HAZED AND CONFUSED:
AIR POLLUTION, DEMENTIA, AND FINANCIAL DECISION MAKING

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We study whether long-term exposure to air pollution impairs cognition among the US Medicare population. We link fifteen years of administrative records for 7.4 million adults age 65 and older to the Environmental Protection Agency's air-quality monitoring network to track the evolution of individuals' health, onset of Alzheimer's disease and related forms of dementia, financial decisions, and cumulative exposure to fine-particulate air pollution ($PM_{2.5}$) based on their precise residential locations. We see evidence of Tiebout's mechanism at work: movers tend to move to less polluted neighborhoods but, among movers, those who are older and those with dementia tend to move to more polluted neighborhoods. We develop several strategies to address residential sorting, including utilizing quasi-random variation in $PM_{2.5}$ exposures stemming from the EPA's initial (2005) designation of nonattainment counties for $PM_{2.5}$. Across a range of models and identification strategies, we find that a 1 microgram per cubic meter ($\mu g/m^3$) increase in decadal exposure to $PM_{2.5}$ (8.5% of the mean) increases the prevalence of dementia by 1% to 3%. We also find that higher cumulative exposures to $PM_{2.5}$ impair financial decision making among those not diagnosed with dementia, where the magnitudes of the effects are 4% to 6% of the effect of dementia on decision making. Finally, we find no evidence that exposure to $PM_{2.5}$ affects the diagnosis rates for morbidities thought to be unrelated to air-pollution and no evidence that pollutants other than $PM_{2.5}$ impair cognition, providing evidence against confounding.

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Air pollution degrades human capital. Economists have demonstrated that short-term exposures to elevated pollution during childhood constrain the production of human capital as observed from increased school absences (Currie et al. 2009) and decreased scores on high-stakes exams (Ebenstein, Lavy and Roth 2016). Among adults, higher daily levels of air pollution reduce contemporaneous measures of labor productivity in both cognitive and manual tasks (Graff-Zivin and Neidell 2012; Chang et al. 2016a,b). These short-term exposures can have long-lasting implications: higher exposures during the fetal period and early infancy have been shown to decrease wages and labor-force participation in adulthood (Sanders 2012; Isen, Rossin-Slater, and Walker 2017). By contrast, little is known about the effects of long-term exposure on cognition among older adults. Medical and epidemiological studies point to potential pathways through which air pollution may impair cognition, especially in the case of fine particulate matter ($PM_{2.5}$)—airborne particulates smaller than 2.5 microns in diameter. The small size of $PM_{2.5}$ allows it to remain airborne longer, to penetrate buildings, to be inhaled easier, and to reach and accumulate within brain tissue.[1] Accumulation over time can cause neuroinflammation, leading to symptoms of dementia (Block et al. 2012; Wilker et al. 2015; Cacciottolo et al. 2017; Underwood 2017). While suggestive, this medical evidence comes primarily from animal studies or small non-representative human cohorts. The degree to which long-term exposure to air pollution systematically impairs cognition among people age 65 and above remains unknown.

This article is the first nationally representative longitudinal study of how long-term exposure to air pollution affects cognitive impairment among people age 65 and above. We assembled fifteen years of administrative Medicare records on 7.4 million people, tracking their health, demographics, residential exposures to air pollutants, and financial decisions. These data are well-suited to studying how pollution affects cognitive impairment. First, adults over 65 are the wealthiest and the fastest-growing age group in the United States. Second, the data include two important measures of their cognitive impairment—the diagnosis of dementia, and the outcomes of their financial decisions in

[1] A recent study published in the Proceedings of the National Academy of Sciences of the United States has found accumulated particulate matter in brain samples from thirty-seven autopsied individuals (Maher et al. (2016)).

high-stakes markets. Dementia is prevalent among this age group, impairing their "memory, thinking, orientation, comprehension, calculation, learning capacity, language and judgement…emotional control [and] social behavior", thereby disrupting people's relationships and limiting their abilities to perform basic activities of daily living.[2] About one percent of Americans are diagnosed with Alzheimer's disease and related forms of dementia in their early sixties, and the share roughly doubles with each additional five years of age; for Americans aged eighty-five and above, roughly one-third have received a dementia diagnosis (Hirtz et al. 2007, Querfurth and LaFerla 2010). Alzheimer's disease specifically is the sixth leading cause of death in the United States, and in 2016 alone it accounted for $240 billion in direct expenditures on healthcare services and 18 billion hours of labor by unpaid caregivers (Alzheimer's Association 2017).[3] Furthermore, this age group faces a host of complex and important financial decisions, such as health insurance choices and retirement planning, which economists have often used to evaluate the quality of financial decision making (see Keane and Thorp (2016) for a summary).[4]

Our empirical research designs leverage the strengths of these data to mitigate potential sources of bias and strengthen our ability to draw causal inferences. The data allow us to track each person's residential address from 1999 through 2013 including the timing and location of any moves. We link these data to the US Environmental Protection Agency's (EPA's) national network of air quality monitors to construct individual-specific, long-term measures of cumulative exposures to $PM_{2.5}$ and five other federally regulated air pollutants (particulate matter smaller than 10 microns, ozone, carbon monoxide, nitrogen dioxide, and sulfur dioxide). The Medicare data also provide many individual characteristics, including the diagnosis dates of dementia and a wide range of other illnesses. In

[2] The ICD-10 defines dementia (F00-F03) as "a syndrome due to disease of the brain, usually of a chronic or progressive nature, in which there is disturbance of multiple higher cortical functions, including memory, thinking, orientation, comprehension, calculation, learning capacity, language and judgement. Consciousness is not clouded. The impairments of cognitive function are commonly accompanied, and occasionally preceded, by deterioration in emotional control, social behavior, or motivation. This syndrome occurs in Alzheimer disease, in cerebrovascular disease, and in other conditions primarily or secondarily affecting the brain."

[3] 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".

[4] The US Census Bureau (2017) projects that the share of Americans over age 65 will increase from 15% in 2014 to 21% by 2030 and 24% by 2060.

addition to serving as controls, these other diagnoses allow us to design placebo tests for illnesses that may be related to socioeconomic status and location decisions, but are not *a priori* suspected to be caused by air pollution. These placebo tests are designed to assess whether our research design overcomes many of the well-known threats to identifying how air pollution affects people, such as residential sorting which could bias our estimates away from zero if people who were healthier, wealthier, and better-educated systematically migrated to less-polluted areas based on characteristics that may mitigate health shocks or improve economic outcomes (Kuminoff, Smith, and Timmins 2013, Dominici, Greenstone, and Sunstein 2014).

We address residential sorting and other potential sources of confounding through two complementary econometric designs. First, we adapt Chay and Greenstone's (2005) instrumental variables approach to utilize exogenous variation in $PM_{2.5}$ concentrations caused by a strengthening of Clean Air Act regulations in 2005. We show that the new regulations lowered $PM_{2.5}$ in relatively polluted areas that the EPA designated as being out of attainment with a new federal standard on maximum allowable $PM_{2.5}$ concentrations. We leverage the difference in regulatory intensity between attainment and nonattainment counties as an exogenous source of variation in their residents' decadal exposures to $PM_{2.5}$ conditional on a large set of covariates and baseline conditions. We find that a 1 microgram per cubic meter ($\mu g/m^3$) (8%) decrease in ten-year annual average hourly concentrations of $PM_{2.5}$ led to a 0.4 percentage point (3%) reduction in the prevalence of dementia between 2004 and 2013. We show that this result is stable across a range of model specifications and that the effects are driven by $PM_{2.5}$, rather than other pollutants.

Second, we rely on within-county variation across individuals in residential exposure to $PM_{2.5}$ and other pollutants to identify their effects on dementia and financial decision making. We focus on individuals who were not diagnosed with dementia at the start of the decade and analyze how pollution exposure affects their probability of being diagnosed with dementia over the decade. Our specifications include county fixed effects and numerous individual-specific covariates, including baseline medical expenditures and

health, race, and flexible spline functions in age-by-gender, along with covariates describing socioeconomic characteristics of the individual's neighborhood including Census block group measures of educational attainment, income, and the housing stock. Thus, for those who do not change residences, the effects of $PM_{2.5}$ are identified by within-county heterogeneity in long-term cumulative air-pollution exposures. This heterogeneity arises from local variation in emissions, wind patterns, and geography. For those who change residences, we observe an additional source of variation in pollution exposure. Based on these sources of variation in $PM_{2.5}$, a variety of econometric specifications suggest that higher cumulative exposures increase an individual's probability of being diagnosed with dementia. Among those who are still alive at the end of the decade, a 1 $\mu g/m^3$ increase in average decadal exposure increases the diagnosis probability by 1%. We show that this result is stable over time and driven by cumulative, rather than short-term, exposure. Further, the effect is observed for $PM_{2.5}$ alone, not other federally regulated air pollutants. This non-finding, coupled with evidence from the placebo tests that $PM_{2.5}$ appears not to affect medical diagnoses thought to be unrelated to air pollution, reinforce the causal interpretation of our findings for the effects of $PM_{2.5}$ on dementia.

We find similar evidence for the impact of $PM_{2.5}$ on financial decision making. As expected, we find that dementia impairs financial decision making. However, even among those individuals not diagnosed with dementia, long-term cumulative exposure to $PM_{2.5}$ negatively impacts a range of measures that prior studies used to assess the quality of consumers' choices in markets for prescription drug insurance plans (Keane and Thorp 2016). In particular, a 1 $\mu g/m^3$ increase in average decadal exposure to $PM_{2.5}$ is shown to increase potential savings by $4 (a 1% increase relative to the mean) and increase the probability of choosing a plan that is dominated by another in terms of cost, risk protection, and quality by 0.25 to 0.43 percentage points (a 0.5% to 1.2% increase relative to the mean). These effects are 3% to 6% of the size of the effects that a dementia diagnosis has on the same outcomes.

I. Air Pollution, Human Capital, and Cognitive Impairment

Air pollution reduces the aggregate stock of human capital by increasing morbidity

and mortality (Chay and Greenstone (2003), Currie and Neidell (2005), Currie and Walker (2011), Neidell (2004, 2009), Dominici, Greenstone, and Sunstein (2014), Schlenker and Walker (2016), Deryungina et al. (2016), Deschenes, Greenstone and Shapiro (2017)).[5] Air pollution degrades human capital even apart from its effects on specific medical diagnoses and death. Higher exposure to air pollution during childhood, beginning in utero, constrains the production of human capital via increased school absences and lower scores on standardized exams (Ransom and Pope (1992), Currie et al. (2009), Sanders (2012), Ebenstein, Lavy, and Roth (2016)). These negative effects persist to adulthood. Isen, Rossin-Slater, and Walker (2017) demonstrate that, all else constant, people who were exposed to more air pollution in their birth year have lower earnings and lower labor force participation at age 30. Conditional on participation, hourly and daily spikes in pollution have also been shown to reduce workers' labor productivity in both manual and cognitive tasks (Graff-Zivin and Neidell (2012), Chang et al. (2016a,b)). Complementary evidence from laboratory experiments suggests that adults perform worse on tests of cognition on days with higher pollutant concentrations (Chen et al. (2017b)). Among the various pollutants, $PM_{2.5}$ is believed to pose the greatest threat to contemporaneous cognition, in part because it easily penetrates buildings and pollutes indoor air (Graff-Zivin and Neidell (2013), Chang et al. (2016b)).

In contrast, little is known about the extent to which long term exposure to air pollution affects cognitive impairment outside of suggestive evidence from medical studies. Studies tracking small, specialized cohorts across time and space have found that higher cumulative exposure to air pollution over multiple years is positively associated with the prevalence of dementia.[6] This evidence is complemented by clinical studies suggesting

[5] See Graff-Zivin and Neidell (2013) for a review of the literature.

[6] Wu et al (2015) found associations between 3 years of exposure to particulate matter between 2.5 and 10 microns (PM_{10}) and ozone and diagnosis of dementia using a case-control study of several hundred northern Taiwanese people. Weuve et al (2014) found associations between long term exposure to $PM_{2.5}$ and PM_{10} and cognitive decline in a cohort study of several hundred American female nurses ages 70-81. 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. Chen et al (2017b) relied on a panel survey in China to find that greater current and cumulative pollution exposure to an index measure of sulfur dioxide (SO2), nitrogen dioxide (NO2), and PM10 was associated with declines in survey-based cognitive test scores. However, like prior studies these two most recent articles were unable to directly observe individuals' exposures to individual pollutants, health behaviors, socioeconomic status and long-term migration patterns. As a result, prior work has had limited ability to account for confounding factors such as low wealth, education or general health that might result in individuals at higher risk for dementia choosing to live in more polluted areas.

multiple pathways by which air pollution, and fine particulates specifically, may impair cognition (Chen and Schwartz (2009), Block et al. (2012), Weuve et al. (2012), Wilker et al. (2015), Wu et al. (2015), Allen et al. (2016), Cacciottolo et al. (2017)). First, pollution exposure is linked to increased risk for strokes among older adults (e.g., Wellenius et al. (2012)), which cause vascular dementia (Ng, Turek, and Hakim (2013)). Second, post-mortem analysis has detected $PM_{2.5}$ in human brains. 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)). Additionally, controlled exposure of mice to air pollution in laboratory experiments results in neuroinflammation (Block et al. (2012)). These pathways suggest that prolonged exposure to elevated levels of pollution may trigger and/or accelerate dementia (Underwood (2017)).

Furthermore, economic research points to several pathways through which cognitive impairment may negatively affect financial decision making. First, cognitive impairment can affect decision making by increasing the cost of cognitive processing which is required to make decisions, or even render them impossible apart without assistance from others, which may introduce agency problems. Agarwal et al. (2009) document that as people age they tend to leave more money on the table in credit markets while Keane et al. (2017) find that Medicare beneficiaries diagnosed with dementia are less likely to comprehend key institutional features of insurance markets and more likely to make decisions that appear to be suboptimal. 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 one of the key sources of heterogeneity in financial decision making; old age, life expectancy, and diagnosis of chronic conditions specifically increase discount rates (Huffman, Maurer, and Mitchell (2016), Oster, Shoulson, and Dorsey (2013)). Fourth, because cognitive impairment can reduce life expectancy (or quality-adjusted life expectancy) it may reduce investments in health capital, leading to additional chronic conditions that increase the complexity of decisions about health insurance (Fang et al. 2007).

II. Data

We used administrative records from the Centers for Medicare and Medicaid Services (CMS) to develop a national longitudinal database linking people's residential address histories, medical claims, demographic characteristics, and enrollment decisions for prescription drug insurance plans. We start with a random 10% sample from the universe of Medicare beneficiaries aged 65 and over between 1999 and 2013 (about 12.5 million people). We then obtain an independent, random 20% sample from the universe of beneficiaries aged 65 and over who purchase standalone prescription drug insurance plans (PDPs) through Medicare Part D at any point between 2006 and 2010 without the aid of low-income subsidies (about 2 million people).[7] The union of these samples contains 13.6 million people.

We drop two groups of people due to data limitations. First, we drop 2.7 million people who have an unidentifiable residential location at any point between 1999 and 2013.[8] Second, we drop 3.6 million 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. These two exclusions seem unlikely to compromise external validity. Appendix Tables A1 and A2 report summary statistics for our estimation sample and the excluded subsets. The excluded individuals are generally 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 final estimation sample consists of 7.4 million individuals who we observe for 61.9 million person-years.[9] Administrative records identify 44% of these individuals as male and 83% as white. The mean age upon entering our dataset 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, typically when

[7] We exclude those receiving low-income subsidies because they are autoenrolled into PDPs. 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 quality in the Medicare Part D PDP markets have excluded those receiving low-income subsidies.
[8] This includes addresses that are post-office boxes.
[9] In comparison, there were 40.3 million individuals age 65 and over in the United States in 2010.

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 69% of individuals survive through the end of 2013. For those who die, the mean age at death is 81.

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 medical conditions. For the subset that choose to enroll in prescription-drug insurance plans (PDP) through the Medicare Part D markets (1.2 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 together with the cost calculator developed by Ketcham, Lucarelli and Powers (2015) to construct a series of metrics that have been used in prior literature to assess the quality of older adults' financial decisions. These metrics are described in Section VI.

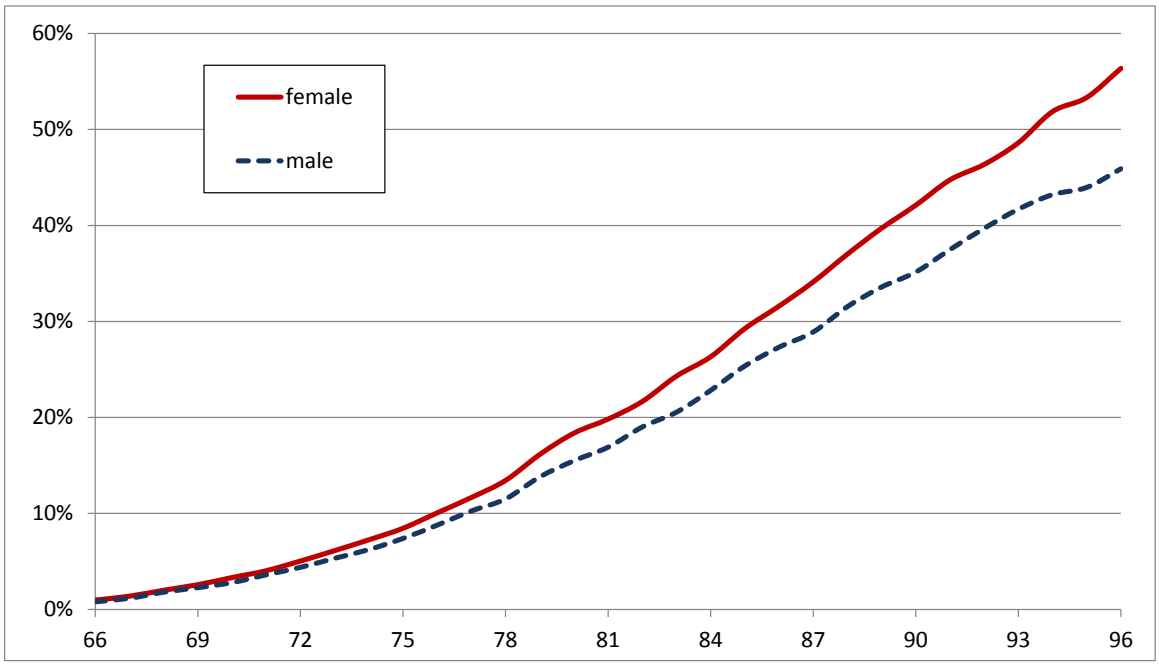
A. Clinical Measures of Dementia and Known Risk Factors

For each person who receives a dementia diagnosis, we observe the initial diagnosis date on record in CMS's Chronic Conditions Data Warehouse file. This file tracks if and when each individual is diagnosed with a specific chronic medical condition, based on the presence of diagnosis codes on insurance claims. A diagnosis of dementia 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. The leading cause of dementia is Alzheimer's disease, which accounts for between 60% and 80% of all cases according to the Alzheimer's Association (2017). The next most common cause is a stroke (which causes vascular dementia), accounting for about 10% of cases.

