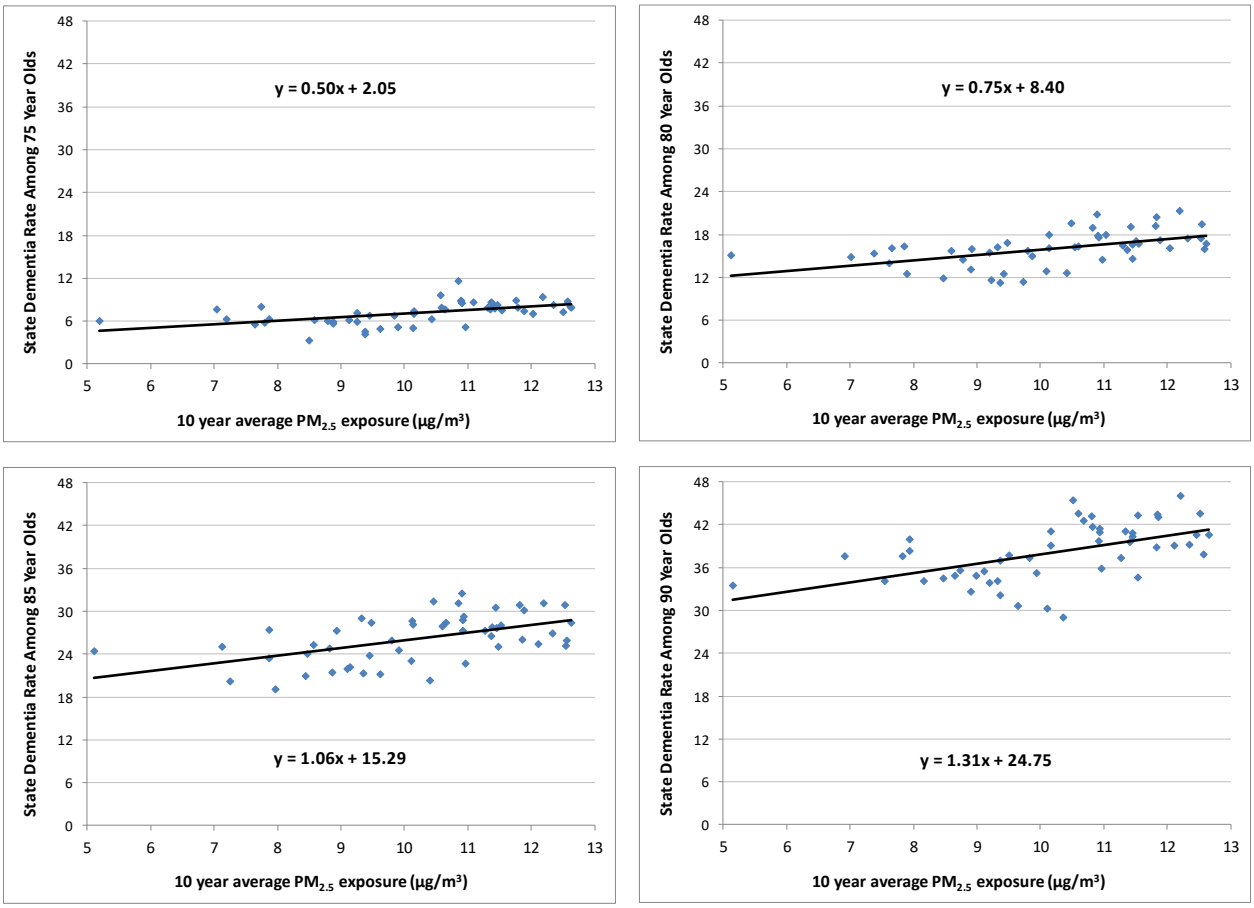
SUPPLEMENTAL APPENDIX A: FOR ONLINE PUBLICATION

FIGURE A1: SPATIAL CORRELATION BETWEEN $PM_{2.5}$ AND DEMENTIA IN 2013 BY AGE



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).

