Hazed and Confused:
The Effect of Air Pollution On Dementia*

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We study whether long-term cumulative exposure to airborne small particulate matter ($PM_{2.5}$) affects the probability that an individual receives a new diagnosis of Alzheimer's disease or related dementias. We track the health, residential location, and $PM_{2.5}$ exposures of Americans aged 65 and above from 2001 through 2013. The expansion of Clean Air Act regulations led to quasi-random variation in individuals' subsequent exposures to $PM_{2.5}$. We leverage these regulations to construct instrumental variables for individual-level decadal $PM_{2.5}$ that we use within flexible probit models that also account for any potential sample selection based on survival. We find that a 1 $\mu g/m^3$ increase in decadal $PM_{2.5}$ increases the probability of a new dementia diagnosis by an average of 2.15 percentage points. All else equal, we find larger effects for women, older people, and people with more clinical risk factors for dementia. These effects persist below current regulatory thresholds.

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Research shows that airborne particulate matter increases mortality. This effect persists around the world and over time, from the historically high exposures in London in the 1960s (McMillan and Murphy 2017) and China in the 2000s (Li et al. 2019) to the historically low exposures in the US in the 2000s (Deryugina et al. 2019). Research also shows that air pollution constrains the production and the productivity of human capital (Graff-Zivin and Neidell 2013). For instance, daily pollution spikes have been found to reduce students' scores on high-stakes exams (Ebenstein, Lavy, and Roth 2016). Among working-age adults, daily pollution spikes have been found to reduce performance of both manual and cognitive tasks (Chang et al. 2016, Archsmith, Heyes, and Saberian 2017). However, prior research has not applied causal methods to evaluate whether airborne particulate matter degrades human capital later in life apart from mortality.

Our study is the first to use a causal research design to evaluate whether long-term, later-in-life exposure to airborne small particulates (i.e., $PM_{2.5}$, particulates smaller than 2.5 microns in diameter) plays a role in causing dementia. Medical research has documented associations between long-term, later-in-life exposure to $PM_{2.5}$ specifically and the probability of individuals receiving a new dementia diagnosis, although as with other suspected causes of dementia, the precise mechanisms remain unknown (Peters et al. 2019, Underwood 2017, Block et al. 2012). Further, these associations may not be causal due to omitted variables, errors in measuring individuals' pollution exposures, or selection bias.

We develop a research design to account for potential biases due to prior residential sorting (driven by pollution, health, and/or preferences), measurement error in pollution, and selection on survival. Specifically, we estimate the effects of individuals' later-in-life exposure to $PM_{2.5}$ for up to a decade, the longest duration of quasi-random variation available to us. This conditionally exogenous variation resulted from the Environmental Protection Agency's (EPA) expansion of the Clean

Air Act (CAA). Based on air quality monitor readings from 2001-2003, the EPA began to enforce a maximum threshold on $PM_{2.5}$, prompting local regulators to clean up polluted areas beginning in 2004. The regulatory incentives for cleanup were larger in nonattainment counties that exceeded the maximum threshold on $PM_{2.5}$. The incentives caused differences within counties as well. As a result, individuals with the same $PM_{2.5}$ exposures from 2001-2003 experienced different $PM_{2.5}$ exposures over the next decade.

We use this individual-level variation from the EPA's nonattainment designations as instruments to identify how cumulative $PM_{2.5}$ exposure from 2004-2013 affected the probability of receiving a new diagnosis of dementia during this period among Medicare beneficiaries age 65 and above who did not have dementia in 2004. Specifically, we use county nonattainment status flexibly interacted with individual-level $PM_{2.5}$ from 2001-2003 as instruments for the individual's cumulative $PM_{2.5}$ exposure from 2004-2013. In addition to addressing bias from omitted variables, including genetics, earlier-in-life exposure, and other latent risk factors for dementia, our estimators also address the inevitable error in measuring an individual's pollution exposure.