Twenty-three percent of people in our sample are diagnosed with dementia by the end of 2013. Figure 1 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 at some point.[10] The diagnosis rate is higher for women and this gender gap widens with age.

FIGURE 1: DEMENTIA BY AGE AND GENDER IN 2013



According to the Alzheimer's Association (2017) physical risk factors for dementia include chronic medical conditions that can reduce the flow of blood and oxygen to the brain.[11] Appendix Table A1 shows that most individuals in our data are diagnosed with at least one of these risk factors by the end of the study period: stroke (19%), diabetes (32%), congestive heart failure (36%), ischemic heart disease (48%) and hypertension (71%).[12] Factors believed to reduce the risk of dementia include greater education, better nutrition and physical health, 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

[10] Within our sample, diagnosis rates are slightly higher for those in Medicare Part D without a low-income subsidy. For comparison, Figure A1 reports diagnosis rates separately for men and women conditional on enrollment status in Medicare Part D in 2010.
[11] Genetics are also believed to play a role.
[12] Appendix Table A1 provides summary statistics for additional chronic medical conditions.

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.

B. Using Residential Location Histories to Measure Long Term Pollution Exposure

CMS uses information from the US Social Security Administration to track Medicare beneficiaries' residential locations. We obtain ZIP+4 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—about one for every four households.

Migration patterns for the individuals in our sample are similar to those reported by the Census Bureau for individuals aged 65 and older. More than 80% of individuals live in the same ZIP+4 (and presumably the same address) throughout our study period. Of the 18% of people who move at least once, 10% move between counties and 5% moved between states.[13] We use this information to measure each individual's cumulative exposure to air pollution, accounting for migration.[14]

Individuals in our sample live in 9.8 million 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 particulate matter smaller than 10 microns, ozone, carbon monoxide, nitrogen dioxide and sulfur dioxide. Annual data on ambient pollution levels are drawn from EPA's air quality system, consisting of an unbalanced panel of 6,679 monitors in operation between 1999 and 2013.[15] To approximate annual average exposure in

[13] Among the ever-movers, 73% moved once during our study period, 19% moved twice, 5% moved three times and 2% moved four or more times.

[14] We are unable to observe seasonal migration by people with more than one residence (e.g. snowbirds) because we only observe the residential address on record with the Social Security Administration and CMS for administrative purposes. Fortunately, the scope for measurement error appears to be 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.

[15] 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 $PM_{2.5}$. EPA also regulates lead as a criteria air pollutant but had far fewer monitors (477) during our study period.

each ZIP+4 Code, we use the latitude and longitude coordinates of these monitors along with the coordinates of the ZIP+4.[16] Specifically, we use the Great Circle algorithm to calculate the surface distance from each ZIP+4 centroid to each monitor.[17] 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. 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 cumulative exposure histories for each person in our data.

III. New Facts About Heterogeneity in Dementia, Migration, and Pollution

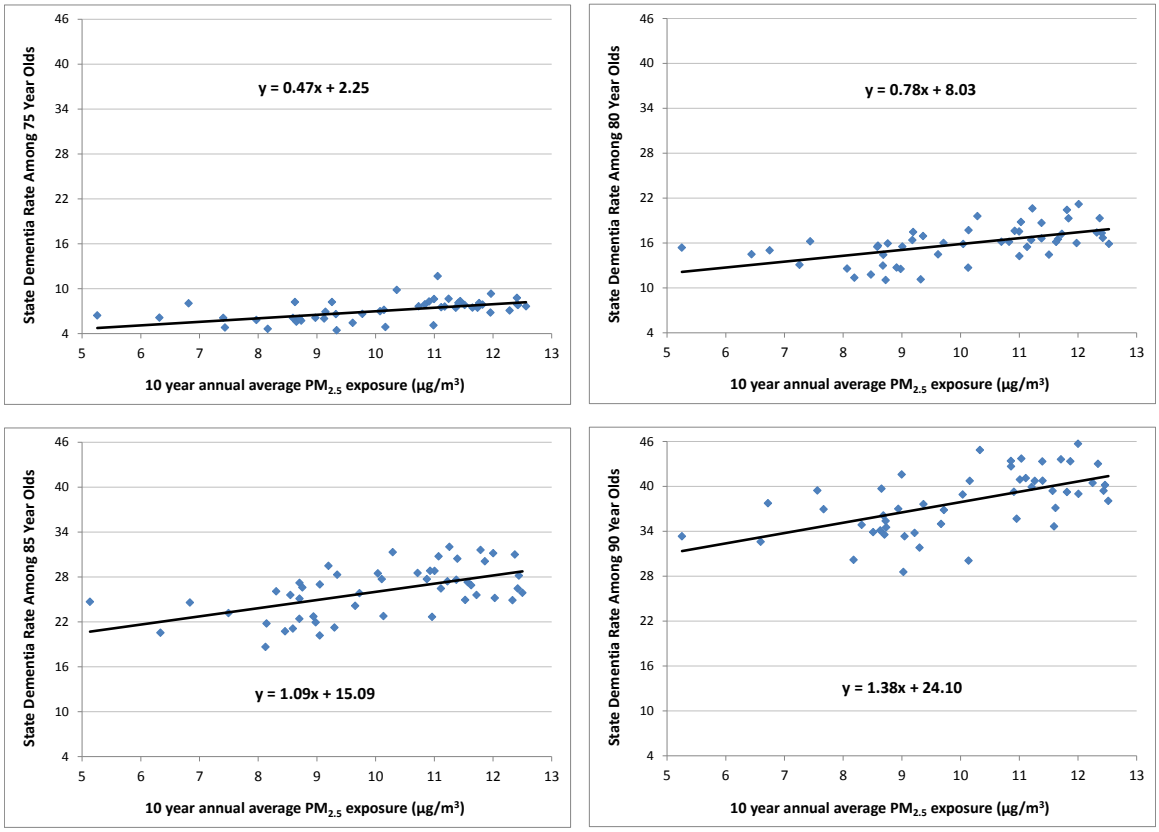
Figure 2 provides initial evidence on the spatial relationship between the prevalence of dementia and long-term exposure to $PM_{2.5}$. This figure plots the 2013 state-level, age-specific dementia rate against annual average exposures to $PM_{2.5}$ from 2004 through 2013. Specifically, the plots are constructed using all of the 75-, 80-, 85- and 90-year olds in our data in each state.[18] At each age, the prevalence of dementia across states varies substantially and is strongly, positively correlated with $PM_{2.5}$. The strength of this correlation increases with age. For example, the trend line for 75-year olds indicates that a 1 $\mu g/m^3$ increase in annual average $PM_{2.5}$ exposure between the ages of 64 and 75 is associated with a 0.47 percentage point increase in a state's dementia rate. This is equivalent to a 6.1% increase relative to the national average among 75-year olds. The same 1 $\mu g/m^3$ increase in exposure from age 81 to 90 is associated with a 1.38 percentage point increase (3.5% of the national average). In the remainder of this article, we leverage our data to determine the extent to which the associations in Figure 2 are causal versus a statistical artifact of sorting and spatiotemporal heterogeneity.

[16] 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.

[17] 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.

[18] The median state has 1,990 75-year olds, 1,406 80-year olds, 1,080 85-year olds and 635 90-year olds. While their measures of decadal $PM_{2.5}$ exposure include changes due to migration between states, such moves are rare. As noted earlier, only 5% of all people in our data ever move between states.

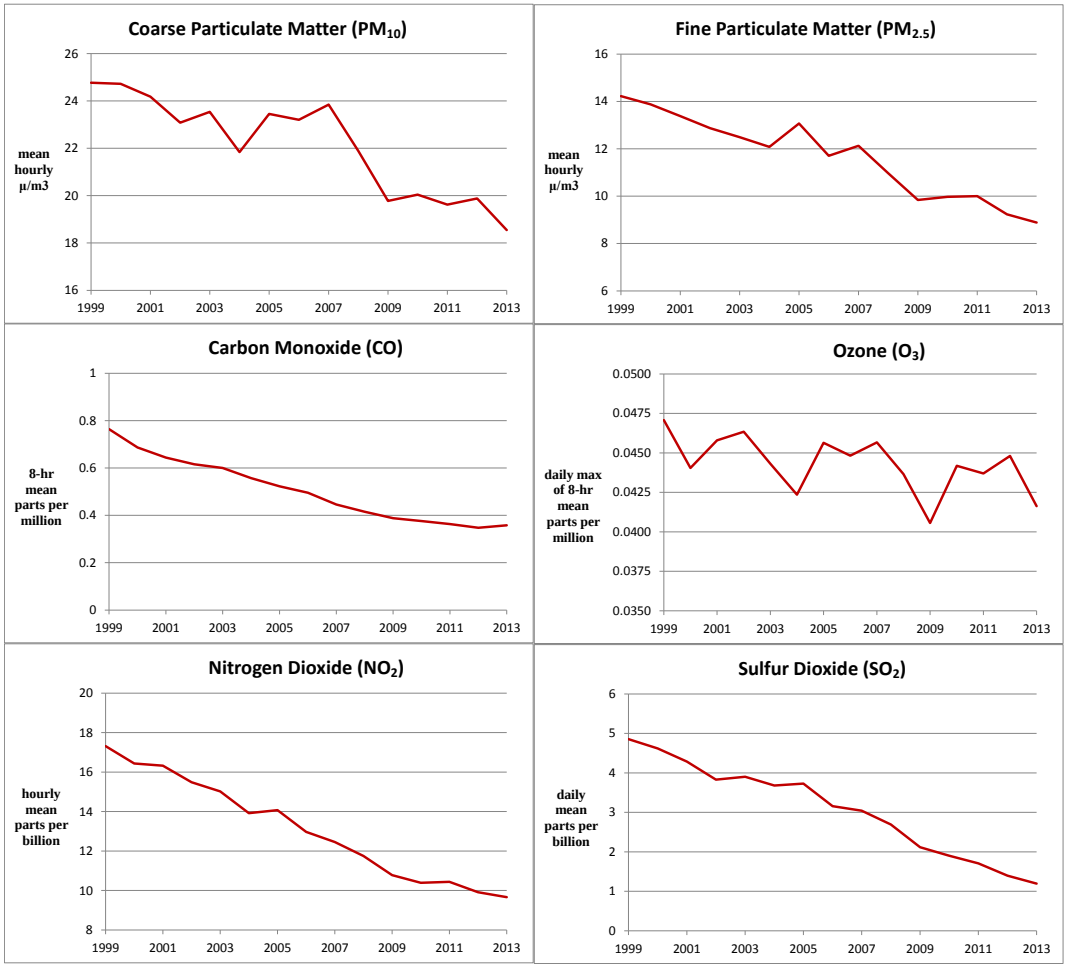
FIGURE 2: SPATIAL CORRELATION BETWEEN $PM_{2.5}$ AND DEMENTIA IN 2013, BY AGE



Note: Each data point represents the fraction of individuals living in a given state who had been diagnosed with dementia before the end of 2013. The figures are conditional on age: 75 (top left), 80 (top right), 85 (bottom left) and 90 (top right).

Our data also show that average exposure to air pollution among the US Medicare population declined substantially over time. For example, in 2013 the average person in our data was exposed to average $PM_{2.5}$ concentrations of 9 $\mu g/m^3$, down from 14 $\mu g/m^3$ in 1999. Figure 3 shows similarly large declines in other air pollutants. Factors that may have contributed to these declines include increased regulation of emissions (Shapiro and Walker 2016), substitution from coal to shale gas in electricity generation, and migration to less-polluted neighborhoods.

FIGURE 3: SAMPLE ANNUAL AVERAGE AIR POLLUTION EXPOSURES

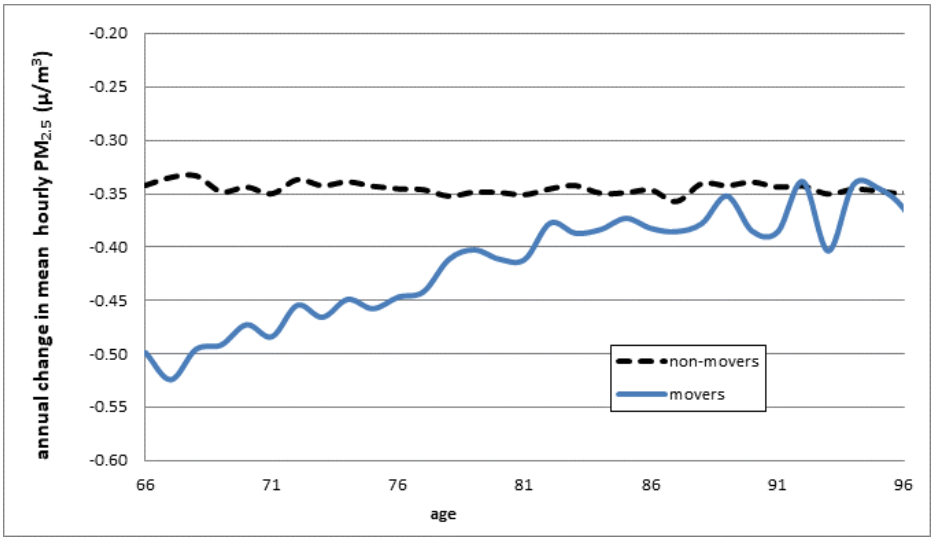


Note: The figures report annual average exposure to federally regulated air pollutants among our sample of Medicare beneficiaries in each year.

Migration plays a significant role in explaining these reductions because on average, movers go to cleaner areas. The top panel of Figure 4 shows the annual average reduction in $PM_{2.5}$ experienced by movers (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 annual average reduction in $PM_{2.5}$

concentrations of about 0.35 μg/m^3 at all ages. By contrast, the average 65 year-old mover experienced a reduction of about 0.5 μg/m^3. The difference between movers and non-movers narrows with age. One potential explanation for this trend is that the demand for cleaner air declines with health shocks, as decreased health induces relocation based on access to medical care and family caregivers (rather than local amenities such as air quality).

FIGURE 4: ANNUAL AVERAGE CHANGE IN $PM_{2.5}$ FOR MOVERS AND NON-MOVERS BY AGE



The bottom panel of Figure 4 subdivides movers into those who had been diagnosed

with dementia at the time they moved and those who had not. While splitting the move-by-age sample reduces statistical precision, movers with dementia tend to experience smaller improvements in air quality compared to movers without dementia, at least through their mid-80s. This may signal that dementia patients' location decisions are driven more by access to caregivers. Moreover, the fact that, conditional on moving, dementia patients tend to move to more polluted areas highlights the potential for residential sorting and life-cycle dynamics to confound econometric models.[19]

IV. Evidence on Dementia from a County-Level Instrumental Variables Design

In our first econometric specification, we recover the causal impact of $PM_{2.5}$ exposure on cognitive impairment by leveraging a change in the federal Clean Air Act regulations in the spirit of Chay and Greenstone (2005). Specifically, we recover a set of 2SLS estimates for the effect of changes in residential $PM_{2.5}$ exposure on changes in county-level dementia rates. We use the EPA's 2005 designation of counties as being in or out of attainment of a new federal $PM_{2.5}$ standard to develop an instrument for county level changes in $PM_{2.5}$ between 2004 and 2013.

A. Modeling the Effect of $PM_{2.5}$ on County-level Dementia Rates

We begin by specifying a linear approximation to the relationship between county-level changes in dementia rates and their residents' cumulative exposure to $PM_{2.5}$:

$$(1) \quad \Delta y_{jr} = \alpha \Delta \mathrm{PM2.5}_{jr} + \beta X_{jr,2004} + \gamma \Delta X_{jr} + \theta \Delta C_{jr} + \delta_r + \Delta \epsilon_{jr}.$$

where Δy_{jr} denotes the change between 2004 and 2013 in the fraction of older adults living with a dementia diagnosis in county j in region r, and $\Delta PM2.5_{jr}$ denotes the decadal change in the average resident's cumulative exposure to fine particulate matter. Based on medical literature, one would expect changes in dementia rates to vary across counties as

[19] The potential for confounding begins in childhood. Children who grow up in poorer areas are more likely to attend worse performing schools and receive lower wages as adults (Chetty et al. 2014). They also tend to be exposed to more $PM_{2.5}$ while their brains are developing, which may reduce their health, wealth and human capital as adults (Graff-Zivin and Neidell 2013, Isen, Rossin-Slater and Walker 2016).

a function of the average resident's baseline health and other demographics, $X_{jr,2004}$. For the same reason, changes in dementia rates may vary with changes in the demographic characteristics of counties' average residents, ΔX_{jr}, due to aging, migration and death. Changes in the local supply of medical care, ΔC_{jr}, may also affect diagnosis rates. Finally, we include a set of dummy variables, δ_r, to allow region-specific trends in county-level diagnosis rates.