TABLE A1—SUMMARY STATISTICS FOR MEDICARE BENEFICIARY SAMPLES

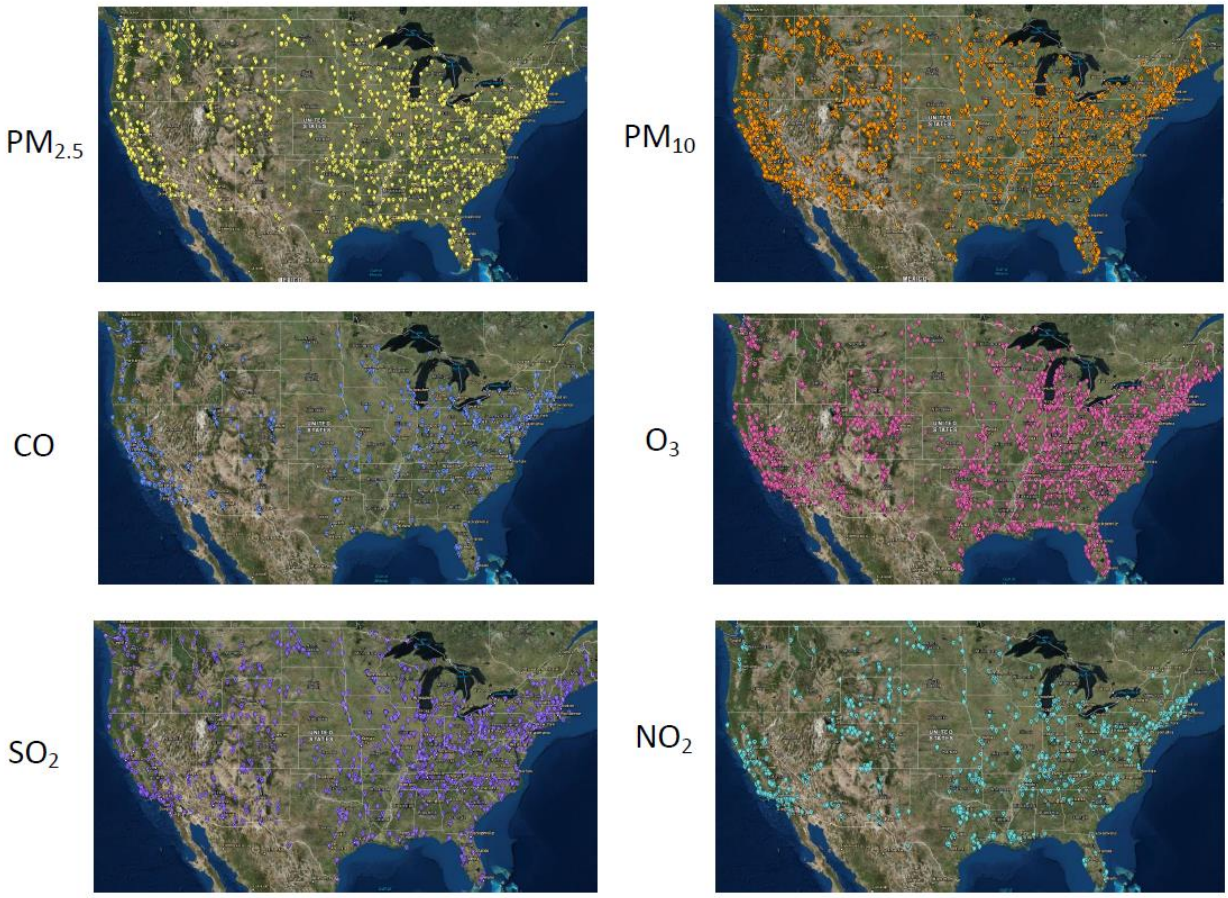
	(1)	(2)	(3)	(4)	(5)
	Traditional Medicare and Part D	Traditional Medicare and Part D	Part D	Traditional Medicare, Medicare Advantage, and Part D	Traditional Medicare, Medicare Advantage, and Part D
# people in sample	6,901,476	1,851,175	1,136,336	10,319,644	13,603,253
mean # years per person	8	14	12	9	8
always observe ZIP+4	yes	yes	yes	yes	no
Individual demographics					
mean age at sample entry	70.7	69.1	69.4	70.3	70.7
mean age in 2013	80.5	82.0	80.7	80.3	80.8
male (%)	44	40	37	43	44
white (%)	83	87	93	81	81
black (%)	8	6	3	8	8
asian (%)	3	2	1	3	3
hispanic (%)	5	4	2	6	7
alive at beginning of 2013 (%)	63	100	77	66	64
mean age at death	83	86	85	83	83
ever moved (%)	19	28	25	21	24
ever moved county (%)	9	14	13	10	9
ever moved state (%)	5	8	7	5	5
2013 gross Medicare expenditures ($)	9,076	12,680	11,808		
Ever diagnosed with					
dementia (%)	23	19	23		
stroke (%)	19	20	21		
diabetes (%)	32	38	37		
congestive heart failure (%)	36	34	37		
ischemic heart disease (%)	48	58	58		
hypertension (%)	72	86	87		
glaucoma (%)	19	29	27		
breast cancer (%)	5	7	8		
prostate cancer (%)	6	8	7		
fibromyalgia (%)	15	24	23		
viral hepatitis (%)	1	1	1		
periphreal vascular disease (%)	24	28	28		

Note: Column (1) describes the individuals used in county level models. They comprise an unbalanced panel from 2004 to 2013. Column (2) describes the individuals used in micro level models. They comprise a balanced panel from 2001 to 2013. Column (3) describes the individuals used in models of Medicare Part D outcomes. They comprise an unbalanced panel from 2006 to 2010. Column (4) adds to column (1) the subset of people we exclude because they enrolled in Medicare Advantage plans at some point during our study period, preventing us from directly observing if and when they were first diagnosed with dementia. Column (5) adds to column (1) the subset of people we exclude because they had mail delivered to a post office box at some point during our study period, preventing us from observing their residential location.