We apply this design to thirteen years of individual-level data on a random sample of millions of Americans age 65 and above. These data track their diagnosis dates for many illnesses including Alzheimer's disease and related dementias, their demographics, and their sequence of residential addresses from 2001 through 2013. We use these residential addresses to link to measures of individual-level $PM_{2.5}$ exposure using data from EPA air quality monitors.

We estimate year-specific probit models that allow for heterogeneity in the effects of $PM_{2.5}$ across individuals and across exposure duration while flexibly controlling for individual characteristics associated with dementia risk, including race, gender-by-integer-age interactions, baseline medical expenditures, baseline expo-

sure to $PM_{2.5}$, fully interacted sets of baseline medical conditions, and the socioeconomic composition of individuals' baseline neighborhoods (defined as a US Census block group). Further, we include Core-Based Statistical Area fixed effects to absorb spatial variation in diagnostic standards, health care quality and access, and latent environmental quality. Finally, we account for the fact that our main estimation sample is limited to individuals who survived through the model year following Heckman (1979). Specifically, we estimate the probability of survival in a separate first stage, using additional instruments constructed from data on individuals' diagnoses of cancers that, based on medical literature, are unrelated to dementia.

We find that a 1 $\mu g/m^3$ increase in average $PM_{2.5}$ concentrations increases the probability of receiving a new dementia diagnosis by the end of the decade by an average of 2.15 percentage points (pp). For reference, a 1 $\mu g/m^3$ increase in average $PM_{2.5}$ was 9.1% of the decadal mean and 59% of the decadal standard deviation during the period 2004-2013. The estimated marginal effects are larger at lower levels of $PM_{2.5}$. We also find that the estimated marginal effects of $PM_{2.5}$ increase with age, illness and duration of exposure, and that they are larger for women relative to men and larger for Black or African-American individuals relative to non-Hispanic White individuals.

We conduct additional analyses to explore the possibility that nonattainment designations are conditionally associated with unobserved earlier-in-life factors that cause dementia, which would violate the exclusion restriction assumption of our instrumental variables. First, we estimate a model with dementia in 2004 as the outcome. The point estimate is negative, small in absolute value, and statistically indistinguishable from zero. This suggests that our model is unlikely to be confounded by unobserved differences in earlier-in-life or other factors that contribute to differences in dementia diagnoses and are conditionally associated with our instruments. Second, we evaluate other placebo health outcomes that may be linked

to earlier-in-life factors but have no known link to $PM_{2.5}$. We do not find a relationship between these placebo outcomes and individuals' cumulative $PM_{2.5}$ exposures.[1] Third, our results persist across a wide range of alternative modeling decisions including controlling for ancillary measures of air pollution exposure.

These findings indicate that air pollution's effects on dementia make its detriments to health and human capital substantially larger than previously realized. Incorporating these effects will be important for comprehensively evaluating the ongoing efforts to improve air quality worldwide.

I. Later-in-Life $PM_{2.5}$ Exposure and New Dementia Diagnoses

A. Existing Knowledge from the Medical Literature

Recent research has documented a positive association between long-term cumulative exposure to fine-particulate air pollution later in life and dementia (Peters et al. 2019, Underwood 2017, Block et al. 2012). In addition, the literature has identified a number of potential pathways to explain this association, even if the details of the accumulation process remain yet unknown. Two physiological hallmarks of Alzheimer's disease specifically are the accumulation of tau protein and amyloid beta (Iaccarino et al. 2021), and recent research has established a link between this accumulation and $PM_{2.5}$ exposure (Park et al. 2021). Research has also found relationships consistent with other potential neurological mechanisms underlying the link between $PM_{2.5}$ and dementia and/or Alzheimer's disease (Alemany et al. 2021), including neuroinflammation caused by accumulation of $PM_{2.5}$ in brain tissue (Kang et al. 2021, Maher et al. 2016), and associations between long-term, later-in-life exposure to $PM_{2.5}$ and accumulated $PM_{2.5}$ in the brain, smaller brain volume, and higher rates of brain infarcts or areas of necrosis and accelerated rates of brain atrophy, which is predictive of Alzheimer's disease (Younan et al. 2020,

[1] In contrast, we find statistically significant positive effects for two outcomes with known links to $PM_{2.5}$ (chronic obstructive pulmonary disease and chronic kidney disorder).