We define $\Delta \text{PM2.5}_{jr}$ as the decadal change in cumulative exposure to fine particulate matter, measured as the difference between annual average exposure during 2004-2013 and pre-decadal exposure, measured by a three-year average from 2001 to 2003.[20]

$$(2) \qquad \Delta \text{PM2.5}_{jr} = \sum_{t=04}^{13} \frac{PM2.5_{jr,t}}{10} - \sum_{s=01}^{03} \frac{PM2.5_{jr,s}}{3}.$$

Our coefficient of interest, α, measures how a marginal increase in 10-year annual average exposure to $PM_{2.5}$ affects a county's dementia rate. This effect is measured conditional on the average health, healthcare spending and other demographics of its older adult population at the start of the decade; decadal changes in the demographic composition of its older adult population due to migration and death; changes in access to health care; and region indicators used to absorb unobserved variables that may be correlated with regional trends in both pollution and dementia. Further, measuring the dependent variables in changes allows us to control for any time-invariant unobservables at the county level that may lead to persistently higher dementia rates in some counties, e.g. pre-period pollution exposure, local climate, or the supply of retirement communities or long-term care facilities which may attract migrants who are at higher risk of dementia.

A concern is that the OLS estimation of (1) could be biased by measurement error in $\Delta \text{PM2.5}_{jr}$ and/or by correlation between $\Delta \text{PM2.5}_{jr}$ and $\Delta \epsilon_{jr}$. We address this concern by

[20] The 2001-2003 monitoring period is a natural choice for measuring baseline exposure because, as we explain below, 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. As explained below, we control for compositional changes in counties' average residents over the decade, including their pre-period pollution exposures, using several measures of health and other demographics. We cannot directly observe pollution exposure prior to 1999, the year in which EPA established a national network of air pollution monitors for $PM_{2.5}$.

implementing a 2SLS version of the model that uses an instrument to isolate the exogenous variation in decadal $PM_{2.5}$ exposure. The first stage is given by:

$$(3) \qquad \Delta \text{PM2.5}_{jr} = \pi Z_{js,2005} + \beta X_{jr,04} + \gamma \Delta X_{jr} + \theta \Delta C_{jr} + \delta_r + \Delta \epsilon_{jr}$$

where the instrumental variable, $Z_{jr,2005}$, is an indicator equal to 1 if the EPA classified county *j* in region *r* as being in "nonattainment" for $PM_{2.5}$ in 2005. Chay and Greenstone (2005) first illustrated how the EPA's county-level nonattainment designations could serve as valid instruments for changes in federally-regulated air pollutants. Our approach focuses on a hitherto unexploited strengthening of the EPA's air-quality regulation: its initial, 2005 designation of nonattainment counties for $PM_{2.5}$.[21]

B. County Nonattainment Designation as an Instrumental Variable for the Change in Cumulative Exposure to $PM_{2.5}$

A signature feature of the Clean Air Act Amendments was the establishment of national ambient air quality 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 "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 regulation. These threats spur local regulation, leading to relatively large air quality improvements in the regulated counties.

Among the criteria pollutants, particulate matter is believed to have the most pernicious effects on human health at commonly observed concentrations (EPA 2011). During the 1970s and 1980s, the EPA regulated total suspended particulates (TSP). Evidence that the negative health effects were being driven by the smallest of these particulates led the EPA to replace the standard on TSP with new standards on PM_{10} in 1987 and new stand-

[21] Because the rationale for using county nonattainment status as an instrument for changes in air pollution has been thoroughly explained by prior studies, we keep our review of the institutional details brief. Readers seeking additional background on federal regulation of air pollutants should see the discussions in Chay and Greenstone (2005), Walker (2013) and U.S. EPA (2005, 2016).

ards on $PM_{2.5}$ in 1997. Enactment of each new standard was followed by new, county-level nonattainment designations. By spurring local regulation, 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-level TSP attainment status 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 change in the EPA's county-level attainment designations by focusing on the 1997 $PM_{2.5}$ standard.

In 1997, the EPA established new 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-level attainment designations in 2005 which were based on average monitor readings over the period 2001 to 2003.[22] Two hundred and eight counties containing approximately 90 million people (30% of the U.S. population) were classified as living in a nonattainment county at that time. Remaining counties were classified as "attainment/unclassifiable" because they either met the standard or were lacking the necessary information to make a designation (US EPA 2005). We will henceforth denote these counties as the attainment group. States were directed to take actions to ensure that nonattainment counties met the 15.05 $\mu g/m^3$ standard by 2010.[23]

Figure 5 provides evidence that $PM_{2.5}$ concentrations near air-quality monitors changed following the 2005 designations. The solid and dashed lines show the trend in $PM_{2.5}$ readings, averaged over all air-quality monitors in each county in each year by their

[22] Nonattainment designations at a given monitor were based on a 3-year average from 2001-2003 of annual averages over quarterly averages over daily averages over hourly average monitor readings. Nonattainment counties were classified as partly or wholly nonattainment. EPA's air quality system includes annual data on monitors in 132 of the 208 nonattainment counties. Counties need not have air quality monitors to be classified as nonattainment. Counties without monitors were classified as nonattainment if they were believed to contribute to violations in nearby counties with monitors (Federal Register 2005).

[23] 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. 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. Appendix Figure A3 provides a timeline of major regulatory changes.

respective attainment status.[24] Prior to 2005, $PM_{2.5}$ concentrations were declining at a similar rate in both attainment and nonattainment counties. The dotted line shows that the difference between the two trend lines was fairly stable from 1999 to 2005. After the nonattainment designations were made, concentrations of $PM_{2.5}$ declined at a faster rate in nonattainment counties. The difference between the average monitor readings in nonattainment and attainment counties dropped by more than half between 2005 to 2013. Figure 5 mirrors Chay and Greenstone's (2005, Figure 2) analysis of the 1975 attainment designations for TSP. Like them, we use an indicator for a county's attainment status as an instrument for the change in concentrations of the regulated air pollutant observed in the county over the following decade.[25]

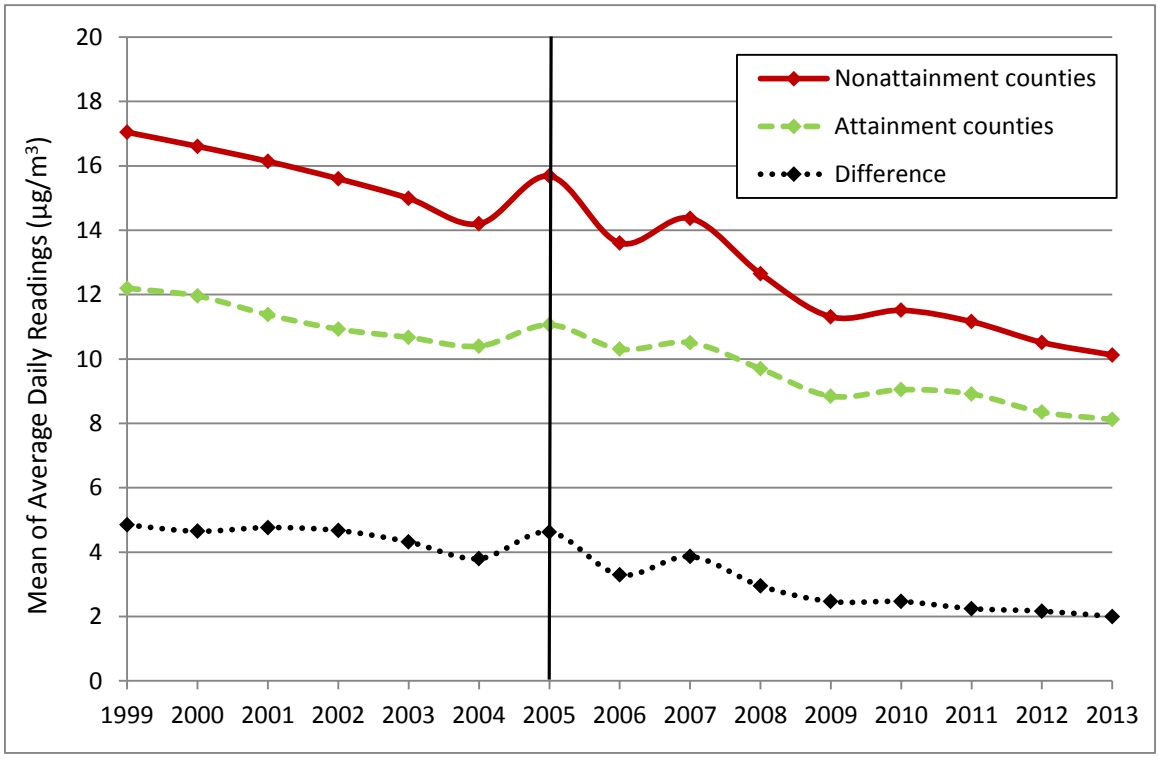
Figure 6 shows how county-level attainment status varies with the $PM_{2.5}$ exposure of the average individual in our Medicare sample over the period 2001 to 2003. To construct this figure, we first calculate the inverse-distance-weighted annual measures of residential $PM_{2.5}$ exposure for each individual in our sample. We then average over all individuals in each county-year and group counties into 0.3 $\mu g/m^3$ bins based on average exposure in 2001-2003. The left panel of Figure 6 shows the fraction of counties in each bin that the EPA designated as being in nonattainment and is constructed using data on 644 counties with air quality monitors in operation from 2001 to 2003.[26] No county with concentrations below 12.31 $\mu g/m^3$ was designated as nonattainment; every county with concentrations above 15.16 $\mu g/m^3$ was designated as nonattainment; and as a county's average concentrations increased from 12.31 to 15.16 $\mu g/m^3$, so too did its probability of receiving a nonattainment designation. The right panel of Figure 6 shows the CDFs of average exposure for people in attainment and nonattainment counties with and without air quality monitors.

[24] To construct the figure we started by extracting records from EPA's air quality system for all monitors that satisfied EPA's monitoring standards for regulatory decisions, had at least 10 reading per year, and did not exclude events such as forest fires. This yielded an unbalanced panel of between 880 and 1,148 monitors each year. Appendix Figure A4 shows that the figure looks virtually identical if we construct it from a balanced panel of 393 monitors that were in continuous operation from 1999 through 2013.

[25] Chay and Greenstone's preferred version of this instrument was an indicator for mid-decade attainment status. This was partly because, in their context, using mid-decade attainment status limited the scope for unobserved spatial sorting by households to complicate interpretation of results. By contrast, we are able to observe and control for migration at the individual level, as well as run robustness checks that limit the estimation sample to non-movers.

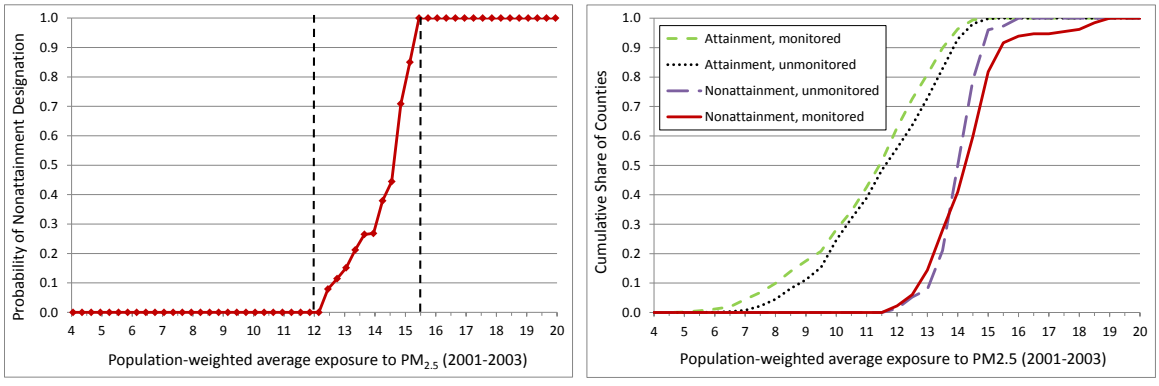
[26] Figure 5 includes an additional 16 counties that we drop because county populations are below the federal thresholds for disclosing information on variables that enter our model as covariates. These include Census block group demographics and Medicare Advantage enrollment shares.

FIGURE 5: AIR POLLUTION BY COUNTY ATTAINMENT STATUS IN 2005



Note: The figure reports annual average daily concentrations of particulates smaller than 2.5 microns ($PM_{2.5}$). Measurements are taken from air quality monitors in 660 counties designated in 2005 as attainment (528) or nonattainment (132) with the federal standard. Each data point in the nonattainment line is a simple average over monitors in nonattainment counties. 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 the 528 attainment counties and 27% living in the 132 nonattainment counties. Corresponding general population shares were 43% (attainment) and 28% (nonattainment).

FIGURE 6: COUNTY ATTAINMENT STATUS BY 2001-2003 $PM_{2.5}$ EXPOSURE



Note: The left figure displays the fraction of counties that EPA designated as nonattainment in 2005 within 0.3 microgram per cubic meter bins for average county exposure. Average county exposures are calculated using our inverse distance measure for the people we observe living in each county between 2001 and 2003. EPA used this 3-year interval to define 2005 nonattainment status. The points denote bin midpoints. The left figure is constructed for 646 counties with air quality monitors. The right figure is constructed for all 3,062 counties in our data. It displays CDFs of the average exposure for people we observe living in attainment and nonattainment counties with and without air quality monitors.

Figure 6 highlights three important features of our data. First, our inverse distance measures of older adults' residential exposures to $PM_{2.5}$ are consistent with EPA's attainment designations. Virtually no attainment counties have average exposures over the regulatory threshold. This is equally true for counties with and without air quality monitors. Second, we can use overlap in the exposure ranges for attainment and nonattainment to construct a "matching" design in which a subset of counties with similar baseline exposures faced different regulatory intensity. Among the subset of counties with air quality monitors, there are 108 nonattainment and 250 attainment counties with average 2001-2003 exposures between 12 and 15.5 $\mu g/m^3$. Most of the nonattainment counties in this group had average exposures below the regulatory threshold but were classified as nonattainment because (1) they contained pollution "hot spots" that violated the standard or (2) 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 instrument outside a narrow range for baseline exposure: $E\left[\Delta\epsilon_{jr}\middle|Z_{js,2005}\right]\forall j: 12 \leq \sum_{s=01}^{03}\frac{PM2.5_{jr,s}}{3} \leq 15.5$. Finally, we can leverage the fact that attainment status was based on the dirtiest monitor in each county to construct a sample consisting of attainment counties just below the regulatory threshold and nonattainment counties just above the threshold, allowing us to relax the exogeneity condition outside a window around the threshold. Following Chay and Greenstone (2005) we treat these matching and subsample designs as robustness checks on our main results.

C. Estimates for the Effect of $PM_{2.5}$ Exposure on Dementia Diagnoses

Table 1 summarizes our estimates for the effect of a 1 $\mu g/m^3$ increase in 10-year annual average residential $PM_{2.5}$ exposure on county-level dementia rates. The first four columns report results for our full sample of 3,022 counties.[27] Column (1) is the simplest specification. The explanatory variables are $\Delta PM_{2.5}$ and covariates describing the health of the county's older adult population in 2004: Medicare Part A and B expenditures per

[27] There are 3,142 counties and county equivalents in the United States. The 120 that are excluded from our analysis are generally 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.

capita and diagnosis rates for chronic heart failure, ischemic heart disease, diabetes, hypertension, and stroke. We calculate county means for these and other variables using the micro data (an unbalanced panel of 7.4 million people; 3.3 to 3.6 million people per year). Column (2) adds covariates describing changes between 2004 and 2013 in the demographic composition of county populations and local access to health care. These include the change in mean age interacted with indicators for 1-year bins for mean age in 2004; changes in hospital beds per capita and medical doctors per capita; changes in the fraction of seniors enrolled in Medicare Advantage plans; changes in Medicare expenditures per capita and morbidity rates; changes in percent female, black, Asian and Hispanic; and changes in mean Census block group variables describing the residents' neighborhood household income, income per capita, year the housing stock was built, mean and average house value, gross rent, percent owner occupied, and fractions of people in four race bins and seven educational attainment bins. Finally, columns (3) and (4) add indicators for geographic regions to complete the econometric specification in equation (1). The indicators denote nine Census divisions in column (3) and states in column (4). All regressions are weighted by county sample sizes to make the coefficients nationally representative.[28]

As a baseline for comparison, panel A reports OLS estimates of the first differenced model. The coefficients are positive and mostly statistically distinguishable from zero. The point estimates from our full specifications in columns (3)-(4) imply that a 1 $\mu g/m^3$ increase in decadal exposure to $PM_{2.5}$ increases a county's dementia rate by six to eight hundredths of a percentage point.

Panel B shows that county nonattainment status is a strong instrument for $\Delta PM_{2.5}$. It causes the point estimates to increase in magnitude and stabilize across specifications. As we incrementally add covariates, moving from column (1) to column (2) to column (3), our point estimates decrease but remain statistically significant. The model in column (3) mirrors the main specification from Chay and Greenstone (2005) in that it is identified by

[28] We weight each county by the minimum of the number of people we observe living in the county 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.

variation in air quality changes between attainment and nonattainment counties in the same Census division, conditional on covariates describing temporal changes in county populations. Column (4) shows that our estimate hardly changes if we replace the Census division dummies with state dummies that force the identification to come from variation between attainment and nonattainment counties in the same state.