TABLE A1 CONTINUED—SUMMARY STATISTICS FOR MEDICARE BENEFICIARY SAMPLE

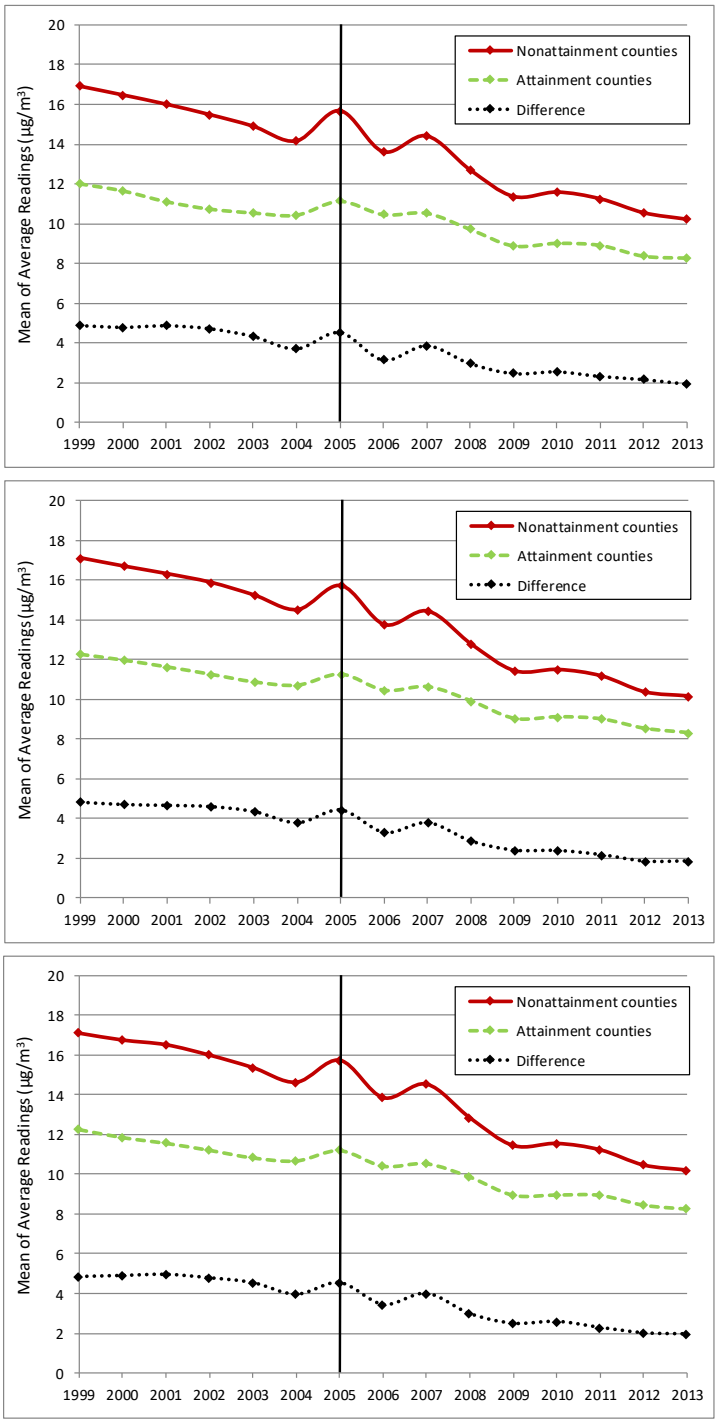
	(1)	(2)	(3)	(4)	(5)
	Traditional Medicare and Part D	Traditional Medicare and Part D	Part D	Traditional Medicare, Medicare Advantage, and Part D	Traditional Medicare, Medicare Advantage, and Part D
# people in sample	6,901,476	1,851,175	1,136,336	10,319,644	13,603,253
mean # years per person	8	14	12	9	8
always observe ZIP+4	yes	yes	yes	yes	no
Annual average pollution (2013)					
$PM_{2.5}$ (hourly $\mu g/m^3$)	9.03	9.00	9.01	9.05	
PM_{10} (hourly $\mu g/m^3$)	18.45	18.26	18.32	18.72	
ozone (daily max of 8-hr mean ppm)	0.04	0.04	0.04	0.04	
carbon monoxide (8-hr mean ppm)	0.30	0.30	0.29	0.30	
sulfur dioxide (daily mean ppb)	10.53	10.39	10.13	10.66	
nitrogen dioxide (daily mean ppb)	1.13	1.15	1.15	1.14	
Census block group data (2012)					
household income (median)	61,939	63,119	64,696	61,079	
income per capita	31,285	32,267	33,644	30,615	
year built (median)	1971	1971	1971	1971	
house value (median)	238,779	244,732	245,055	238,711	
house value (average)	120,682	126,751	132,769	118,856	
gross rent (median)	2,440	2,587	2,695	2,363	
population over 65 (%)	18	18	19	17	
population white not hispanic (%)	71	73	78	69	
population black (%)	11	10	8	11	
population hispanic (%)	11	10	9	13	
education: 8th or less (%)	5	5	4	5	
education: 9th to 12th (%)	8	7	7	8	
education: high school grad (%)	28	28	28	29	
education: some college (%)	21	21	21	21	
education: associate degree (%)	8	8	8	8	
education: bachelor's degree (%)	18	19	19	18	
education: graduate degree (%)	12	12	13	11	
owner occupied (%)	63	64	66	62	
renter occupied (%)	27	25	23	27	
vacant (%)	11	11	11	10	

FIGURE A2: LOCATIONS OF EPA MONITORING STATIONS FOR CRITERIA AIR POLLUTANTS



Each map shows the locations of air quality monitors for a particular air pollutant: particulate matter smaller than 2.5 microns in diameter ($PM_{2.5}$), particulate matter smaller than 10 microns (PM_{10}), ozone (O_3), carbon monoxide (CO), nitrogen dioxide (NO_2) and sulfur dioxide (SO_2). The maps were 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 A3: AIR POLLUTION TRENDS: UNBALANCED AND BALANCED MONITOR PANELS



The middle figure is identical to Figure V. It displays air pollution trends based on a balanced panel of 488 monitors in operation continuously from 2001-2013. For comparison, the top figure is based on averages taken each year over an unbalanced panel of all operating monitors (between 787 and 1,106 monitors per year). The bottom figure is based on a balanced panel of 393 monitors in operation continuously from 1999 through 2013.

Table A2 reports coefficients, robust standard errors clustered by Census block group, and 95% confidence intervals for the specification from Table I, column (6). For brevity we do not report dummy variable coefficients for approximately one thousand Core Business Statistical Areas. Table A2.A reports second-stage results and Table A2.B reports first-stage results.

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)	1.2880	0.4590	0.3883	2.1877
Chronic conditions in 2004				
H	1.8210	0.0899	1.6448	1.9972
S	8.7642	0.5983	7.5916	9.9368
S, H	10.2099	0.3660	9.4926	10.9272
D	4.2402	0.2584	3.7337	4.7466
D, H	4.6247	0.1518	4.3271	4.9224
D, S	15.3799	1.9692	11.5202	19.2395
D, S, H	13.9235	0.6562	12.6374	15.2097
I	3.0074	0.1784	2.6577	3.3571
I, H	3.6437	0.1253	3.3982	3.8892
I, S	10.7376	0.8798	9.0131	12.4621
I, S, H	12.0605	0.3593	11.3562	12.7648
I, D	5.2295	0.4674	4.3135	6.1455
I, D, H	6.5407	0.1837	6.1807	6.9007
I, D, S	10.4270	2.1400	6.2326	14.6214
I, D, S, H	15.3417	0.5227	14.3173	16.3661
C	5.0845	0.6057	3.8973	6.2716
C, H	5.3820	0.3329	4.7295	6.0346
C, S	8.8339	2.8206	3.3055	14.3622
C, S, H	12.6208	1.1028	10.4593	14.7822
C, D	8.6904	1.6104	5.5340	11.8467
C, D, H	9.1610	0.4977	8.1855	10.1365
C, D, S	21.8199	6.4948	9.0902	34.5496
C, D, S, H	19.2555	1.5805	16.1577	22.3533
C, I	4.7408	0.5345	3.6931	5.7885
C, I, H	6.6400	0.2178	6.2132	7.0669
C, I, S	10.3657	1.9153	6.6118	14.1196
C, I, S, H	14.8014	0.5195	13.7832	15.8196
C, I, D	7.9863	1.1358	5.7602	10.2125
C, I, D, H	10.4413	0.2623	9.9271	10.9555
C, I, D, S	21.3867	4.5025	12.5619	30.2115
C, I, D, S, H	21.8414	0.5792	20.7061	22.9766