Wilker et al. 2015).

Each of these potential pathways between cumulative $PM_{2.5}$ exposure and a diagnosis of dementia are potentially moderated by a number of factors. These factors may include differences in $PM_{2.5}$ chemical composition (Li et al. 2021), earlier-in-life exposure, cardiovascular risk (Grande et al. 2020), and genetics. While less than half of the genetic factors that contribute to late-onset dementia have been identified (Ridge et al. 2016), recent research has found that genes play a role in moderating environmental factors' relationship to cognitive decline and dementia, including moderating the relationship between $PM_{2.5}$ and dementia, specifically (Alemany et al. 2021, Kulick et al. 2020, Cacciottolo et al. 2017).[2]

B. An Overview of our Research Design

The medical literature described above, along with our data and policy setting, described in Sections II and III, respectively, inform several aspects of our research design. We preview this research design here.

We follow prior medical studies and assess the role of later-in-life, long-term exposure to $PM_{2.5}$ as measured by single- or multiple-year annual average ambient concentrations in explaining new diagnoses of dementia (Wang et al. 2022, Li et al. 2022, Mortimais et al. 2021, Ran et al. 2021, Shi et al. 2021, Shi et al. 2020, Grande 2020, Cacciottolo et al. 2017).[3] Specifically, we observe the timing of individuals' initial diagnosis (or lack thereof) and how it relates to thirteen years (2001-2013) of annual average exposure to $PM_{2.5}$ for them individually based on their precise residential locations each year, allowing us to measure individual-specific exposure histories.[4]

[2] These issues make it difficult to allocate the shares of dementia cases due to genetic risk factors for dementia itself and due to environmental factors directly. Earlier research (e.g., Gatz et al. 1997) provided such shares under the strong assumption of additive separability between environmental factors and genetics.

[3] Like nearly all of the large-scale studies using secondary data, we cannot observe progression or severity of dementia over time. Clinical research commonly refers to this as "incident dementia" or "incidence of dementia". Peters et al. (2019) provides a review.

[4] Dementia is an absorbing state. Therefore, we model the occurrence of the initial diagnosis and exclude from our sample those who had been diagnosed previously.

We depart from the prior medical literature by employing a causal research design to account for potential sources of confounding. Specifically, we observe quasi-random variation in individuals' $PM_{2.5}$ exposures beginning in 2004. As a result, we are able to model the effects of $PM_{2.5}$ exposure across a full decade (2004-2013), conditional on baseline levels of $PM_{2.5}$ (2001-2003).

In Section IV, we present a flexible probit model of how cumulative exposure to $PM_{2.5}$ affects the probability of an individual receiving a new dementia diagnosis. We allow for heterogeneity by letting this effect vary flexibly with the level of cumulative $PM_{2.5}$ exposure over the sample and with the levels of the other controls. We feature models using increasing durations of $PM_{2.5}$ exposure. Finally, we include an extensive set of individual and neighborhood characteristics that may be correlated with new dementia diagnoses. These controls are described in detail in Section II.

Even with this extensive set of controls, identifying the effect of cumulative $PM_{2.5}$ exposure on a new diagnosis of dementia presents several challenges. These include scope for measurement error in $PM_{2.5}$ exposure, the potential for sorting on latent health, genetics, and earlier-in-life pollution exposures, and selection on survival. Our econometric approach, described in Section IV, is designed to account for each of these challenges.

First, to address measurement error in $PM_{2.5}$ exposure and any geographic differentials in unobserved factors, we follow prior work (Chay and Greenstone 2005, Auffhammer, Bento, and Lowe 2009) and develop instrumental variables from the quasi-random variation in $PM_{2.5}$ exposures (conditional on baseline) that was induced by the CAA regulations. Our control-function approach (Rivers and Vuong 1988) relies on the familiar assumptions of relevance and exogeneity for two-stage least squares. The policy environment and the variation-inducing CAA regulations are described in detail in Section III.