TABLE 1—FIRST DIFFERENCE ESTIMATES FOR THE EFFECT OF $PM_{2.5}$ ON DEMENTIA

	(1)	(2)	(3)	(4)	(5)	(6)
	A. First Difference - OLS					
$\Delta PM_{2.5}$ (1 ug/m^3)	0.140***	0.054*	0.081**	0.064	0.101*	0.09
	(0.038)	(0.032)	(0.035)	(0.046)	(0.054)	(0.069)
R^2	0.108	0.509	0.53	0.557	0.694	0.736
	B. First Difference - 2SLS					
$\Delta PM_{2.5}$ (1 ug/m^3)	0.647***	0.488***	0.428***	0.400***	0.489***	0.440**
	(0.081)	(0.076)	(0.085)	(0.131)	(0.124)	(0.182)
F statistic on attainment IV	940	693	620	429	150	98
county change covariates		x	x	x	x	x
Census division dummies			x		x	
state dummies				x		x
monitored county sample					x	x
mean dementia rate (2013)	13.2	13.2	13.2	13.2	13.4	13.4
number of counties	3,022	3,022	3,022	3,022	644	644

Note: The dependent variable is the change between 2013 and 2004 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 annual average exposure from 2004 to 2013 and annual average exposure from 2001 to 2003. Panel A summarizes OLS regressions. Panel B summarizes 2SLS regressions that instrument for $\Delta PM_{2.5}$ with an indicator for nonattainment designation in 2005. Col (1) controls for average heath among people we observe living in the county in 2004. Col (2) extends the model from Col (1) to include a set of covariates describing changes in county populations between 2004 and 2013. Col (3) extends the model from Col (2) to include dummies for the nine Census divisions. Col (4) replaces the Census division dummies in Col (3) with state dummies. Cols (5)-(6) repeat the regressions from Cols (3)-(4) for the subset of counties that had air quality monitors from 2001-2003, the period used to determine nonattainment status. Asterisks indicate statistical significant at the 10%, 5%, and 1% levels based on robust standard errors. See the text for additional details.

The last two columns of Table 1 report results from repeating estimation of the models in columns (3)-(4), limiting the sample to 644 counties that had air quality monitors in place throughout the 2001-2003 period EPA used as the basis for making nonattainment designations. These counties tend to be larger as the EPA tends to place monitors in more populous places. In 2013, they contained 69% of the 3.55 million people in our data. Focusing on this subsample has the twin advantages of improving the precision of our esti-

mates for county dementia rates and reducing the scope for measurement error in $\Delta PM_{2.5}$. As can be seen in Table 1, these precautions leave the results virtually unchanged.

TABLE 2—ROBUSTNESS OF 2SLS ESTIMATES FOR THE EFFECT OF $PM_{2.5}$ ON DEMENTIA

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
$\Delta PM_{2.5}$ (1 ug/m^3)	0.489*** (0.124)	0.750*** (0.199)	0.371** (0.172)	1.019** (0.475)	0.784** (0.365)	0.649** (0.299)	0.661* (0.333)
F statistic on attainment IV	150	79	71	41	68	33	52
other criteria pollutants		x					
balanced monitor sample			x				
matching sample				x			
regression discontinuity sample					x		
large county sample						x	x
balanced person sample							x
mean dementia rate (2013)	13.4	13.3	13.3	13.7	13.5	13.5	21.3
number of counties	644	644	433	358	298	292	125
number of people in 2013 (million)	2.44	2.44	1.82	1.45	1.02	2.09	0.53

Note: The dependent variable is the change between 2013 and 2004 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 annual average exposure from 2004 to 2013 and annual average exposure from 2001 to 2003. The instrument for $\Delta PM_{2.5}$ is an indicator for nonattainment designation in 2005. Col (1) is the same as Col (5) of Table 1. Col (2) adds data on changes in other air pollutants: PM_{10}, ozone, carbon monoxide, nitrogen dioxide, and sulfur dioxide. Col (3) through Col (7) repeat the specification from Col (1) for subsets of counties. Col (3) uses counties that monitored $\Delta PM_{2.5}$ continuously from 2001-2013. Col (4) uses a "matching" sample of counties with average 2001-2003 $PM_{2.5}$ concentrations between 12 and 15.5 ug/m^3. Col (5) uses a "regression discontinuity" sample of attainment counties with 2001-2003 $PM_{2.5}$ concentrations between 11.55 and 12.05 ug/m^3 and nonattainment counties with concentrations between 12.05 and 18.55 ug/m^3. Col (6) is an unweighted regression for counties with at least 2,000 individuals. Col (7) uses a balanced panel of people living in the same county from 2001 through 2013, using counties with at least 2,000 people. Asterisks indicate statistical significant at the 10%, 5%, and 1% levels based on robust standard errors. The text gives additional details.

The 2SLS estimates in columns (3)-(6) collectively suggest that a 1 μg/m^3 decrease in exposure to $PM_{2.5}$ between 2004 and 2013 (an 8% decrease relative to the sample-weighted mean) caused county dementia rates to decrease by about 0.4 percentage points (a 3% decrease relative to the sample-weighted mean). To put this finding in context, the people we observe living in nonattainment counties experienced a reduction in decadal average exposure to $PM_{2.5}$ of 3.09 μg/m^3 compared to a reduction of 1.64 μg/m^3 for people in attainment counties. A back-of-envelope calculation in which we multiply 0.4 by the difference in $\Delta PM_{2.5}$ experienced by people living in nonattainment and attainment counties implies that EPA's nonattainment designations reduced dementia rates in those counties by about six tenths of a percentage point (or 4.5%) on average.

Table 2 summarizes the robustness of our 2SLS estimates to controlling for changes in other pollutants and narrowing our focus to specialized subsamples. For convenience, column (1) replicates the coefficients that we obtain from estimating the model with Census division dummies on the subset of monitored counties (Table 1, col. 5). Column (2) shows that adding changes in other criteria pollutants as covariates (PM_{10}, ozone, carbon monoxide, sulfur dioxide and nitrogen dioxide) causes the $PM_{2.5}$ coefficient to increase, suggesting that it drives the effect on dementia. In comparison, coefficients on the other pollutants are negative and/or statistically indistinguishable from zero. Column (3) shows results from estimating the same model as (1) after dropping 211 counties that were ever missing monitor data between 2004 and 2013.[29] Column (4) reports results for the matching sample of 358 counties with pre-decadal exposures between 12 and 15.5 $\mu g/m^3$. Column (5) reports results for the regression discontinuity sample of attainment counties just below the regulatory threshold (11.55 to 15.05 $\mu g/m^3$) and nonattainment counties just above the threshold (15.05 to 18.55 $\mu g/m^3$).[30] Column (5) reports results for an unweighted regression that ignores differences in county population sizes. As an alternative to weighting, we limit the sample to the 292 monitored counties that had at least 2,000 people in 2004 and 2013. In summary, as we compare column (1) to columns (3)-(6) it is unsurprising to find that reducing the sample size and changing the sample geography reduces the stability of point estimates and yields wider confidence intervals. Yet, the coefficients are remarkably robust in indicating that higher decadal exposures to $PM_{2.5}$ cause dementia rates to increase.

Finally, column (7) limits the sample to a balanced panel of individuals who we observe continuously living in the same county from 2001 through 2013. Focusing on this group avoids potential concerns about ecological fallacy and aggregation bias. A drawback is that it reduces the number of individuals in our sample substantially, leaving far fewer counties where we can estimate dementia rates precisely. Dementia rates are espe-

[29] Of the 211 counties that we drop, 18 are nonattainment. There are several reasons why a county may be missing one or more years of monitor data. Some monitors are discontinued or temporarily inactivated; some are dropped because they report fewer than 10 readings per year, and some are dropped because EPA chose to exclude readings triggered by extreme events such as forest fires when calculating the annual averages used in making nonattainment designations.

[30] If we look within a narrower 5 $\mu g/m^3$ window from 12.55 to 17.55, our sample declines to 210 counties, yielding a moderately larger point estimate and standard error: 1.055** (0.481).

cially low for this group in 2004 because few people survive for 10 years following a diagnosis. We address this limitation by again limiting the sample to counties where we observe at least 2,000 people per year. This yields a balanced panel of 0.53 million people continuously living in 125 counties. Only 3% had received a dementia diagnosis by 2004. This increased to 21.3% in 2013, consistent with the increase in their mean age from 74 to 83. Remarkably, the point estimate continues to indicate that a 1 $\mu g/m^3$ increase in decadal exposure to $PM_{2.5}$ increases the 2013 dementia rate by about 3%. To investigate the mechanisms that drive this effect, we now utilize our micro-data to develop an administrative records design.

V. Evidence on Dementia from a Micro-Level Administrative Data Design

This section draws on the full scope and scale of our micro data to estimate how a 1 $\mu g/m^3$ increase in decadal exposure to $PM_{2.5}$ affects an individual's probability of being diagnosed with dementia. We use all of the available information on each person's residential address history, pollution exposures, medical diagnoses and demographics. The econometric identification strategy complements the preceding county-level analysis by focusing on variation in individual $PM_{2.5}$ exposures stemming from residential migration and local (within-county) heterogeneity in ambient $PM_{2.5}$ concentrations.

A. A Probabilistic Model of Individual Dementia Diagnoses

Let D_{it}, be an indicator for whether person *i* was diagnosed with dementia by the end of year *t*. To mitigate potential confounding from residential sorting by dementia patients, we focus on the subset of individuals who were in our Medicare sample in year *t-9*, who had not received a dementia diagnosis at that point, and who were still alive in year *t*. Then we define an indicator for whether person *i* was newly diagnosed with dementia during the decade: $y_{it} = D_{it} - D_{it-9}$, where $y_{it} \in \{0,1\}$ and $D_{it-9} = 0 \; \forall \; i$. This indicator is the dependent variable in our linear probability model,

$$(4) \qquad y_{ibjt} = \alpha_t \left(\frac{1}{10}\sum_{s=t-9}^{t} PM2.5_{is}\right) + \beta_t \left(\frac{1}{10}\sum_{s=t-9}^{t} CP_{is}\right) + \gamma_t X_{it} + \theta_t W_b + \eta_{jt} + \epsilon_{ibt}.$$

Additional subscripts denote the Census block group (b) and county (j) where the person resided in year t. As in our county level models, the coefficient of interest, α, measures the effect of 10-year annual average residential exposure to $PM_{2.5}$. This metric is derived from each person's residential location history so it incorporates changes in pollution experienced as a result of moving. CP_{is} is a similarly defined vector of average decadal exposures to other federally regulated air pollutants.

Dummy variables for counties, η_{jt}, are used to absorb the effects of environmental factors that might be spatially correlated with pollution and cognition, such as extreme temperatures, the presence of lead pipes, and chemical exposures via hazardous waste sites. In particular, extreme temperatures are known to cause morbidities that serve as risk factors for dementia (Deschenes 2014). Equally important, county dummies will absorb regional variation in access to medical care and doctors' diagnostic procedures that could lead to spatial variation in dementia diagnosis rates. Furthermore, the dummies will control for any pre-period sorting across counties on the basis of medical conditions that may serve as risk factors for dementia (Finkelstein, Gentzkow and Williams 2016).

To explain within-county variation in the probability of a dementia diagnosis we include covariates for all of the individual demographic information (X_{it}) present in Medicare administrative records. This includes indicators for race, indicators for age-by-gender bins from age 65 through age 99, and several measures of the individual's health in year t-9.[31] We measure baseline health using the individual's gross expenditures on health care services covered by Medicare parts A and B, and diagnostic indicators for hypertension, diabetes, congestive heart failure, ischemic heart disease and stroke.[32] Since air pollution is a risk factor for these morbidities, controlling for them will help to absorb the manifested effects of individual differences in pollution exposure prior to our study period. To control for socioeconomic status and within-county heterogeneity in non-airborne pollution we include the same Census variables from our county models describ-

[31] All centenarians are grouped into two gender-specific bins because their relatively small numbers prevent us from precisely estimating year-specific coefficients. Our findings on air pollution are unaffected by adding year-specific bins beyond age 100.

[32] 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.

ing education, income, value of the housing stock and other measures of the demographic composition (W_b) of the individual's year *t* Census block group. We also multiply an indicator for whether the person ever changed residences over the decade with the covariates for their race, age, gender, and block group demographics. This is the first of multiple strategies we employ to avoid reverse causality that could be introduced, in principle, by movers with dementia moving to areas with higher $PM_{2.5}$ concentrations (Figure 4).

B. Main Results

We estimate separate regressions for each year from 2008 through 2013.[33] While this approach allows all model coefficients to evolve flexibly over time, we generally find temporal variation in model coefficients to be economically unimportant. Table 3 summarizes our findings, using 2010 as an example. Column (1) reports results from a univariate OLS regression of y_{ibjt} on decadal $PM_{2.5}$ exposure for people who had not received a dementia diagnosis by the end of 2001. The estimate reveals that a 1 microgram per cubic meter increase in decadal exposure is associated with a 0.32 percentage point increase in the rate of dementia, which is equivalent to a 1.7% increase from the sample mean.[34] The coefficient declines when we add covariates for baseline medical expenditures and morbidities, along with individual and neighborhood demographics in column (2). After controlling for these variables, the point estimate is unaffected by adding dummy variables for states in column (3) and counties in column (4).

[33] This is the first six years over which it is possible to consistently observe decadal exposure to fine particulates across the United States because, as noted earlier, EPA's national monitoring network was established in 1999.

[34] The coefficient increases to 0.45*** if we include people who had received a dementia diagnosis by 2001. The increase could reflect pre-period sorting by dementia patients and/or the effects of pre-decadal pollution exposure.

TABLE 3—DECADAL EXPOSURE TO AIR POLLUTION AND DEMENTIA IN 2010

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
$PM_{2.5}$ (1 μg/m^3)	0.32***	0.20***	0.19***	0.20***	0.20***	0.25***	0.26***	0.15**
	(0.02)	(0.02)	(0.02)	(0.05)	(0.06)	(0.03)	(0.05)	(0.07)
PM_{10}						-0.01	0.04	0.07
						(0.02)	(0.04)	(0.05)
ozone						-0.04	0.14*	0.12
						(0.05)	(0.08)	(0.11)
carbon monoxide						-0.02	0.00	0.00
						(0.02)	(0.03)	(0.03)
sulfur dioxide						-0.01	-0.06**	0.05*
						(0.01)	(0.02)	(0.03)
nitrogen dioxide						-0.02**	-0.04**	-0.06***
						(0.01)	(0.02)	(0.02)
hypertension by 2001		1.11***	1.08***	1.06***	1.15***	1.08***	1.06***	1.15***
		(0.06)	(0.06)	(0.06)	(0.07)	(0.06)	(0.06)	(0.07)
ischemic heart disease by 2001		2.18***	2.10***	2.06***	2.24***	2.10***	2.06***	2.24***
		(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)	(0.08)
chronic heart failure by 2001		3.18***	3.16***	3.12***	3.20***	3.17***	3.12***	3.20***
		(0.13)	(0.13)	(0.13)	(0.14)	(0.13)	(0.13)	(0.14)
diabetes by 2001		3.57***	3.56***	3.53***	3.24***	3.56***	3.53***	3.24***
		(0.09)	(0.09)	(0.09)	(0.10)	(0.09)	(0.09)	(0.10)
stroke by 2001		8.86***	8.82***	8.81***	8.64***	8.82***	8.80***	8.64***
		(0.17)	(0.17)	(0.17)	(0.19)	(0.17)	(0.17)	(0.19)
individual covariates		x	x	x	x	x	x	x
block group covariates		x	x	x	x	x	x	x
state dummies			x			x		
county dummies				x	x		x	x
excludes ever movers					x			x
R^2	0.0003	0.1130	0.1139	0.1172	0.0757	0.1139	0.1172	0.0757
Number of individuals	1,739,385	1,739,385	1,739,385	1,739,385	1,400,712	1,739,385	1,739,385	1,400,712
Mean of dependent variable	19.5	19.5	19.5	19.5	16.5	19.5	19.5	16.5

Note: The dependent variable equals 100 if an individual was diagnosed with dementia before the end of 2010 and 0 otherwise. Pollution exposures are based on 10-year annual averages corresponding to the individual's residential address history. The estimation sample includes all individuals alive in 2010 for whom we continuously observe pollution exposure at their home address between 2001 and 2010. Col (1) is a univariate regression. Col (2) adds covariates for baseline health in 2001, individual demographics and mean demographics for the person's 2010 Census block group. Col (3) adds state dummies and Col (4) replaces them with county dummies. Col (5) drops everyone who moved between 2001 and 2010. Finally, Cols (6)-(8) repeat the specifications from cols (3)-(6) after adding other criteria pollutants. To facilitate comparability, all pollutants are scaled to have the same mean as $PM_{2.5}$ (11.82) so that coefficients can be interpreted as the effect of an 8.5% increase in annual average exposure relative to the mean. Robust standard errors are clustered by Census block group. The text gives additional details.

Column (4) is our preferred model. With the inclusion of county dummies, the coeffi-

cient on $PM_{2.5}$ is identified by heterogeneity in decadal air pollution exposure experienced by people who lived in similar neighborhoods in the same county in 2010, holding constant their health status at the start of the decade, their individual demographics, and their migrant status. This identification strategy requires the full scope and scale of our data; the county dummy variables alone explain 89% of the individual variation in decadal $PM_{2.5}$ exposure. Our sample for 2010 includes just over 600 people per county. The effect of $PM_{2.5}$ on dementia diagnoses is identified by two sources of variation. Among never-movers, $PM_{2.5}$ exposures vary based on where they lived within the county.[35] Among ever-movers, $PM_{2.5}$ exposures vary based on their migration-induced changes in exposure together with the variation in exposure within their 2010 county of residence.