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 CONTINUED— SECOND STAGE RESULTS FROM THE MAIN 2SLS SPECIFICATION

	coefficient	Robust standard error	95% Confidence Interval	
2004 Gross Medicare Expenditures ($10,000)				
expenditures	4.0505	0.1326	3.7906	4.3104
expenditures^2	-0.5336	0.0380	-0.6080	-0.4592
expenditures^3	0.0213	0.0025	0.0164	0.0262
expenditures^4	-0.0002	0.0000	-0.0003	-0.0002
Age (females)				
75	-0.2761	0.1575	-0.5848	0.0326
76	0.2240	0.1652	-0.0999	0.5479
77	1.3026	0.1767	0.9563	1.6489
78	2.2219	0.1865	1.8563	2.5876
79	4.0613	0.1994	3.6705	4.4521
80	5.6679	0.2139	5.2486	6.0872
81	6.6002	0.2185	6.1720	7.0284
82	8.3129	0.2295	7.8631	8.7627
83	10.6962	0.2361	10.2335	11.1589
84	12.2144	0.2474	11.7295	12.6992
85	14.6477	0.2592	14.1396	15.1557
86	16.8637	0.2708	16.3329	17.3945
87	19.0497	0.2874	18.4864	19.6130
88	21.6289	0.3022	21.0367	22.2211
89	24.2650	0.3176	23.6424	24.8875
90	26.1576	0.3419	25.4875	26.8278
91	28.8127	0.3726	28.0823	29.5430
92	30.1713	0.3945	29.3980	30.9446
93	32.2683	0.4382	31.4094	33.1271
94	35.4521	0.5104	34.4518	36.4525
95	37.0542	0.5600	35.9566	38.1517
96	39.1820	0.6432	37.9213	40.4427
97	40.9426	0.7502	39.4723	42.4129
98	42.3773	0.8749	40.6626	44.0920
99	45.8055	0.9676	43.9090	47.7021
100 and over	18.7867	0.4718	17.8620	19.7114

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

	coefficient	Robust standard error	95% Confidence Interval	
male	-0.6739	0.1594	-0.9863	-0.3615
Age (males)				
75	-0.0810	0.2281	-0.5282	0.3661
76	0.0804	0.2406	-0.3912	0.5519
77	-0.2161	0.2561	-0.7181	0.2859
78	-0.6464	0.2714	-1.1783	-0.1145
79	-1.0907	0.2891	-1.6574	-0.5240
80	-1.2386	0.3131	-1.8523	-0.6249
81	-1.3260	0.3221	-1.9574	-0.6946
82	-1.4963	0.3403	-2.1633	-0.8294
83	-2.4149	0.3513	-3.1034	-1.7263
84	-2.3897	0.3744	-3.1234	-1.6559
85	-2.6583	0.3978	-3.4380	-1.8786
86	-2.8728	0.4199	-3.6957	-2.0498
87	-3.9090	0.4490	-4.7890	-3.0289
88	-4.0464	0.4815	-4.9900	-3.1028
89	-4.5280	0.5150	-5.5373	-3.5186
90	-5.0953	0.5630	-6.1987	-3.9919
91	-6.3202	0.6154	-7.5264	-5.1140
92	-4.7300	0.6773	-6.0575	-3.4024
93	-4.8036	0.7682	-6.3093	-3.2979
94	-7.0531	0.9083	-8.8332	-5.2729
95	-7.0787	1.0512	-9.1391	-5.0184
96	-8.2181	1.2269	-10.6228	-5.8134
97	-7.4071	1.4974	-10.3419	-4.4723
98	-9.8520	1.7825	-13.3457	-6.3583
99	-13.7820	2.1583	-18.0122	-9.5519
100 and over	-12.9798	0.7490	-14.4478	-11.5118

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

	coefficient	Robust standard error	95% Confidence Interval	
White	1.6698	0.2972	1.0874	2.2522
Black	3.4040	0.3353	2.7468	4.0613
Asian	1.8820	0.3541	1.1880	2.5761
Hispanic	3.1176	0.3387	2.4539	3.7813
2004 Census Block Group Demographics				
median household income / 1000	-0.0001	0.0024	-0.0047	0.0045
per capita income / 1000	-0.0132	0.0037	-0.0205	-0.0060
median year built	0.0006	0.0025	-0.0043	0.0055
median house value / 1000	-0.0014	0.0004	-0.0022	-0.0007
average house value / 1000	0.0000	0.0001	-0.0002	0.0003
median gross income / 1000	0.0106	0.0060	-0.0011	0.0224
% over 65	1.3938	0.3754	0.6580	2.1296
% white	0.5297	0.4110	-0.2758	1.3352
% black	0.9974	0.4528	0.1099	1.8849
% hispanic	0.2138	0.4743	-0.7158	1.1434
% 9th through 12th	-0.1943	1.1348	-2.4184	2.0298
% high school graduate	-2.0321	0.8658	-3.7291	-0.3352
% some college	-4.1794	0.8634	-5.8716	-2.4873
% associate degree	-4.2027	1.0842	-6.3277	-2.0778
% bachelor's degree	-3.3805	0.8607	-5.0675	-1.6936
% graduate degree	-4.0097	0.9067	-5.7869	-2.2325
% owner occupied	-1.6389	0.3902	-2.4038	-0.8741
% renter occupied	2.1927	0.4402	1.3299	3.0555
$PM_{2.5}$ (1 $\mu g/m^3$) (Baseline, 2001-2003)				
exposure	-1.9583	1.6131	-5.1199	1.2033
$exposure^2$	0.1601	0.1766	-0.1860	0.5062
$exposure^3$	-0.0086	0.0082	-0.0246	0.0074
$exposure^4$	0.0002	0.0001	-0.0001	0.0004