Second, to address selection based on survival, we employ a selection-correction approach (Heckman 1979, Heckman and Robb 1986). To implement this approach, we use a set of additional instruments from the medical literature that are correlated with survival, but independent of the unobserved determinants of dementia. We also present a Lee (2009)-style bounds approach in Appendix H that does not rely on this additional set of instruments.

In addition, we consider the potential for sorting on genetics and omitted earlier-in-life factors. Prior research found that individuals' residential exposures to $PM_{2.5}$ do not differ by APOE genotypes (Cacciotolo et al. 2017). In addition, Shin, Lillard, and Bhattacharya (2019) find "no correlation between Alzheimer's Disease polygenic risk score and net worth, housing assets and nonfinancial assets." This indicates that dementia-related genetics are not associated with sorting into neighborhoods based on economic status. These studies provide evidence that genetic factors are unlikely to be correlated with our instrument. To test this directly, we examine the estimates of instrumented $PM_{2.5}$ exposure on the presence of a dementia diagnosis by 2004. In addition to genetics, this assesses whether our results are likely to be explained by association between our instruments and any omitted earlier-in-life factors including other clinical risk factors, prior exposure to $PM_{2.5}$, or different chemical compositions of $PM_{2.5}$.

II. Data and Measures

A. Medicare Data and Sample

The US Medicare program provides universal health insurance for citizens over age 65.[5] The US Centers for Medicare and Medicaid Services (CMS) maintains a comprehensive national database on beneficiaries, including their addresses at each

[5] We analyze "traditional" Medicare (TM) administrative records from CMS. CMS manages and pays claims for services provided to TM enrollees. Beneficiaries can opt out of TM and enroll in a private Medicare Advantage (MA) managed care plan. MA enrollees are left out of most studies of Medicare because MA plans historically did not report claims to CMS. We are able to overcome these limitations and include MA enrollees in some specifications described in Appendix J2.

point in time, medical claims and diagnoses, and demographics. We track individuals from as early as 1999 through the end of 2013.[6] Our featured estimation sample starts with a random 20% sample of all traditional Medicare beneficiaries who were 65 and older on January 1, 2004. We then limit our sample to those who lived in counties with $PM_{2.5}$ monitors, and for whom we can observe their health and residential locations.[7]

B. Measuring dementia and its risk factors

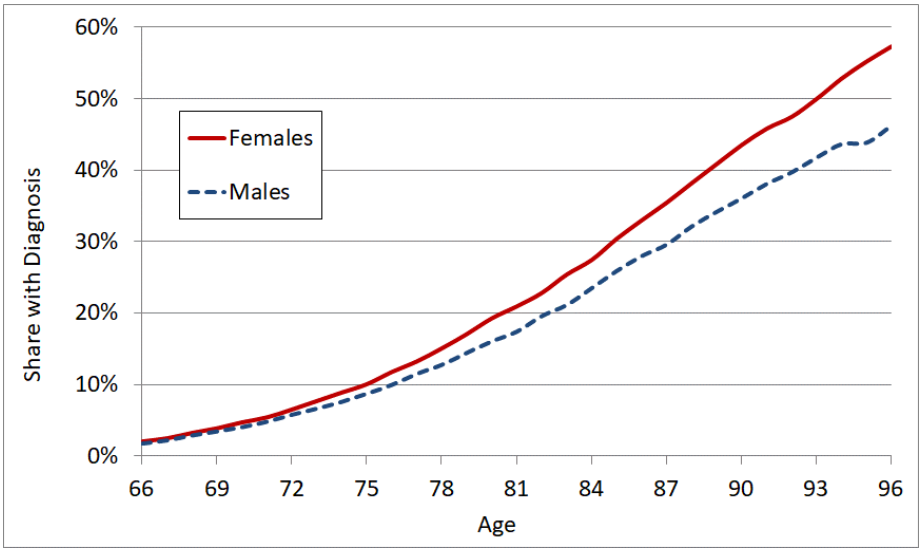
CMS's Chronic Conditions Data Warehouse (CCW) files use codes on Medicare insurance claims to track if and when each individual is diagnosed with specific chronic medical conditions. A dementia diagnosis is based on the presence of multiple symptoms of cognitive impairment that significantly impact daily functioning.[8] Examples include memory loss, impaired judgement, loss of spatial awareness, depression, and behavioral changes. Alzheimer's Disease is the primary type of dementia, accounting for 60% to 80% of all cases. Our claims-based approach to identifying dementia diagnoses is well validated (Lee et al. 2019).