The $PM_{2.5}$ coefficient in column (4) implies that a 1 microgram per cubic meter increase in 10-year annual average $PM_{2.5}$ increases the probability of a dementia diagnosis by 0.20 percentage points, or about 1% relative to the sample mean. This effect is substantial. In comparison, a seminal cohort study by Pope et al.'s (2002) found the same increase in $PM_{2.5}$ to be associated with a 0.4% increase in all-cause mortality among adults over 30. More recently, Chen et al. (2013) found a 0.4% reduction in life expectancy at birth from an additional 10 microgram per cubic meter increase in total suspended particulates near China's Huai river.

While our migration dummies control for possible confounding caused by dementia patients moving to more polluted areas, a remaining concern is that people who are more likely to be diagnosed with dementia in the future may tend to move to polluted areas prior to their diagnoses. To investigate whether this could be driving our results, column (5) estimates the same model as column (4) after excluding everyone who ever moved during the decade, forcing the identification to come from within-county variation in exposure. The resulting point estimate is virtually the same as our main specification, suggesting that our main results do not suffer from reverse causality.[36]

[35] Appendix Figure A5 provides an illustration of this variation for Maricopa County, Arizona—the fourth most populous county in the United States with more than four million residents as of 2015.

[36] For readers interested in comparing the relative effects of air pollutants with coefficients on demographics characteristics, Appendix Table A3 reports coefficients for the non-mover sample in columns (5) and (8) of Table 3. Conditioning on the age and gender effects already seen in Figure 1, we note that diagnosis rates tend to be higher for blacks (+3%), Asians (+1%) and Hispanics (+2%) relative to whites. Meanwhile, diagnosis rates decline by about 0.2% for every $10,000 of additional median household income and tend to be

The coefficients on baseline morbidities allow us to interpret the $PM_{2.5}$ coefficients in terms of relative risks. For example, the probability of a dementia diagnosis is approximately 1% higher for people diagnosed with hypertension at the beginning of the decade. This increase in risk is equivalent to a 10-year increase in annual average $PM_{2.5}$ exposure of 5 to 6 $\mu g/m^3$. A change of this size would have been experienced by someone moving from a relatively clean city in coastal California (e.g. Santa Cruz) to a relatively dirty city (e.g. Los Angeles). Someone moving from one of the cleanest cities in the United States (Honolulu) to one of the dirtiest (Fresno) would have experienced an increase in annual average exposure of 12 $\mu g/m^3$, increasing their probability of a dementia diagnosis by a similar amount as being diagnosed with ischemic heart disease.

C. Robustness and Mechanisms

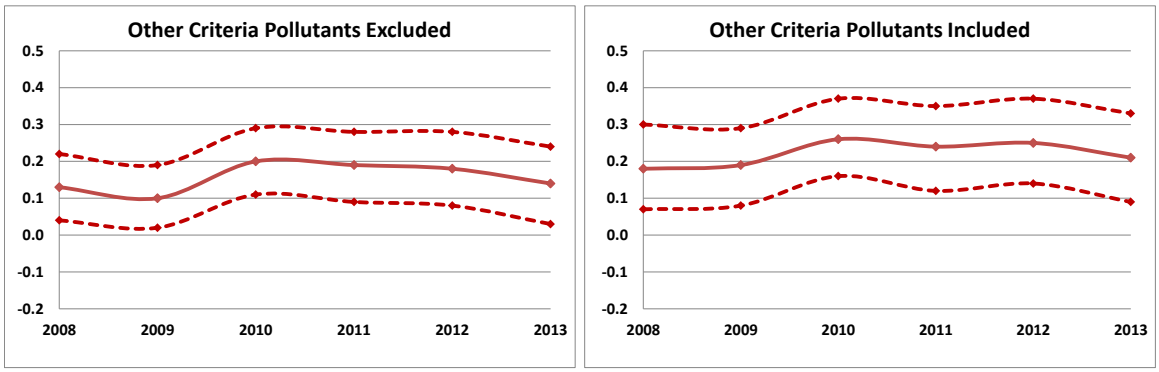
Since a large share of $PM_{2.5}$ emissions are generated by the same sources of other federally regulated air pollutants (e.g. power plants, manufacturing plants, automobiles) the $PM_{2.5}$ coefficients in columns (3)-(5) of Table 3 could be reflecting the effects of those pollutants. To examine whether this is the case, the last three columns of Table 3 report results for the same models as columns (3)-(5) after adding other pollutants as covariates. To facilitate comparisons, all pollutants are normalized to have the same mean decadal exposure as $PM_{2.5}$ (11.82). Their coefficients measure an 8.5% increase relative to the sample average. Results suggest that the effect of air pollution on dementia is driven by fine particulates. Column (6) shows that when we include state dummies the coefficients on other pollutants are an order of magnitude smaller than the $PM_{2.5}$ coefficient and wrong signed. The county dummies that we add to the model in columns (7) and (8) absorb much of the conditional variation in individual pollutants, making it harder to distinguish their effects. Nevertheless, the relative magnitudes of the point estimates continue to point toward $PM_{2.5}$ as the main channel through which pollution affects dementia.

Figure 7 illustrates that our point estimates for the effect of decadal $PM_{2.5}$ exposure are fairly stable from 2008 through 2013, regardless of whether we control for other crite-

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 8th grade education) is associated with a 0.6% reduction in the dementia diagnosis probability.

ria pollutants. The figure graphs point estimates and their 95% confidence intervals for annual models with county dummies. The left panel excludes other criteria pollutants and the right panel includes them, corresponding to columns (4) and (7) of Table 3.

FIGURE 7: THE EFFECT OF DECADAL $PM_{2.5}$ EXPOSURE ON DEMENTIA, 2008-2013



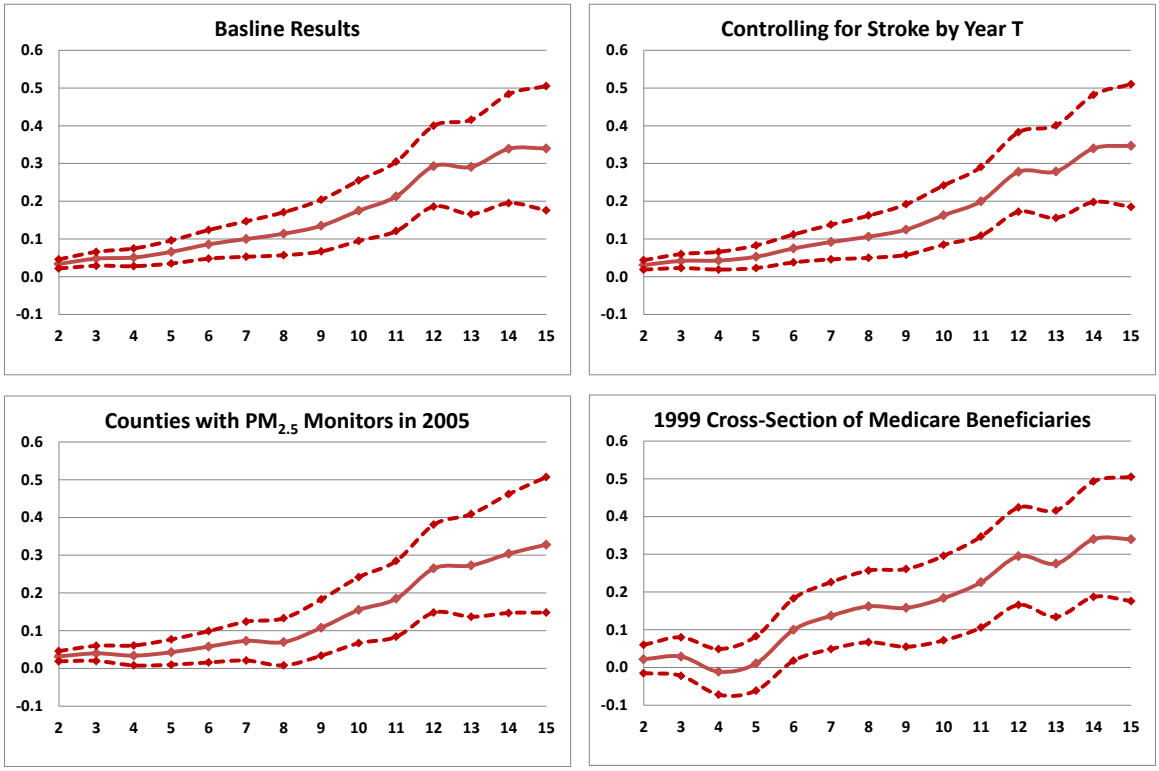
Note: The figures report point estimates and 95% confidence intervals for the $PM_{2.5}$ coefficient from annual regressions. Point estimates give the percentage point increase in the probability of a dementia diagnosis in the indicated year from a 1 microgram per meter increase in 10-year annual average exposure to fine particulates. The figure on the left is based on the specification from col (4) of Table 3. The figure on the right is based on col (7) of Table 3. See the footnote to Table 3 and the text for additional details.

Figure 8 analyzes how the effect of air pollution on dementia varies with the duration of exposure. In the upper left panel, the coefficients and confidence intervals are calculated by estimating the model in (5) for all of the individuals in our data as we incrementally increase the duration of exposure, *T*, from 2 to 15 years. Because the terminal year of exposure varies over individuals depending on when they enter our sample (*s*) and when they die, we add dummies for terminal year, π_{it}, to control for trends in diagnostic methods.

$$(5) \qquad y_{ibjt} = \alpha_t \left(\frac{1}{T}\sum_{s=1}^{T} PM2.5_{is}\right) + \beta_t \left(\frac{1}{T}\sum_{s=1}^{T} CP_{is}\right) + \gamma_t X_{it} + \theta_t W_b + \eta_{jt} + \pi_{it} + \epsilon_{ibt}.$$

The effect of two-year exposure is precisely estimated and close to zero. It increases gradually over time. The confidence intervals widen as the duration of exposure increases because our sample size declines.

FIGURE 8: VARIATION IN THE EFFECT OF $PM_{2.5}$ ON DEMENTIA BY EXPOSURE DURATION



Note: The figures report point estimates and 95% confidence intervals for the $PM_{2.5}$ coefficient from estimating equation (5) as T increases from 2 to 15. Each data point represents the percentage point increase in the probability of a dementia diagnosis (vertical axis) caused by a 1 microgram per cubic meter increase in hourly average residential exposure to $PM_{2.5}$ over a T-year period (horizontal axis) conditional on survival. The top left panel uses all individuals. Each of the other three panels changes one aspect of the estimation. The top right panel replaces the indicator for whether an individual had a stroke as of year 1 with an indicator for whether they had a stroke as of year T. The lower left panel limits the sample to 644 counties with air pollution monitors in 2005. The bottom right panel limits the sample to the initial cross-section of Medicare beneficiaries in 1999. Standard errors are clustered at the Census block group level. See the text for additional details.

To further explore the channel through which pollution affects dementia, we repeat estimation of the model in (5) after replacing the dummy for whether person *i* had a stroke by year *s* with a dummy for whether they had a stroke by the end of year *T*. This dummy will absorb the effect of air pollution on vascular dementia—the second most common form of dementia behind Alzheimer's disease. The upper left panel of Figure 8 shows that this hardly changes the cumulative exposure figure, allowing us to rule out strokes as the primary channel. The lower left panel of Figure 8 shows that our results are robust when we address potential concerns about measurement error by restricting the estimation sample to people living in the subset of 644 counties that had air pollution monitors in 2005 (lower left panel). The lower right panel shows that our results continue

to hold when we address potential concerns about changes in sample composition by limiting our estimation sample to the randomly selected cross-section of Medicare beneficiaries that we started with in 1999.[37]

VI. Evidence on Financial Decisions from a Micro-Level Administrative Data Design

Since 2006, adults age 65 and over have been able to choose standalone prescription drug plans (PDPs) from competing private insurers. The plans are sold in markets that are federally subsidized and regulated as part of the Medicare program. Economists have thoroughly studied financial decision making in these environments, developing several measures of decision making quality that have been used to analyze older adults' abilities to make complex financial decisions in novel market settings (Keane and Thorp 2016). We use these metrics to analyze whether the negative effect of long-term $PM_{2.5}$ exposure on cognition affect financial decision making with or without a dementia diagnosis.

A. Measures of Decision Making in Medicare Prescription Drug Insurance Markets

CMS divides the country into 34 geographic regions, defined as states or groups of adjacent states. Private insurers may design different plans for different regions; they may sell multiple plans in the same region; and they may change the attributes of a given plan in a given region from year to year. Hence, a market is a region-year pair. All Medicare beneficiaries in a given market choose among the same set of plans. Over the first five years these markets operated (2006-2010) the average beneficiary chose among 50 plans sold by more than 20 private insurers. Plans differ in terms of premiums, cost sharing, the generosity of coverage for specific drugs, pharmacy networks and customer service. CMS developed a webpage and a 1-800 number to help consumers gather information about plan quality and the cost of the drugs they take under every plan available to them.

The default for new Medicare beneficiaries is to be uninsured, but from 2006 to 2010 between 64% and 72% of beneficiaries chose to enroll in a PDP.[38] After a beneficiary

[37] The same trends hold if we exclude other air pollutants from the model, though the point estimates are slightly smaller. This is reported in Appendix Figure A6.

[38] An additional 28-36% obtained prescription drug coverage from a Medicare Advantage managed health care plan that provided drug coverage, and the rest obtained insurance from an employer or other sources or chose to forego insurance for prescription drugs

chooses a plan, she is automatically re-enrolled in the same plan for each subsequent year unless she switches plans during the annual open enrollment period. The choice among plans is financially important: excluding those receiving low-income subsidies, the median enrollee spent approximately 6% of her annual household income on premiums and OOP costs and the difference between the total cost of the highest and lowest cost plan averages over $1,000.

Prior studies developed three nonparametric measures of enrollment behavior and used them to assess heterogeneity in the quality of consumers' enrollment decisions.

1. **Inertia**, defined as whether an individual was passively reenrolled in her default plan because she did not actively switch to a different one during the annual open enrollment period. Studies analyzing this metric include Ketcham et al. (2012), Kling et al. (2012), Ho, Hogan and Scott Morton (2015) and Heiss et al. (2016).

2. **Potential savings**, defined as the amount of money an individual spent on her chosen bundle of drugs in her chosen plan minus what she would have spent had she purchased those same drugs under the cheapest plan available to her. Studies analyzing this metric include Heiss, McFadden and Winter (2010), Ketcham et al. (2012), and 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.[39] Studies analyzing this metric include Ketcham, Kuminoff and Powers (2016a, 2016b) and Keane et al. (2017).

We define potential savings using the cost calculator developed by Ketcham, Lucarelli and Powers (2015). While interested readers can refer to that paper for details, the in-

(Hoadley, Cubanski and Neumann, 2015).

[39] For example, let c_{ijt} represent the 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 the bundle of prescription drugs used by the consumer. Ketcham, Kuminoff and Powers (2016a) prove that 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 $q_{jt} < q_{kt}$.

tuition is that we start with data on the universe of prescriptions filled for each person in each year and then hold 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.[40] This approach assumes that informed consumers have unbiased expectations of their drug needs for the upcoming year, and further that their demand for drugs is perfectly inelastic.[41]

TABLE 4—MEDICARE PRESCRIPTION DRUG PLAN ENROLLMENT AND OUTCOMES

		2008	2009	2010
(1)	# beneficiaries	1,325,628	1,366,338	1,389,157
(2)	mean # of plans available	55	50	47
(3)	active enrollment decisions (%)	21	21	16
(4)	actively switched out of default plan (%)	11	10	9
(5)	mean saving from switching	235	251	218
(6)	mean premium + out of pocket costs ($)	1,289	1,409	1,477
(7)	mean potential savings ($)	296	347	340
	chose plan off efficient frontier in terms of:			
(8)	cost, variance (%)	75	78	63
(9)	cost, variance, star rating (%)	49	43	50
(10)	cost, variance, star rating, insurer (%)	25	19	18

Note: The table summarizes characteristics of decisions made by those enrolled in a PDP without a low-income subsidy. Cols (8) and (9) report the faction of all enrollees choosing plans off the efficient frontier. Col (10) is calculated only for those enrollees whose chosen insurer offered multiple plans. See the text for additional details.

Table 4 reports summary statistics for enrollment decisions and outcomes experienced by the subset of people in our data who purchased PDPs during the three year win-

[40] To define the person-plan-year specific variance, we use our full sample and assign each individual in the 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.

[41] In prior work we have evaluated the robustness of the key findings to both of these assumptions and found that they were both qualitatively immaterial.

dow (2008-2010) in which we can link enrollment decisions to the enrollees' decadal pollution exposures. The average person chose from between 47 and 55 plans. Rows (3) and (4) show the prevalence of inertia. Each year, 10% to 11% of people switched out of their default plans with the remaining active choices coming from a mix of (i) new enrollees, (ii) people whose old plans were eliminated, and (iii) people who moved to a different region and consequently had to choose a plan from a new menu of options. Row (5) shows that people who chose to actively switch plans saved an average of $218 to $251 by switching.[42] This is substantial savings given that the average person's annual expenditures in these markets ranged from $1,289 to $1,477. Dividing row (7) by row (6) reveals that the average person could have reduced her annual expenditures by 22% to 24% by choosing the cheapest plan available to her. Rows (8) to (10) show the fraction of people choosing plans off the efficient frontier as we expand the frontier's dimensionality to measure the variance of potential expenditures and two quality measures. The first measure is a plan-year specific star rating that CMS reports to consumers based, in part, on customer satisfaction surveys. The second quality measure is a set of indicators for insurance companies. Including these measures recognizes that consumers may have heterogeneous preferences over insurer-specific characteristics such as pharmacy access and the ability to get drugs by mail order.