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

	coefficient	Robust standard error	95% Confidence Interval	
Chronic conditions in 2004				
H	-0.0012	0.0008	-0.0029	0.0004
S	0.0003	0.0050	-0.0096	0.0102
S, H	-0.0002	0.0026	-0.0053	0.0049
D	-0.0031	0.0025	-0.0079	0.0017
D, H	-0.0034	0.0012	-0.0058	-0.0010
D, S	-0.0032	0.0141	-0.0309	0.0244
D, S, H	-0.0005	0.0046	-0.0096	0.0086
I	-0.0017	0.0017	-0.0050	0.0015
I, H	-0.0021	0.0011	-0.0042	0.0000
I, S	-0.0028	0.0070	-0.0164	0.0109
I, S, H	0.0007	0.0026	-0.0045	0.0058
I, D	-0.0019	0.0038	-0.0094	0.0056
I, D, H	-0.0052	0.0015	-0.0081	-0.0022
I, D, S	-0.0238	0.0164	-0.0559	0.0082
I, D, S, H	-0.0016	0.0037	-0.0089	0.0057
C	-0.0051	0.0047	-0.0144	0.0041
C, H	-0.0027	0.0026	-0.0079	0.0025
C, S	-0.0368	0.0254	-0.0866	0.0129
C, S, H	-0.0027	0.0080	-0.0183	0.0130
C, D	0.0027	0.0141	-0.0250	0.0304
C, D, H	-0.0024	0.0039	-0.0101	0.0054
C, D, S	-0.0153	0.0327	-0.0794	0.0489
C, D, S, H	-0.0145	0.0110	-0.0361	0.0070
C, I	0.0026	0.0043	-0.0057	0.0110
C, I, H	0.0022	0.0017	-0.0012	0.0056
C, I, S	-0.0042	0.0138	-0.0313	0.0229
C, I, S, H	-0.0060	0.0039	-0.0137	0.0017
C, I, D	-0.0046	0.0093	-0.0229	0.0137
C, I, D, H	0.0028	0.0021	-0.0013	0.0069
C, I, D, S	0.0141	0.0373	-0.0590	0.0871
C, I, D, S, H	0.0005	0.0044	-0.0081	0.0091

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 CONTINUED— FIRST STAGE RESULTS FROM THE MAIN 2SLS SPECIFICATION

	coefficient	Robust standard error	95% Confidence Interval	
2004 Gross Medicare Expenditures ($10,000)				
expenditures	5.3730	9.9780	-14.1830	24.9290
expenditures^2	-1.4370	2.6470	-6.6250	3.7510
expenditures^3	0.1050	0.1500	-0.1900	0.4000
expenditures^4	-0.0015	0.0018	-0.0050	0.0020
Age (females)				
75	0.0000	0.0021	-0.0040	0.0041
76	0.0020	0.0021	-0.0021	0.0062
77	-0.0003	0.0021	-0.0044	0.0038
78	0.0012	0.0021	-0.0029	0.0054
79	-0.0005	0.0022	-0.0048	0.0037
80	0.0027	0.0022	-0.0016	0.0070
81	0.0027	0.0022	-0.0016	0.0069
82	0.0029	0.0022	-0.0015	0.0072
83	0.0036	0.0022	-0.0008	0.0079
84	0.0014	0.0023	-0.0030	0.0058
85	0.0046	0.0023	0.0002	0.0091
86	0.0055	0.0024	0.0009	0.0101
87	0.0030	0.0024	-0.0018	0.0078
88	0.0053	0.0025	0.0004	0.0101
89	0.0058	0.0026	0.0007	0.0110
90	0.0038	0.0027	-0.0015	0.0091
91	0.0022	0.0029	-0.0034	0.0078
92	0.0078	0.0031	0.0018	0.0138
93	0.0029	0.0034	-0.0038	0.0096
94	0.0024	0.0038	-0.0050	0.0098
95	0.0049	0.0042	-0.0034	0.0131
96	0.0032	0.0046	-0.0059	0.0123
97	0.0033	0.0055	-0.0075	0.0140
98	0.0113	0.0060	-0.0005	0.0231
99	0.0038	0.0072	-0.0103	0.0178
100 and over	0.0173	0.0038	0.0099	0.0246