As we move from row (7) to row (10) of Table 4 we incrementally relax the restrictions that we need to place on consumers' preference relations in order to interpret the outcomes in those rows as evidence of decision making quality. At one extreme, treating potential savings as a measure of decision quality effectively restricts utility to be proportional to consumption. At the opposite extreme, row (10) places no restrictions on marginal rates of substitution between cost, variance and quality. Consumer sovereignty is respected unless the consumer could have chosen a different plan sold by their chosen insurer that would have lowered their expected costs, lowered their variance of potential expenditures, and provided equal or higher quality.

[42] This statistic is calculated by the different between the person's expenditures under their new plan and how much they would have spent had they purchased the same drugs under the plan they were enrolled in the prior year.

FIGURE 3: MEASURES OF DECISION MAKING QUALITY BY DEMENTIA DIAGNOSIS

Note: The figures report outcomes of prescription drug insurance plan enrollment decisions in 2008, 2009 and 2010. The figures on the left describe outcomes for enrollees who made active enrollment decisions—either because they were new to the market or they opted out of their default plan. The figures on the right describe outcomes for enrollees who were reenrolled in their default plans because they took no action during the open enrollment period. See the text for additional details.

Figure 9 provides some motivating facts about the statistical association between measures of PDP choice quality and dementia. The bar charts on the left summarize active enrollment decisions and those on the right summarize passive re-enrollment decisions. Each chart reports outcomes in 2008, 2009 and 2010 for three groups of people: (i) those with dementia at the time of their enrollment decisions, (ii) those without dementia at the time of their enrollment decisions who are diagnosed with dementia before the end of 2013 and (iii) those who are not diagnosed with dementia before the end of 2013.

People with dementia are 1.6 percentage points less likely to switch out of their default plans, equivalent to a 15% differential when compared to the mean switching rate. This is important because comparing the figures on the right to their analogs on the left illustrates that active choices lead to better outcomes, regardless of the metric we use, the year we analyze, or the person's cognitive status. There is an equally strong relationship between dementia and decisions at the intensive margin when we condition on active or passive behavior. In both cases, current dementia patients tend to do worse than people who develop dementia before the end of 2013 and they, in turn, tend to do worse than people who do not develop dementia by the end of 2013. This relationship holds in every year for the level of potential savings and for each of the three dominated plan measures. While these figures do not condition on individual demographics they provide suggestive evidence that dementia impairs financial decision making. This evidence is underscored by the fact that dementia patients are 31 percentage points more likely to have their enrollment decisions made by a proxy compared to beneficiaries without dementia according to data from the Medicare Current Beneficiary Survey.

B. Econometric Model and Results

We use the administrative data to assess how decadal $PM_{2.5}$ exposure affects inertia, potential savings, and dominated choices. For each metric, we analyze the effect of $PM_{2.5}$ at the extensive margin (increasing dementia) and at the intensive margin (accelerating symptoms post-diagnosis and/or impairing cognition in the absence of a diagnosis). Equation (6) shows our econometric specification.

$$(6) \quad y_{ibjrt} = \alpha\left(\frac{1}{10}\sum_{s=t-9}^{t} PM2.5_{is}\right) + \beta\left(\frac{1}{10}\sum_{s=t-9}^{t} CP_{is}\right) + \gamma X_{it} + \theta W_b + \eta_j + \delta_{rt} + \epsilon_{ibt}.$$

As in prior models, covariates include individual and neighborhood demographics (X_{it}, W_b) and county dummies (η_j). There are three differences relative to our model of individual dementia diagnoses in (4). First, X_{it} is expanded to include an indicator for whether person *i* had received a dementia diagnosis by year *t*, along with an interaction between dementia and decadal $PM_{2.5}$ exposure. Second, since we control for dementia directly, we exclude other measures of baseline health that previously served as risk factors for dementia. Finally, because relatively few people in our data chose PDPs we pool observations over the three year study period. At the same time, we add state-by-year dummies (δ_{rt}) to absorb all of the spatial and temporal heterogeneity in the structure of the choice set (e.g. variation in the number of plans, the number of brands, and plan characteristics).[43] Since everyone in our sample who lives in a particular state in a particular year chooses among the same set of plans, the state-by-year dummies force the identification to come from variation in how different individuals choose among the same options.

Table 5 reports our results. Panel A summarizes our full model for measuring the extensive margin effect of air pollution (through dementia) and the intensive margin effect of $PM_{2.5}$ on people with and without dementia. Despite including a flexible set of controls for individual demographics and neighborhood characteristics, we see strong negative effects of dementia on decision quality, consistent with the descriptive evidence in Figure 9. Our results imply that, all else constant, having dementia is expected to increase annual potential saving by $62, increase inertia by reducing the probability of switching plans by 3 percentage points, and increase the probability of choosing plans off the cost-variance frontier (10 percentage points), off the cost-variance-star frontier (9 percentage points) and off the cost-variance-star-insurer frontier (7 percentage points). Hence, dementia clearly worsens financial decision making. As a qualitative finding this is unsurprising, however the economic magnitudes and consistency across diverse measures of decision

[43] This is potentially important because different insurers operate in different states; the insurers who operate in multiple states typically sell different plans in different states; and within a given state, insurance companies often change the features of a given plan from year to year (e.g. adjusting premiums or co-pays). In principle, these sources of variation could explain some of the spatiotemporal variation in our measures of decision making quality.

making quality is remarkable. Looking across the five measures of decision quality, the estimated dementia coefficients represent 14% to 34% increases relative to the sample means.

TABLE 5: EFFECTS OF DEMENTIA AND $PM_{2.5}$ ON PRESCRIPTION DRUG PLAN CHOICES

	(1)	(2)	(3)	(4)	(5)
			Probability chosen plan is off efficient frontier in:		
	Potential savings ($)	Probability of actively switching out of default plan	cost, variance	cost, variance, star rating	cost, variance, star rating, insurer
# of enrollment decisions	1,411,985	1,338,684	1,405,779	1,405,781	939,958
mean of dependent variable	330	10	72	47	20
			A. Interaction model: including other pollutants		
I { dementia = 1 }	61.59***	-3.38***	10.18***	9.35***	6.73***
	(6.52)	(0.45)	(0.65)	(0.80)	(0.83)
I { dementia = 1 } * $PM_{2.5}$ (1 µg/m3)	1.23*	0.23***	-0.16**	0.17**	0.09
	(0.64)	(0.05)	(0.07)	(0.08)	(0.08)
I { dementia = 0 } * PM2.5 (1 µg/m3)	3.76***	-0.04	0.33***	0.43***	0.25***
	(0.51)	(0.04)	(0.05)	(0.06)	(0.06)
			B. Interaction model: excluding other pollutants		
I { dementia = 1 }	61.67***	-3.32***	10.19***	9.46***	6.76***
	(6.48)	(0.45)	(0.65)	(0.80)	(0.83)
I { dementia = 1 } * $PM_{2.5}$ (1 µg/m3)	1.45***	0.03	-0.13**	-0.07	-0.08
	(0.56)	(0.04)	(0.06)	(0.07)	(0.07)
I { dementia = 0 } * PM2.5 (1 µg/m3)	3.99***	-0.23***	0.36***	0.20***	0.08*
	(0.43)	(0.03)	(0.04)	(0.05)	(0.05)
			C. No interaction model: including other pollutants		
$PM_{2.5}$ (1 µg/m3)	3.44***	0.00	0.27***	0.40***	0.23***
	(0.50)	(0.04)	(0.05)	(0.06)	(0.06)
			D. No interaction model: excluding other pollutants		
$PM_{2.5}$ (1 µg/m3)	3.64***	-0.19***	0.29***	0.16***	0.06
	(0.41)	(0.03)	(0.04)	(0.05)	(0.05)

Note: Panel A reports coefficients estimates from models regressing decision making outcomes on an indicator for whether the beneficiary is diagnosed with Alzheimer's disease and related dementias at the time of their enrollment decision, measures of $PM_{2.5}$ interacted with the dementia indicator, and other pollutants. Panel B is the same as A except that other pollutants are excluded. Panels C and D report the coefficient on $PM_{2.5}$ from the same model as Panels A and B, but with dementia indicators excluded. All models pool data from 2008 to 2010 and include county dummies, state x year dummies, age x gender dummies, and covariates for individual and neighborhood demographics. Demographic variables are interacted with a dummy for whether the individual ever moved between 2001 and 2010. Robust standard errors are clustered by Census block group. See the text for additional details.

Turning to the intensive margin, we find mixed evidence for the effect of higher $PM_{2.5}$ exposures on those already diagnosed with dementia. Somewhat counterintuitively, two of the $PM_{2.5}$ coefficients that are statistically significant for this group imply that higher exposures *increase* switching rates and *decrease* the probability that beneficiaries enroll in plans off the cost-variance frontier. One potential explanation is that $PM_{2.5}$ may trigger other negative health shocks (e.g. heart attacks) that change the individuals' drug needs in ways that incentivize them to switch plans or that reduce the dementia patient's role in selecting a PDP. On the other hand, the coefficients suggest that higher exposures slightly increase potential savings and the probability of choosing plans off the cost-variance-star frontier.

By contrast, we find robust evidence that $PM_{2.5}$ worsens financial decisions among those not diagnosed with dementia. For example, a one microgram per cubic meter increase in decadal average exposure would be expected to increase potential savings by nearly $4 and increase the probability of choosing off-frontier plans by between 0.25 and 0.43 percentage points depending on which measure we use. These outcomes indicate that the cognitive impairment from $PM_{2.5}$ occurs among those even without dementia per se.

As in our micro-level models of dementia diagnoses, county dummies absorb much of the conditional variation in individual pollutants, making it harder to distinguish their relative effects. Nevertheless, $PM_{2.5}$ again appears to be the primary channel through which pollution affects cognition. Appendix Table A4 shows that 22 of the 25 coefficients on other pollutants are negative and/or indistinguishable from zero. For comparison, Panel B of Table 5 shows coefficients from models that exclude other pollutants. The coefficients on potential savings and dominated choices are qualitatively unaffected but, interestingly, the results on switching flip, suggesting that $PM_{2.5}$ reduces switching rates among those not diagnosed with dementia and has no effect on those diagnosed with dementia. As a further comparison, Panels C and D report coefficients from the same models as Panels A and B, excluding the dementia indicators so that the $PM_{2.5}$ coefficients can be interpreted as the net of the intensive and extensive margin effects. Finally,

Appendix Table A5 shows that the pattern of results in Table 5 is robust to replacing the ex post measures of potential savings, cost and variance with their ex ante analogs. That is, our general conclusions are unaffected by whether we evaluate enrollment decisions based on the assumption that enrollees myopically expect their year *t* drug needs to be identical to their drug use during year *t-1* or whether we assume they have unbiased expectations about changes in their drug needs between years *t* and *t+1*.

Overall, our findings for the effect of $PM_{2.5}$ on PDP enrollment decisions are consistent with the hypothesis that $PM_{2.5}$ impairs market decisions via dementia as well as via decision making by those without dementia. The latter, intensive margin effect is also consistent with prior evidence that daily spikes in exposure to fine particulates impair cognitive functioning (Chang et al. 2016b) because people in areas with more spikes will tend to have higher long term exposure. More importantly, our findings suggest that these effects are widespread, economically important at exposure levels commonly observed in the United States, and irreversible pending a cure for dementia.

VII. Placebo Tests

While our research design controls for sorting across counties, neighborhood demographics and pre-decadal health, it is still possible that people who were pre-disposed to developing dementia on the basis of genetics or other latent characteristics (e.g. early childhood pollution exposures) could have sorted themselves into neighborhoods *within* their home counties that were more polluted at baseline and remained that way over the next decade, biasing our estimates upward. In principle, there may be heterogeneity in individual pollution exposures even within a Census block group based on where people live relative to emission sources and prevailing wind patterns. If this mechanism were confounding our results, then we would expect to see relatively large effects for pollutants that are readily observable to people because they are larger in size (particulates between 2.5 and 10 microns), they contribute to urban smog (ozone), and they are generated by obvious 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 consistently find such effects supports our identifica-

tion strategy.

TABLE 6: EFFECTS OF $PM_{2.5}$ ON PLACEBO MEASURES OF MORBIDITY IN 2010

	Cataract	Glaucoma	Endometrial Cancer	Prostate Cancer	Hypo-thyroidism	Bipolar Disorder
			A. Excluding other pollutants			
$PM_{2.5}$ (1 μg/m^3)	-0.44***	0.04	0.00	0.00	-0.04	0.02
	(0.07)	(0.05)	(0.01)	(0.02)	(0.05)	(0.01)
			B. Including other pollutants			
$PM_{2.5}$ (1 μg/m^3)	-0.21**	-0.07	0.00	-0.04	-0.16***	0.01
	(0.08)	(0.06)	(0.01)	(0.03)	(0.06)	(0.02)
Number of individuals	981,563	1,554,069	1,770,967	1,719,176	1,563,307	1,770,745
mean of dependent variable	65.60	17.62	0.71	4.35	19.55	1.22

Note: Panel A reports results for each morbidity using the same specification as column (4) of Table 3. Panel B uses the same specification as column (7) of Table 3. See the footnotes to that table for details.

As an additional test of identification, we repeat our administrative data design for medical conditions that are not known to be caused by air pollution. We use them as placebos to test for confounding. Our two criteria for inclusion are: (1) the condition is not linked to air pollution in the economic or medical literatures, to the best of our knowledge, and (2) the condition is likely to be reliably diagnosed at the beginning of the decade.[44] These include vision problems (cataracts and glaucoma), certain types of cancers (endometrial and prostate), hypothyroidism, and bipolar disorder. If our research design embeds an upward bias for the estimated effects of $PM_{2.5}$ due to individuals' sorting on latent characteristics, then we would expect to see large positive effects of $PM_{2.5}$ on these morbidities. Appendix Table A6 reinforces the power of these placebos by comparing the average demographic and neighborhood characteristics of people with each condition in 2010. While dementia patients are 3 to 5 years older than the average patient with any of the placebo conditions, they are otherwise similar in race and Census block group demographics. In terms of block group demographics, median house value never differs

[44] The first criterion rules out many common medical conditions. For example, the medical literature has linked osteoporosis to air pollution via cadmium exposure in $PM_{2.5}$ and the risk factors for diabetes include hypertension which is believed to be caused by air pollution. The second criterion rules out conditions such as viral hepatitis that often go undiagnosed until very late stages and post-traumatic stress disorder and chronic pain fatigue which were diagnosed relatively rarely at the beginning of our study period.

by more than 8% between dementia patients and placebo patients, household income never differs by more than 4%, the fraction with a college degree never differs by more than a percentage point.

Table 6 reports results from our placebo regressions. When we exclude other pollutants in Panel A all of the coefficients on $PM_{2.5}$ are negative and/or indistinguishable from zero. This continues to be true when we include other air pollutants in Panel B. Hence, the placebo tests suggest an absence of confounding, further reinforcing the identification of our model.

VIII. Summary

This research finds that long term cumulative exposure to small particulate matter causes cognitive impairment among older adults, increasing dementia rates and reducing the quality of financial decisions in prescription drug insurance markets. This study represents the first large scale nationwide analysis supporting the hypothesis from the medical literature that ultrafine particulates cause dementia by entering the brain. We find that the effect of air pollution on dementia is not driven by exposure to particulates larger than 2.5 microns in diameter; it does not occur via vascular dementia that could result from pollution-induced strokes; and the effects are cumulative, consistent with clinical studies of lab rats and other mammals. Moreover, the human cost of these effects is substantial. Our results imply that reducing annual average concentrations of $PM_{2.5}$ by one microgram per cubic meter (an 11% reduction from 2013 levels, and two-thirds of the average reduction achieved in previously highly polluted counties due to strengthening of Clean Air Act in 2005) would help non-poor consumers alone to save approximately $60 million per year on prescription drugs and yield further potential gains in consumer welfare by reducing the probabilities that people choose drug plans off their efficient frontiers in cost-variance space (by 0.2 percent), cost-variance-quality space (by 0.6 percent), and cost-variance-quality-insurer space (by 0.9 percent). Moreover, our results imply that the same moderate reduction in $PM_{2.5}$ would reduce the rate of dementia by 1% to 3% (approximately 100,000 to 300,000 cases), lowering direct medical expenditures on demen-

tia by $3.5 to $10.5 billion per year in 2017 dollars.[45]

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[45] This calculation assume a $35,000 annual differential in medical expenditures between patients with and without dementia, based on statistics from the Alzheimer's Association (2017).

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SUPPLEMENTAL APPENDIX: FOR ONLINE PUBLICATION

TABLE A1—SUMMARY STATISTICS FOR MEDICARE BENEFICIARY SAMPLE

	(1)	(2)	(3)	(4)
	Traditional Medicare and Part D	Part D	Traditional Medicare, Medicare Advantage, and Part D	Traditional Medicare, Medicare Advantage, and Part D
# beneficiaries	7,356,473	1,182,268	10,952,024	13,603,253
always observe ZIP+4	yes	yes	yes	no
Individual demographics				
mean age at sample entry	71	69	71	71
# years in sample	8	12	8	8
male (%)	44	37	43	44
white (%)	83	94	81	81
black (%)	8	3	8	8
asian (%)	3	1	3	3
hispanic (%)	5	2	6	7
dead by end of 2013 (%)	41	27	38	39
mean age at death	83	85	83	83
ever moved (%)	18	24	20	17
ever moved county (%)	10	14	11	9
ever moved state (%)	5	7	5	5
Ever diagnosed with				
stroke (%)	19	21		
diabetes (%)	32	37		
congestive heart failure (%)	36	37		
ischemic heart disease (%)	48	58		
hypertension (%)	71	87		
dementia (%)	23	23		
Census block group means (2012)				
population over 65 (%)	18	19	18	18
bachelor's degree or higher (%)	30	32	30	30
household income (median)	62,095	64,867	61,303	61,212
house value (median)	240,730	245,717	240,985	240,599
gross rent (median)	992	985	1,003	1,003
year built (median)	1,972	1,973	1,971	1,972
owner occupied (%)	63	66	62	62
vacant (%)	11	11	10	11

Note: Columns (1) and (2) report mean characteristics of beneficiaries in our main estimation samples. Column (3) 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 (4) adds to column (3) 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.

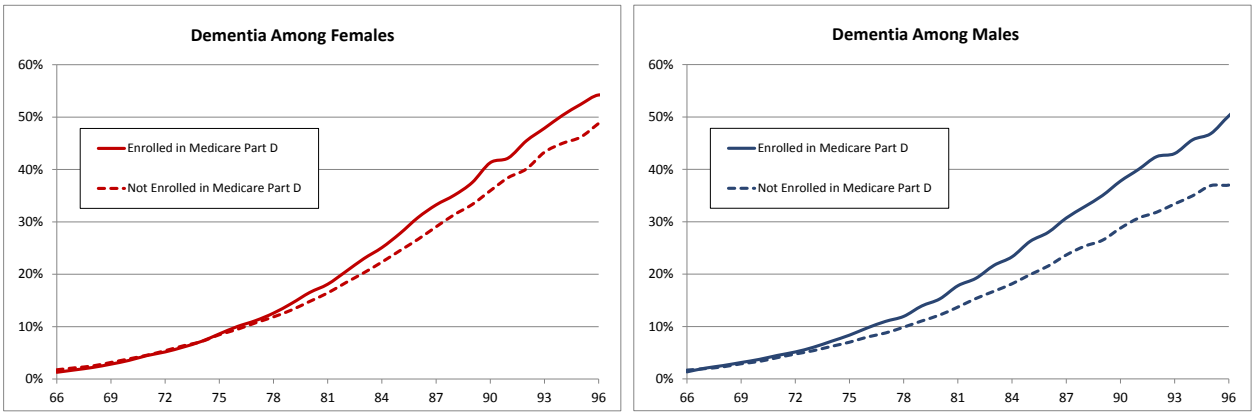
Table A2 summarizes annual average pollution exposures in 2013 and the rate at which people were ever observed to be diagnosed with chronic conditions between 1999 and 2013.

TABLE A2—POLLUTION EXPOSURE AND HEALTH OUTCOMES

	(1) Traditional Medicare and Part D	(2) Part D	(3) Traditional Medicare, Medicare Advantage, and Part D
# beneficiaries	7,356,473	1,182,268	10,952,024
always observe zip+4	yes	yes	yes
annual average pollution (2013)			
$PM_{2.5}$ (hourly $\mu g/m^3$)	8.9	8.9	8.9
PM_{10} (hourly $\mu g/m^3$)	18.5	18.4	18.8
Ozone (daily max of 8-hr mean ppm)	0.04	0.04	0.04
Carbon Monoxide (8-hr mean ppm)	0.4	0.3	0.4
Sulfur Dioxide (daily mean ppb)	9.7	9.2	9.8
Nitrogen Dioxide (hourly mean ppb)	1.2	1.2	1.2
Ever diagnosed with:			
mild cognitive impairment	2	4	
alzheimer's	11	12	
dementia	23	23	
heart attack	7	8	
chronic obstructive pulmonary disease	29	32	
ischemic heart disease	48	58	
stroke	19	21	
hypertension	71	87	
lung cancer	4	4	
cataract	55	80	
glaucoma	19	27	
schizophrenia	1	0	
endometrial cancer	1	1	
prostate cancer	6	7	
hyperthyroidism	23	32	
bipolar disorder	2	2	
chronic pain fatigue	15	23	

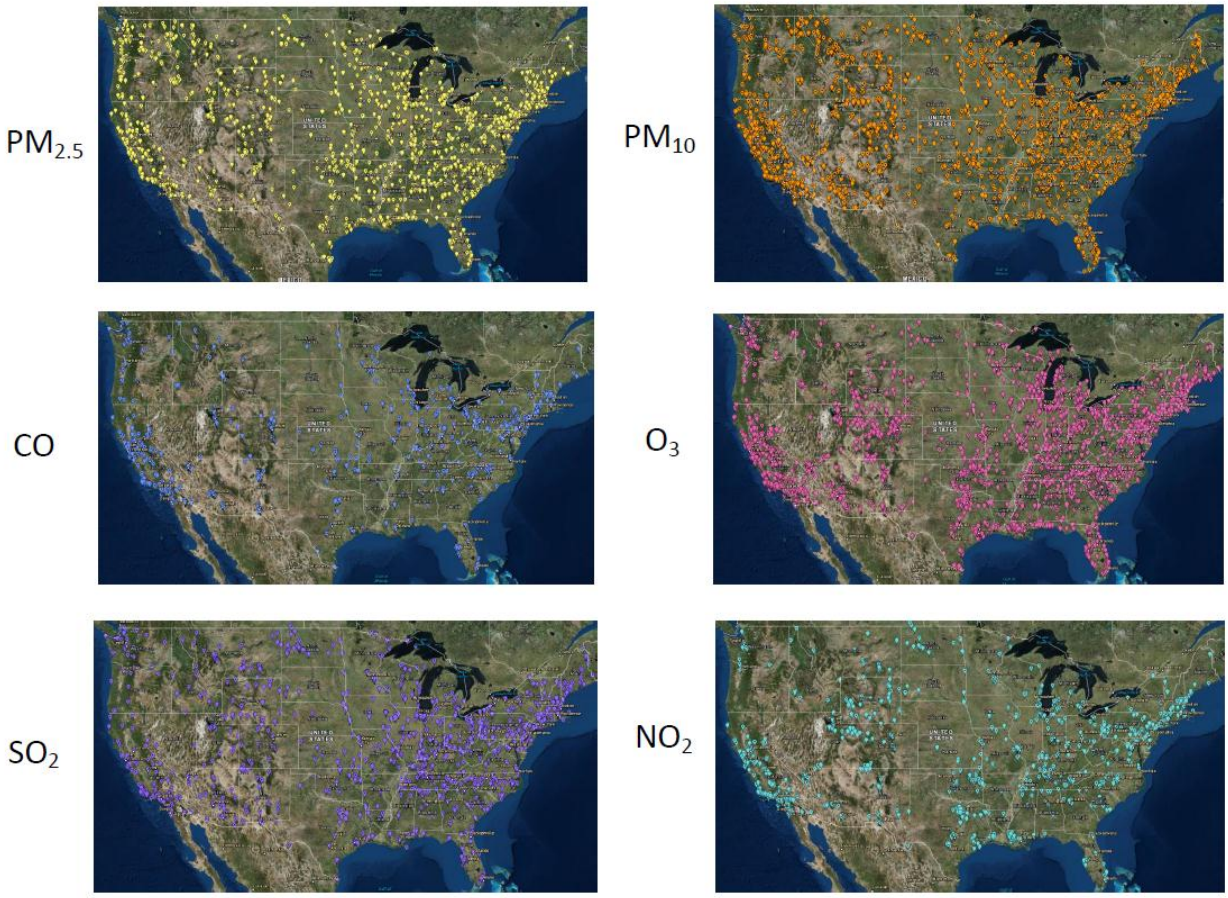
Note: The table summarizes characteristics of beneficiaries.

FIGURE A1: DEMENTIA BY AGE, SEX, AND MEDICARE PART D ENROLLMENT IN 2010



The figures report rates of dementia by age among females (left figure) and males (right figure) among those in our sample who purchased prescription drug insurance plans in Medicare Part D (solid line) and those who did not purchase such plans (dashed line). The figures illustrate that women have higher rates of dementia than men. They also illustrate that people in our Medicare Part D sample have higher rates of dementia, with larger differences for men. For example, at age 82 the rate of dementia among men in Part D is 3.8 percentage points higher those not enrolled in Part D, and the corresponding difference among women is 2.1 percentage points.

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



Each map shows the physical locations of air quality monitors for a particular criteria 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: TIMELINE FOR KEY DEVELOPMENTS IN FEDERAL REGULATION OF $PM_{2.5}$

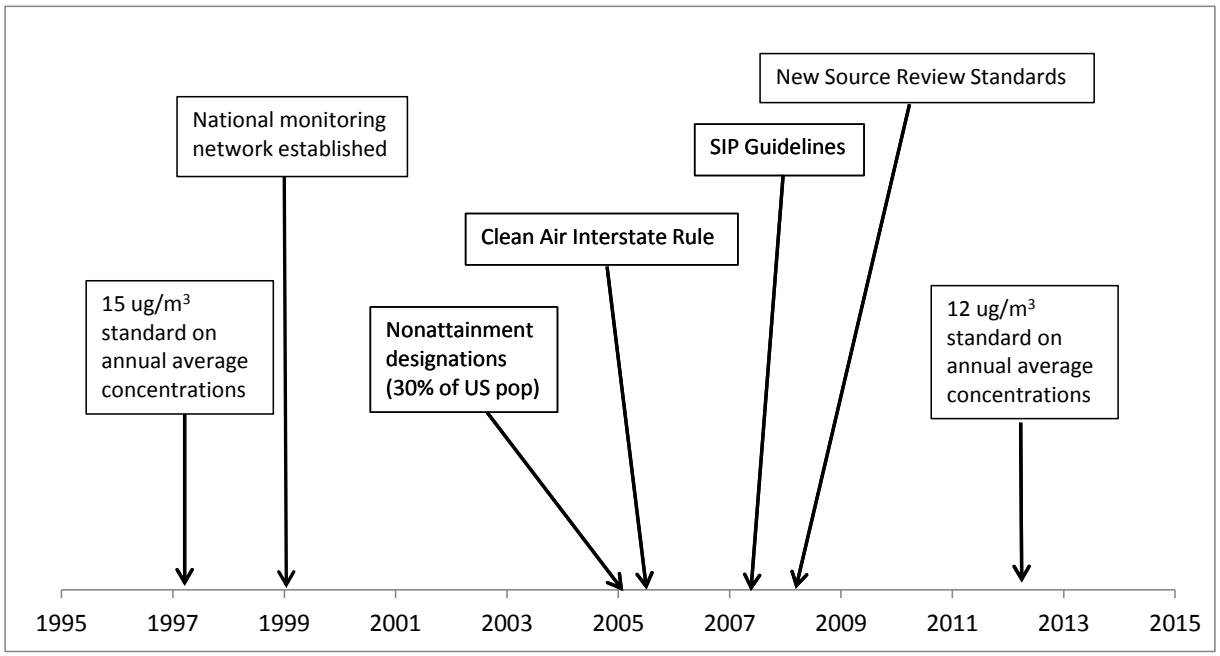
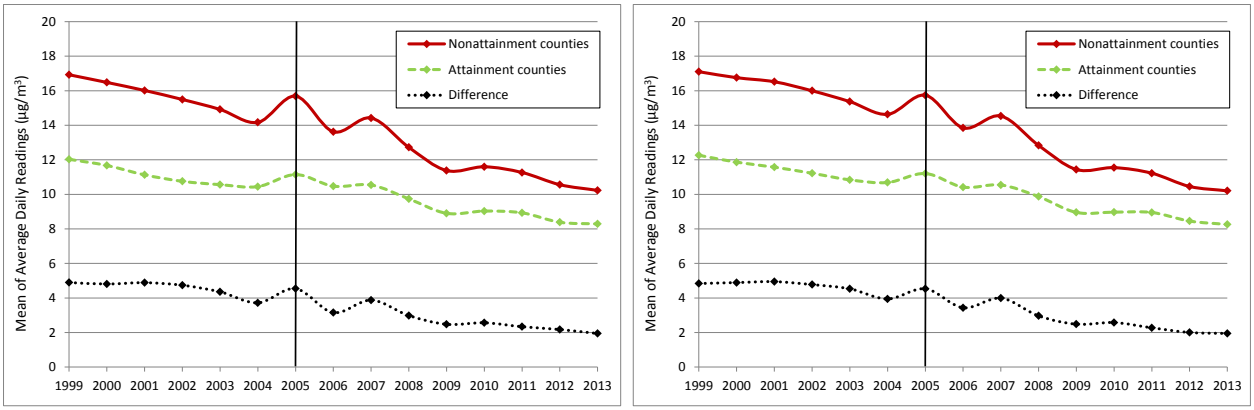
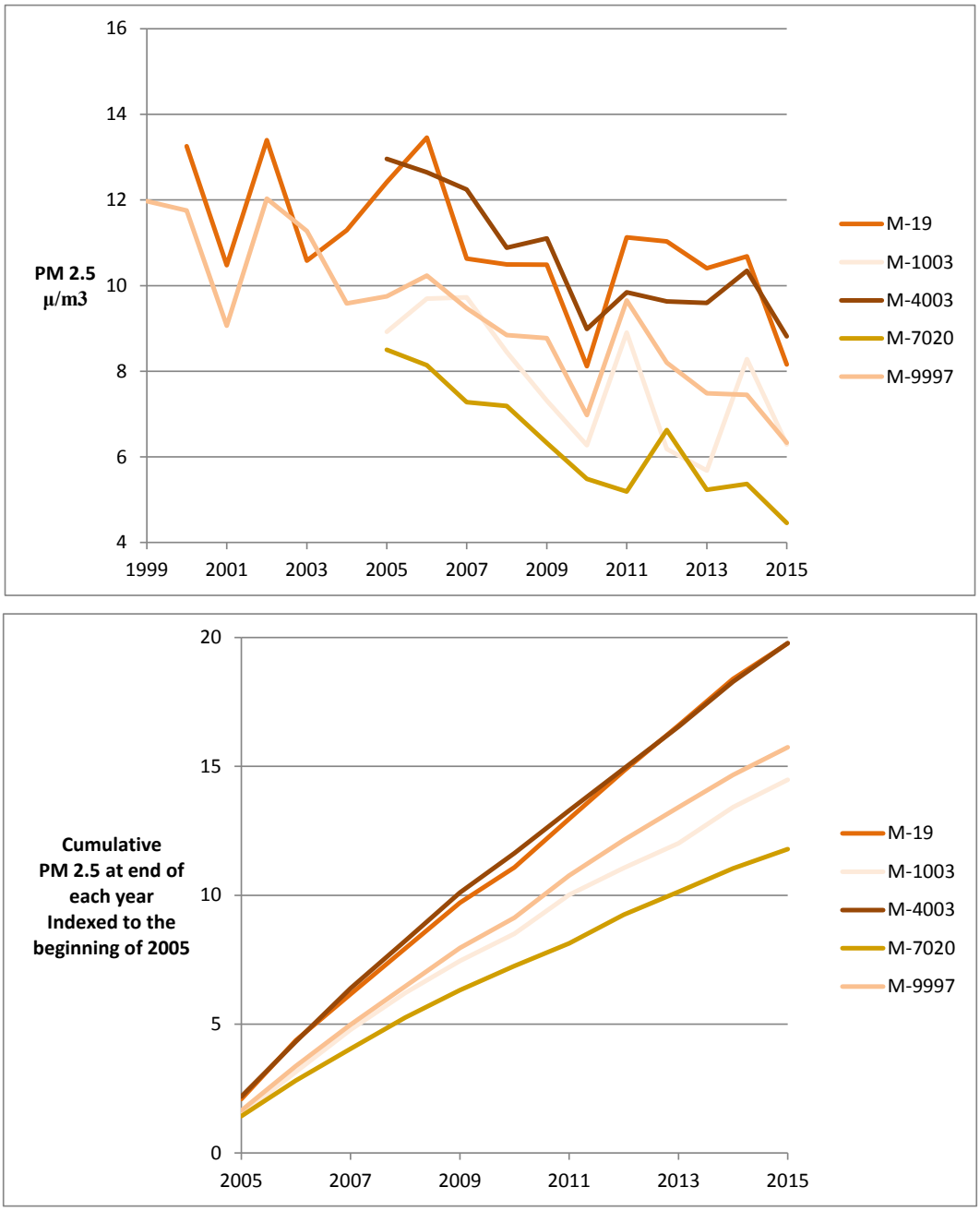


FIGURE A4: AIR POLLUTION TRENDS: UNBALANCED AND BALANCED MONITOR PANELS



The figure on the left is identical to Figure 5. It displays air pollution trends based on simple averages taken each year over an unbalanced panel of between 880 and 1,148 monitors per year. The figure on the right shows the same trends based on a balanced panel of 393 monitors in operation continuously from 1999 through 2013. See the footnote to Figure 5 and the main text for additional details on data construction.

FIGURE A5—AVERAGE $PM_{2.5}$ CONCENTRATIONS AT MONITORS IN MARICOPA COUNTY, AZ



The top figure illustrates within-county variation in annual average $PM_{2.5}$ concentrations for Maricopa County, Arizona, the fourth most populous county in the United States. The bottom figure shows cumulative exposures over a decade for those monitors. The vertical axis in the bottom figure is indexed such that end-of-year cumulative pollution exposure in 2004 is normalized to equal 1.

Table A3 reports additional coefficients and standard errors for the models summarized in columns (5) and (8) of Table 3. Coefficients on dummy variables for more than three thousand counties are not displayed for brevity. See the footnote to Table 3 and main text for details.