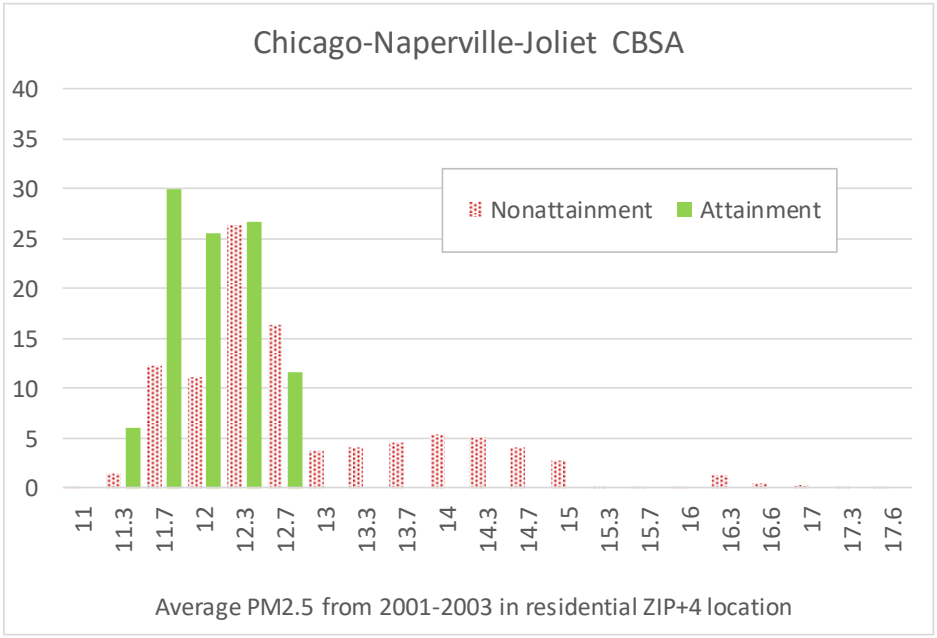
TABLE A2.B CONTINUED— FIRST STAGE RESULTS FROM THE MAIN 2SLS SPECIFICATION

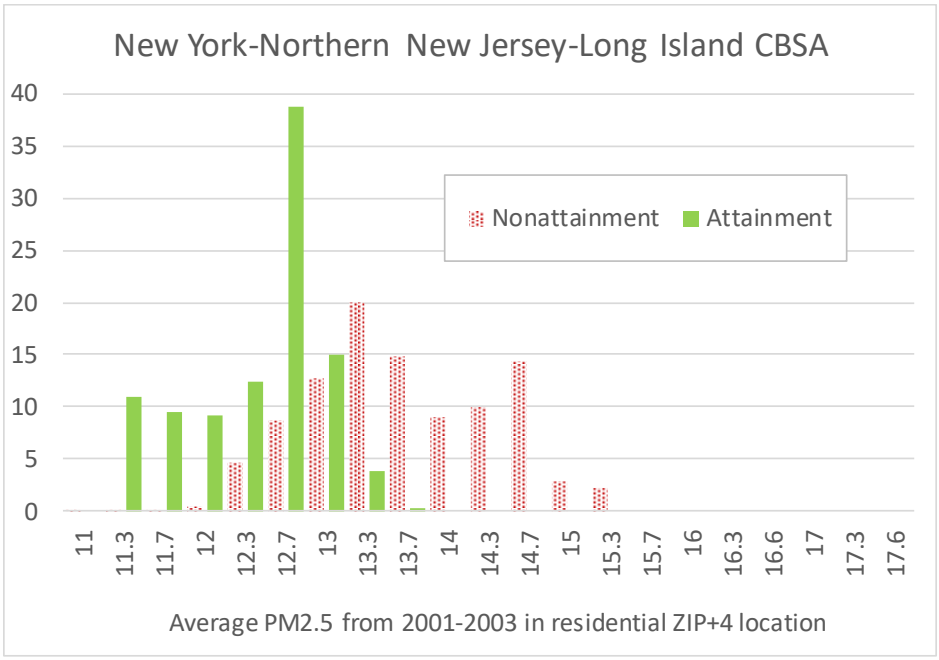
	coefficient	Robust standard error	95% Confidence Interval	
male	-0.0051	0.0023	-0.0097	-0.0006
Age (males)				
75	0.0079	0.0032	0.0017	0.0141
76	0.0050	0.0032	-0.0012	0.0113
77	0.0068	0.0032	0.0005	0.0131
78	0.0067	0.0033	0.0002	0.0131
79	0.0039	0.0033	-0.0027	0.0104
80	0.0057	0.0034	-0.0010	0.0123
81	0.0054	0.0034	-0.0012	0.0120
82	0.0052	0.0034	-0.0015	0.0119
83	0.0047	0.0034	-0.0020	0.0115
84	0.0044	0.0035	-0.0024	0.0113
85	0.0054	0.0035	-0.0015	0.0122
86	0.0042	0.0036	-0.0028	0.0113
87	0.0041	0.0038	-0.0033	0.0115
88	0.0059	0.0039	-0.0018	0.0136
89	0.0050	0.0041	-0.0031	0.0131
90	0.0082	0.0043	-0.0003	0.0167
91	0.0043	0.0047	-0.0049	0.0135
92	0.0005	0.0049	-0.0091	0.0102
93	0.0104	0.0055	-0.0004	0.0213
94	0.0192	0.0068	0.0057	0.0326
95	0.0112	0.0075	-0.0036	0.0260
96	0.0166	0.0087	-0.0004	0.0336
97	0.0150	0.0106	-0.0057	0.0357
98	0.0020	0.0136	-0.0247	0.0286
99	-0.0068	0.0150	-0.0361	0.0225
100 and over	0.0660	0.0095	0.0473	0.0847

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

	coefficient	Robust standard error	95% Confidence Interval	
White	-0.0112	0.0033	-0.0178	-0.0046
Black	-0.0060	0.0037	-0.0133	0.0014
Asian	0.0044	0.0045	-0.0045	0.0133
Hispanic	0.0106	0.0040	0.0028	0.0185
2004 Census Block Group Demographics				
median household income / 1000	-0.0005	0.0001	-0.0006	-0.0004
per capita income / 1000	0.0017	0.0001	0.0015	0.0019
median year built	-0.0003	0.0001	-0.3977	-0.1736
median house value / 1000	-0.0001	0.0000	-0.0001	-0.0001
average house value / 1000	0.0000	0.0000	0.0000	0.0000
median gross income / 1000	-0.0001	0.0002	-0.0004	0.0002
% over 65	-0.0895	0.0120	-0.1130	-0.0661
% white	0.0890	0.0113	0.0668	0.1111
% black	0.0574	0.0116	0.0346	0.0802
% hispanic	0.0846	0.0130	0.0591	0.1101
% 9th through 12th	-0.1291	0.0252	-0.1785	-0.0798
% high school graduate	-0.1102	0.0209	-0.1512	-0.0692
% some college	-0.1615	0.0210	-0.2027	-0.1203
% associate degree	-0.2372	0.0254	-0.2870	-0.1873
% bachelor's degree	-0.0802	0.0209	-0.1212	-0.0393
% graduate degree	-0.0733	0.0227	-0.1178	-0.0289
% owner occupied	-0.0377	0.0091	-0.0555	-0.0199
% renter occupied	0.0231	0.0103	0.0029	0.0432
$PM_{2.5}$ (1 μg/m^3) (Baseline, 2001-2003)				
exposure	0.6445	0.1536	0.3436	0.9455
exposure^2	-0.0452	0.0205	-0.0854	-0.0051
exposure^3	0.0045	0.0012	0.0022	0.0068
exposure^4	-0.0001	0.0000	-0.0002	-0.0001
Nonattainment * $PM_{2.5}$ (1 μg/m^3) (2001-2003)				
Nonattainment	-24.8634	1.4321	-27.6702	-22.0566
Nonattainment * exposure	5.9907	0.3246	5.3545	6.6270
Nonattainment * exposure^2	-0.4037	0.0352	-0.4728	-0.3347
Nonattainment * exposure^3	0.0162	0.0011	0.0140	0.0183
Nonattainment * exposure^4	-0.0001	0.0000	-0.0001	0.0000