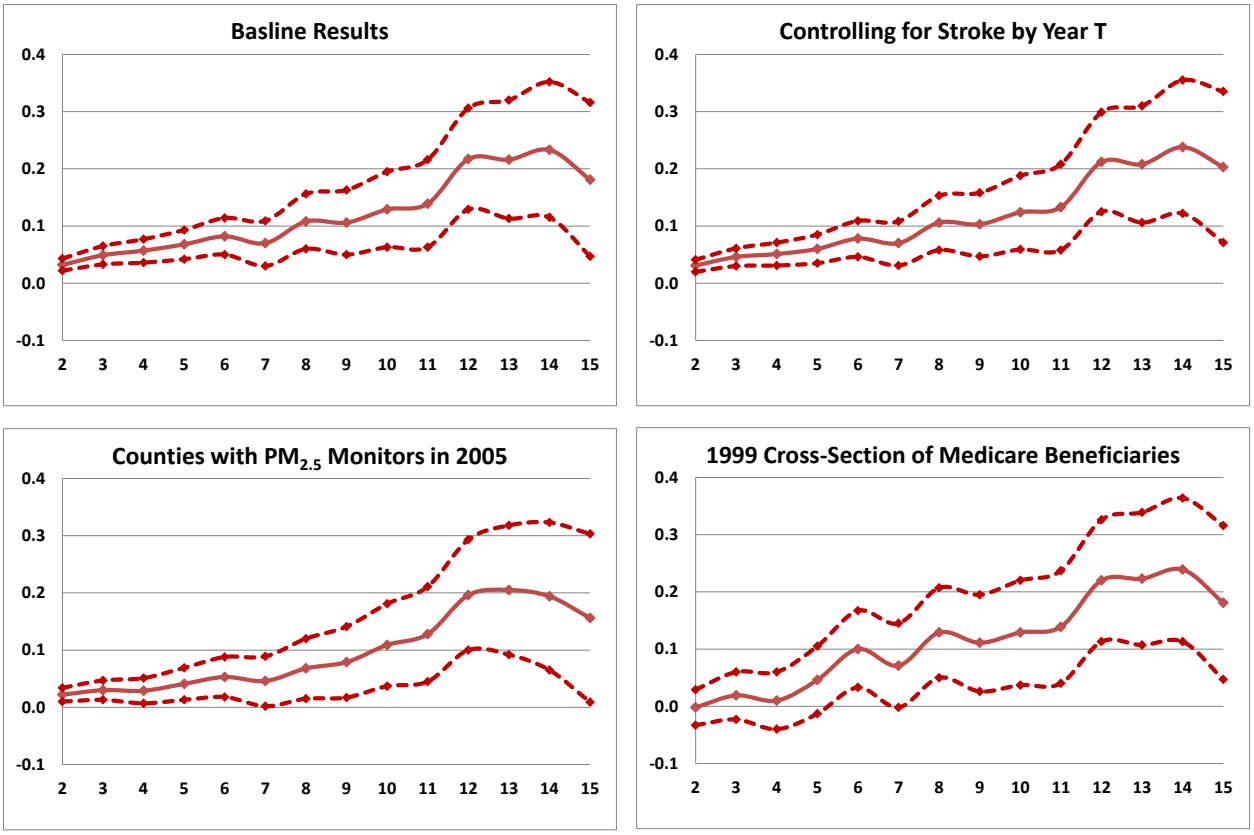
TABLE A3—COEFFICIENT ESTIMATES FOR POLLUTION AND COVARIATES

	Excluding other pollutants		Including other pollutants	
$PM_{2.5}$ (1 μg/m^3)	0.202521	(0.0579166)	0.1500863	(0.0683933)
PM_{10} (normalized)			0.0670632	(0.0451287)
Ozone (normalized)			0.1206198	(0.1082678)
Carbon Monoxide (normalized)			-0.0035541	(0.0332126)
Sulfur Dioxide (normalized)			0.0460945	(0.0273689)
Nitrogen Dioxide (normalized)			-0.0644762	(0.0218003)
hypertension (2001)	1.150499	(0.0681189)	1.150856	(0.0681192)
ischemic heart disease (2001)	2.239822	(0.0848003)	2.239687	(0.0847994)
congestive heart failure (2001)	3.204917	(0.1393372)	3.204329	(0.1393342)
diabetes (2001)	3.235774	(0.0976205)	3.23604	(0.0976219)
stroke (2001)	8.636097	(0.1931395)	8.636241	(0.1931420)
gross medical expenditures (2001)	0.000151	(0.0000051)	0.0001508	(0.0000051)
Census block group demographics				
median household income	0.000019	(0.0000025)	0.0000192	(0.0000025)
per capital income	-0.000020	(0.0000039)	-0.0000203	(0.0000039)
median year built	0.029868	(0.0025084)	0.0300259	(0.0025183)
median house value	0.000001	(0.0000004)	0.00000138	(0.0000004)
average house value	0.000000	(0.0000002)	-0.000000428	(0.0000002)
median gross rent	-0.000007	(0.0000065)	-0.00000692	(0.0000065)
% of residents over 65	-1.457811	(0.3779245)	-1.451791	(0.3778685)
% white	0.663846	(0.4564386)	0.6869844	(0.4568126)
% black	0.194977	(0.5001430)	0.1895814	(0.5001494)
% hispanic	-0.316866	(0.5225033)	-0.2876762	(0.5222435)
% w 9th to 12th grade education	-2.654097	(1.0424260)	-2.633793	(1.0422230)
% with high school degree	-4.912735	(0.8204136)	-4.873154	(0.8203087)
% with some college	-6.015854	(0.8269503)	-5.982908	(0.8268664)
% with associate's degree	-3.824421	(1.0262170)	-3.775751	(1.0260670)
% with bachelor's degree	-4.689287	(0.8403861)	-4.674928	(0.8401255)
% with graduate degree	-6.142397	(0.8823093)	-6.126515	(0.8820682)
% owner occupied	0.729106	(0.3514544)	0.7229988	(0.3515384)
% renter occupied	2.175746	(0.3982013)	2.178072	(0.3984328)
Individual demographics				
black	3.033414	(0.1717863)	3.026481	(0.1717982)
asian	1.198946	(0.2570129)	1.190756	(0.2569887)
hispanic	2.166137	(0.2008535)	2.160633	(0.2009151)
other	-1.608848	(0.3161515)	-1.609054	(0.3161417)

TABLE A3—COEFFICIENT ESTIMATES FOR POLLUTION AND COVARIATES—CONTINUED

	Excluding other pollutants		Including other pollutants	
Age by gender (1=male)				
74 1	-0.452065	(0.1462605)	-0.4527231	(0.1462610)
75 0	0.230916	(0.1476006)	0.2294635	(0.1475998)
75 1	-0.382172	(0.1542232)	-0.3827317	(0.1542272)
76 0	1.391166	(0.1556889)	1.391382	(0.1556913)
76 1	0.461386	(0.1638777)	0.4601025	(0.1638794)
77 0	2.195329	(0.1643268)	2.194231	(0.1643259)
77 1	1.196929	(0.1737528)	1.195012	(0.1737572)
78 0	3.055615	(0.1687662)	3.055357	(0.1687762)
78 1	1.993318	(0.1805435)	1.992399	(0.1805518)
79 0	4.261192	(0.1747583)	4.260243	(0.1747583)
79 1	3.397797	(0.1907840)	3.396609	(0.1907906)
80 0	5.869727	(0.1819218)	5.868654	(0.1819250)
80 1	3.994539	(0.1955403)	3.99428	(0.1955408)
81 0	6.817155	(0.1894657)	6.81645	(0.1894684)
81 1	5.844848	(0.2125458)	5.844732	(0.2125439)
82 0	8.412548	(0.1980570)	8.412395	(0.1980604)
82 1	7.329208	(0.2243064)	7.32785	(0.2243046)
83 0	10.177460	(0.2060225)	10.17695	(0.2060207)
83 1	8.597155	(0.2352052)	8.595785	(0.2352180)
84 0	11.939150	(0.2176864)	11.93858	(0.2176853)
84 1	10.142840	(0.2520206)	10.14232	(0.2520182)
85 0	13.350630	(0.2295327)	13.34903	(0.2295383)
85 1	11.725470	(0.2709061)	11.72455	(0.2709131)
86 0	15.574130	(0.2431751)	15.57323	(0.2431798)
86 1	13.258940	(0.2898449)	13.25728	(0.2898385)
87 0	17.631390	(0.2608278)	17.62961	(0.2608279)
87 1	15.217340	(0.3196120)	15.21476	(0.3195952)
88 0	19.534870	(0.2797663)	19.53416	(0.2797588)
88 1	16.509550	(0.3465544)	16.50966	(0.3465581)
89 0	21.134030	(0.2979182)	21.13183	(0.2979188)
89 1	18.239290	(0.3786842)	18.23769	(0.3786907)
90 0	23.067590	(0.3298822)	23.06496	(0.3298838)
90 1	20.425780	(0.4344944)	20.42509	(0.4345004)
91 0	24.782950	(0.3726342)	24.78063	(0.3726449)
91 1	21.826650	(0.5071934)	21.82352	(0.5071879)
92 0	26.021430	(0.4028841)	26.01992	(0.4028947)
92 1	23.691440	(0.5727839)	23.69113	(0.5728247)
93 0	29.917160	(0.4608692)	29.91654	(0.4608698)
93 1	25.299060	(0.6676821)	25.29966	(0.6676619)
94 0	31.665310	(0.5197983)	31.66342	(0.5197843)
94 1	25.209630	(0.7760374)	25.21077	(0.7760350)
95 0	32.780860	(0.5886554)	32.77925	(0.5886410)
95 1	28.027730	(0.9486133)	28.02776	(0.9485652)
96 0	35.920880	(0.6664952)	35.92172	(0.6664808)
96 1	27.597770	(1.0991730)	27.5899	(1.0991220)
97 0	35.838180	(0.7705567)	35.83749	(0.7705617)
97 1	30.124370	(1.3736920)	30.12235	(1.3738200)
98 0	38.963760	(0.9144481)	38.96259	(0.9143763)
98 1	29.218030	(1.6444260)	29.21611	(1.6444700)
99 0	39.055700	(1.0935020)	39.05168	(1.0934430)
99 1	31.271620	(2.0316090)	31.27654	(2.0315810)
100 0	14.647050	(0.4348911)	14.64512	(0.4349216)
100 1	2.521316	(0.4835784)	2.508665	(0.4837968)

FIGURE A6: VARIATION IN THE EFFECT OF $PM_{2.5}$ ON DEMENTIA BY EXPOSURE DURATION



Note: This figure is the same as Figure 8 except that air pollutants other than $PM_{2.5}$ are dropped from the model. The figures report point estimates and 95% confidence intervals for the $PM_{2.5}$ coefficient from estimating equation (5) as T increases from 2 to 15. Each data point represents the percentage point increase in the probability of a dementia diagnosis (vertical axis) caused by a 1 microgram per cubic meter increase in hourly average residential exposure to $PM_{2.5}$ over a T-year period (horizontal axis) conditional on survival. The top left panel uses all individuals. Each of the other three panels changes one aspect of the estimation. The top right panel replaces the indicator for whether an individual had a stroke as of year 1 with an indicator for whether they had a stroke as of year T. The lower left panel limits the sample to 644 counties with air pollution monitors in 2005. The bottom right panel limits the sample to the initial cross-section of Medicare beneficiaries in 1999. Standard errors are clustered at the Census block group level. See the text for additional details.

TABLE A4: EFFECTS OF DEMENTIA AND $PM_{2.5}$ ON PRESCRIPTION DRUG PLAN CHOICES BY YEAR

	(1)	(2)	(3)	(4)	(5)
			Probability chosen plan is off efficient frontier in:		
	Potential savings ($)	Probability of actively switching out of default plan	cost, variance	cost, variance, star rating	cost, variance, star rating, insurer
			A. Full Model		
I { ADRD = 1 }	61.59***	-3.38***	10.18***	9.35***	6.73***
	(6.52)	(0.45)	(0.65)	(0.80)	(0.83)
I { ADRD = 1 } * $PM_{2.5}$ (1 µg/m3)	1.23*	0.23***	-0.16**	0.17**	0.09
	(0.64)	(0.05)	(0.07)	(0.08)	(0.08)
I { ADRD = 0 } * $PM_{2.5}$ (1 µg/m3)	3.76***	-0.04	0.33***	0.43***	0.25***
	(0.51)	(0.04)	(0.05)	(0.06)	(0.06)
PM_{10}	0.54*	-0.07***	0.04	0.06	-0.08**
	(0.30)	(0.02)	(0.03)	(0.04)	(0.04)
Ozone	-0.70***	0.00	-0.01	0.00	0.05
	(0.23)	(0.02)	(0.02)	(0.03)	(0.03)
Carbon Monoxide	0.12	0.07	0.03	0.59***	0.32***
	(0.63)	(0.05)	(0.07)	(0.09)	(0.08)
Sulfur Dioxide	-0.07	-0.09***	-0.01	-0.32***	-0.16***
	(0.26)	(0.02)	(0.02)	(0.02)	(0.02)
Nitrogen Dioxide	0.03	-0.06***	0.00	-0.03*	0.00
	(0.39)	(0.01)	(0.01)	(0.02)	(0.02)
# of enrollment decisions	1,411,985	1,338,684	1,405,779	1,405,781	939,958
mean of dependent variable	330	10	72	47	20

The table corresponds to Panel A of Table 5, but additionally reports coefficients on other criteria pollutants.

TABLE A5: EFFECTS OF DEMENTIA AND $PM_{2.5}$ ON PRESCRIPTION DRUG PLAN CHOICES BY YEAR

	(1)	(2)	(3)	(4)	(5)
			Probability chosen plan is off efficient frontier in:		
	Potential savings ($)	Probability of actively switching out of default plan	cost, variance	cost, variance, star rating	cost, variance, star rating, insurer
# of enrollment decisions	1,411,985	1,338,684	1,405,779	1,405,781	939,958
mean of dependent variable	330	10	72	47	20
			A. Interaction model: including other pollutants		
I { dementia = 1 }	79.37**	-3.38***	8.03***	7.36***	5.43***
	(33.17)	(0.45)	(0.63)	(0.80)	(0.85)
I { dementia = 1 } * $PM_{2.5}$ (1 µg/m3)	1.41	0.23***	-0.12*	0.24***	0.15*
	(1.52)	(0.05)	(0.06)	(0.08)	(0.09)
I { dementia = 0 } * PM2.5 (1 µg/m3)	6.40***	-0.04	0.27***	0.43***	0.24***
	(2.05)	(0.04)	(0.05)	(0.06)	(0.06)
			B. Interaction model: excluding other pollutants		
I { dementia = 1 }	77.14**	-3.32***	8.03***	7.47***	5.46***
	(30.68)	(0.45)	(0.63)	(0.80)	(0.85)
I { dementia = 1 } * $PM_{2.5}$ (1 µg/m3)	2.62***	0.03	-0.08	0.00	-0.02
	(0.76)	(0.04)	(0.05)	(0.07)	(0.08)
I { dementia = 0 } * PM2.5 (1 µg/m3)	7.43***	-0.23***	0.31***	0.20***	0.07
	(2.68)	(0.03)	(0.04)	(0.05)	(0.05)
			C. No interaction model: including other pollutants		
$PM_{2.5}$ (1 µg/m3)	5.69***	0.00	0.22***	0.41***	0.24***
	(1.69)	(0.04)	(0.05)	(0.06)	(0.06)
			D. No interaction model: excluding other pollutants		
$PM_{2.5}$ (1 µg/m3)	6.71***	-0.19***	0.26***	0.18***	0.06
	(2.29)	(0.03)	(0.04)	(0.05)	(0.05)

The table shows results from repeating estimation of the models shown in Table 5 after replacing ex post measures of potential savings, cost and variance, with their ex ante analogs. The ex ante measures are based on the assumption that when people choose plans during the open enrollment period they expect their drug use over the upcoming year to be identical to the prior year. Thus, the ex ante measures assume that people fail to anticipate changes in their drug needs.

TABLE A6: MEAN CHARACTERISTICS OF PATIENTS AND THEIR NEIGHBORHOODS, BY MORBIDITY IN 2010

		Individual Characteristics					Census Block Group Characteristics					
	# people	birth year	male (%)	white (%)	black (%)	hispanic (%)	median household income ($1,000)	median house value ($1,000)	% college graduates	white (%)	black (%)	hispanic (%)
Dementia (ADRD)	399,173	1924	34	87	7	4	62	237	31	73	11	10
Alzheimer's Disease (AD)	207,108	1924	32	86	8	4	62	239	31	73	11	10
heart attack	117,749	1926	47	90	5	3	59	220	29	75	10	10
COPD	420,149	1927	42	89	6	3	59	223	29	74	10	10
Ischemic heart disease	615,381	1928	40	88	6	3	62	237	30	74	10	10
Stroke	321,133	1926	39	88	7	3	61	235	30	74	10	10
Hypertension	652,732	1929	43	89	5	3	63	241	31	75	9	10
Lung Cancer	40,237	1928	47	90	6	2	61	236	30	74	10	10
Cataract	681,764	1929	42	88	6	3	62	238	30	75	10	10
Glaucoma	289,755	1928	37	85	8	4	64	256	32	73	10	10
Endometrial Cancer	13,791	1928	0	91	5	2	62	244	31	76	9	9
Prostate Cancer	81,101	1929	100	87	8	3	64	252	32	74	10	10
Hypothyroidism	331,059	1927	30	89	5	4	63	251	32	74	9	11
Bipolar disorder	24,798	1927	30	88	6	4	63	247	32	73	10	11

The table reports mean characteristics of individuals and their Census block groups (in 2012) for people who had been diagnosed with each chronic condition before the end of 2010.