FIGURE A4—WITHIN-CBSA VARIATION IN NONATTAINMENT STATUS BY BASELINE $PM_{2.5}$ LEVELS

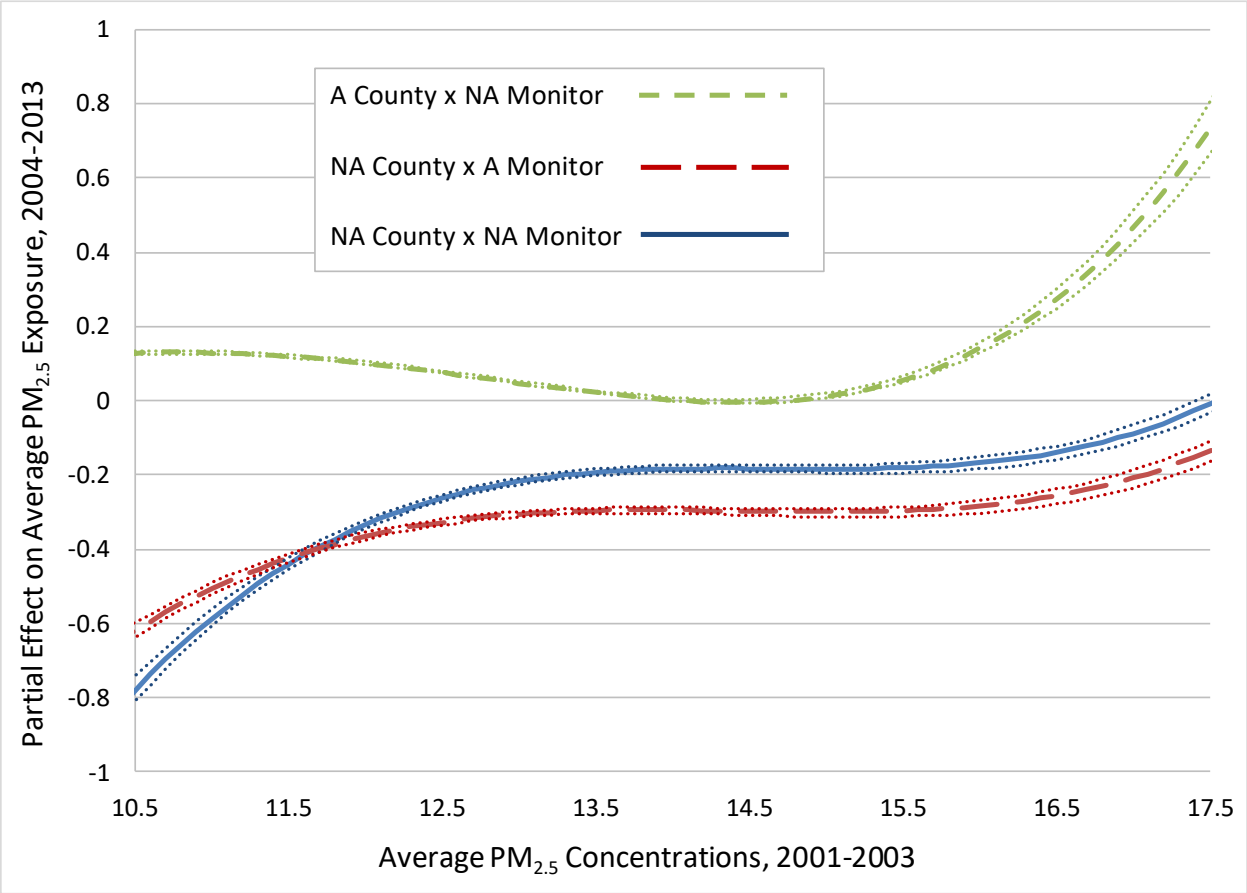




The figures illustrate the variation in county nonattainment status conditional on baseline residential $PM_{2.5}$ concentrations from 2001-2003 within two of the largest Core Business Statistical Areas that jointly account for about 16% of the people in our sample. The vertical axes report the fractions of people in 0.33 microgram per cubic meter bins describing baseline $PM_{2.5}$ concentrations

for residential areas in nonattainment and attainment counties at the time nonattainment designations were made. For example, about 5% of people living in nonattainment counties within the New York - New Jersey - Long Island CBSA in 2005 were living in neighborhoods that had baseline concentrations of 12.3 micrograms per cubic meter. The corresponding fraction is about 12.5% for those living in attainment counties. This is one source of conditional variation that underlies the identification of the effects of $PM_{2.5}$ in our 2SLS models.

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



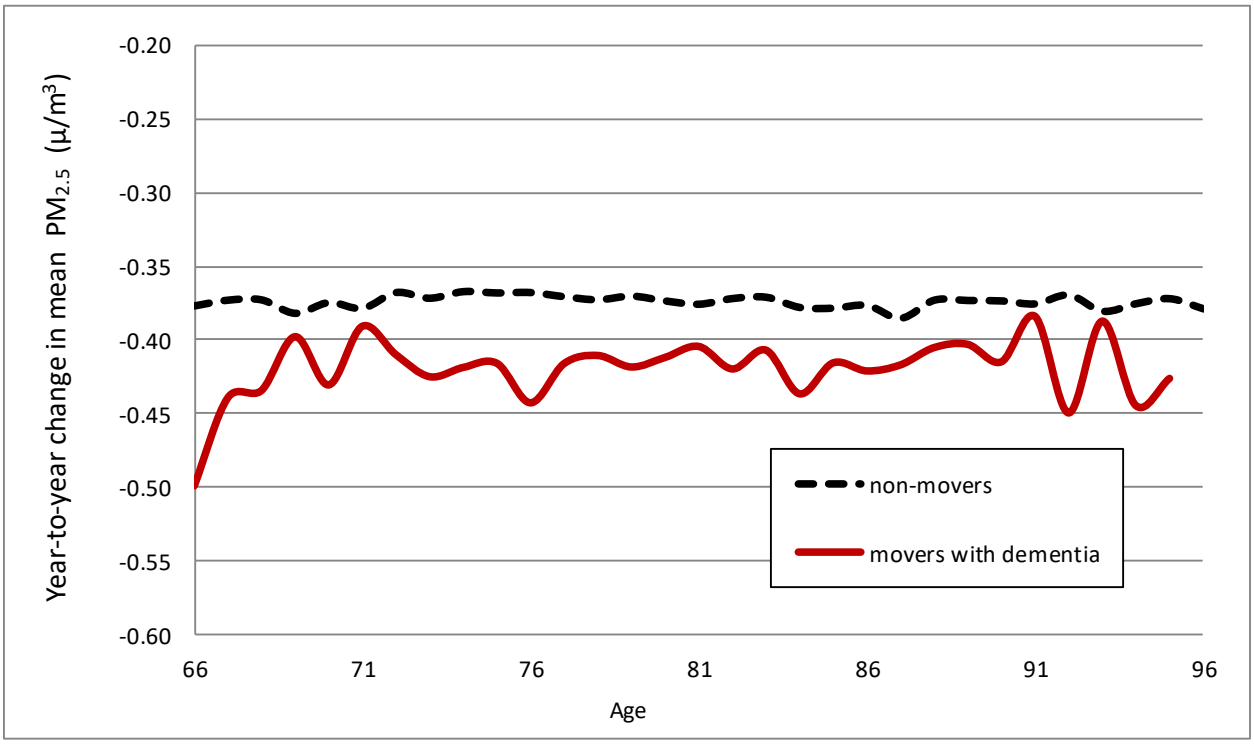
The figure reports conditional variation in decadal $PM_{2.5}$ exposures that arises from nonattainment status of the air quality monitor closest to the individual's residence, conditional on county nonattainment designation. Each solid line is constructed by using our first-stage coefficients on the 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.

TABLE A3—MODEL COEFFICIENTS FOR CRITERIA AIR POLLUTANTS

	$PM_{2.5}$ (1 μg/m^3)	PM_{10} (1 μg/m^3)	Ozone (parts per million)	Nitrogen Dioxide (parts per billion)	Sulfur Dioxide (parts per billion)	Carbon Monoxide (parts per million)
Estimated effect on dementia diagnosis probability	1.357***	-0.13	-74.80	0.13	0.44	22.21
	(0.62)	(0.26)	(194.53)	(0.26)	(0.96)	(13.89)
sample mean	10.947	21.300	0.043	0.383	13.301	2.522
sample standard deviation	1.701	4.199	0.004	0.070	4.097	1.062

The first row report coefficients for each of the federally regulated air pollutants included in the model summarized in Table II, column (7). Standard errors are shown below in the second row. The last two rows report the sample means and standard deviations in the distribution of decadal exposure among the estimation sample.

FIGURE A7: ANNUAL AVERAGE CHANGES IN $PM_{2.5}$ BY AGE, MIGRATORY STATUS, AND DEMENTIA



The figure shows the average year-to-year reduction in $PM_{2.5}$ experienced by movers with dementia (solid line) and non-movers (dashed line) in our sample, conditional on each year of life from age 66 to age 96. These reductions are calculated by subtracting pollution exposure in year *t+1* from pollution exposure in year *t* for each individual in each year and then averaging across all years in our sample for movers and non-movers at each age. Notice that the dashed line has a slope close to zero, implying that the reduction in air pollution exposure among non-movers is approximately uncorrelated with age. The average non-mover experienced an average year-to-year reduction in $PM_{2.5}$ concentrations of about 0.375 $\mu g/m^3$ at all ages. By contrast, the average 66 year-old mover with dementia experienced a reduction of about 0.5 $\mu g/m^3$. The difference between non-movers and movers with dementia persists with age.

TABLE A4—EFFECTS OF DEMENTIA ON PRESCRIPTION DRUG PLAN CHOICES

	Potential savings ($)	Probability of actively switching out of default plan	Probability chosen plan is dominated in:		
			cost, variance	cost, variance, star rating	cost, variance, insurer
dementia	19.42***	-0.36***	2.38***	3.70***	2.74***
	(1.99)	(0.07)	(0.09)	(0.11)	(0.13)
future dementia	6.97***	-0.58***	1.06***	1.37***	0.74***
	(1.93)	(0.07)	(0.09)	(0.10)	(0.12)
mean of dependent variable	346	10	72	52	25
sample size	2,755,892	2,575,534	2,755,892	2,755,892	1,742,957

Note: Each column reports coefficients estimates from models regressing decision making outcomes on indicators for whether the beneficiary is diagnosed with Alzheimer's disease and related dementias at the time of their enrollment decision (dementia) or after their enrollment decision but before the end of 2013 (future dementia) in comparison to beneficiaries not diagnosed with dementia by the end of the 2013. All models pool data from 2006 to 2010 and include residential CBSA dummies, state x year dummies, integer age x gender dummies, covariates describing individual health and Medicare expenditures prior to entering Medicare Part D, and covariates describing individual and neighborhood demographics. Robust standard errors are clustered by Census block group.

The table reports results from repeating estimation of the model in equation (5) after re-defining expected drug use in year *t* to be identical to actual drug use in year *t-1*. This reduces the sample sizes because we do not observe drug use prior to 2006.

SUPPLEMENTAL APPENDIX B: FOR ONLINE PUBLICATION

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 ADRD 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.

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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Spackman, D.E., S. Kadiyala, P.J. Neumann, D.L. Veenstra, and S.D. Sullivan. 2012. "Measuring Alzheimer Disease Progression with Transition Probabilities: Estimates from NACC-UDS." *Current Alzheimer Research,* 9(9): 1050-1058.