Figure I shows how the fraction of individuals diagnosed with dementia in Medicare data varies with age and gender in 2013. Diagnosis rates increase gradually with age through the mid-seventies before accelerating in the late seventies and beyond. The diagnosis rate is higher for women, and this gender gap widens with age. Conditional on age, diagnosis rates also differ by race. Diagnosis rates are generally higher for people denoted by CMS as "Black or African-American" and lower for "Asian/Pacific Islander" relative to "Hispanic" or "non-Hispanic White".

[6] Due to the provenance of our data, we complement the random 20% sample with an independent, random 20% sample of those also age 65 by January 1, 2004 who purchased standalone prescription drug insurance plans through Medicare Part D at any point between 2006 and 2010 without the aid of low-income subsidies.
[7] We provide additional details about sample cuts and data definitions in Appendix A.
[8] 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."

We account for this heterogeneity by creating a vector of demographics, denoted X_i. This vector includes race code indicators and indicators for each of the 52 possible sex-by-integer-age combinations from age 75 through 100 in 2013.[9]

FIGURE I: DEMENTIA DIAGNOSIS BY AGE AND GENDER IN 2013



We further utilize the administrative CCW files to measure clinical risk factors. Specifically, we create a vector of health characteristics, denoted H_i. This includes indicators for whether the individual in 2004 had each one of the 32 possible combinations of hypertension, diabetes, congestive heart failure, ischemic heart disease, and stroke. These are the known diagnostic risks for dementia (Alzheimer's Association 2019). We further measure baseline health by including in H_i a fourth-order polynomial function of total expenditures on all services covered by Medicare Parts A and B in 2004.[10]

US Census data provide socioeconomic characteristics of the Census block

[9] 75 is the minimum age in 2013 within our estimation sample because that sample is limited to people who were 65 or older on January 1, 2004. Centenarians are grouped into two gender-specific bins because their small numbers prevent us from precisely estimating age-specific coefficients. Our results are unaffected by adding age-specific bins beyond age 100.

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

group where the individual lived in 2004 according to CMS records.[11] We define neighborhood as the individual's Census block group and create a vector of neighborhood characteristics, denoted W_i. This vector includes median household income, per-capita income, mean and median house value, median rent, median house age, fractions of the housing stock that are owner occupied, renter occupied, and vacant, fraction of residents over age 65, fractions of residents who report being white, black, and Hispanic, and the fractions of residents in each of seven educational-attainment bins. These variables account for non-clinical factors associated with different risks of dementia. Appendix Table A1 provides summary statistics for each of the variables represented in X_i, H_i and W_i.

Finally, we create indicators (denoted $C_{i,t}$) for the geographic regions where individuals lived in each year of our model. Specifically, we include 977 indicators for the US Census Bureau's Core-Based Statistical Areas (CBSAs) and the non-CBSA rural areas of each state.[12] In our model, these indicators will absorb the effects of otherwise unobserved factors. First, they help to absorb any effects of residential sorting across CBSAs on the basis of latent risk factors for dementia. Second, they help to absorb the effects of environmental factors that could be spatially correlated with both $PM_{2.5}$ and dementia, e.g., the presence of lead pipes or extreme temperatures which may cause morbidities that are risk factors for dementia. Third, they absorb all differences between geographic areas in health care delivery that might contribute to differences in diagnostic decisions, including patients' access to medical care and physicians' treatment styles.

C. Measuring $PM_{2.5}$ Exposure

In 1997, the EPA established monitoring protocols for $PM_{2.5}$, and by 1999, an

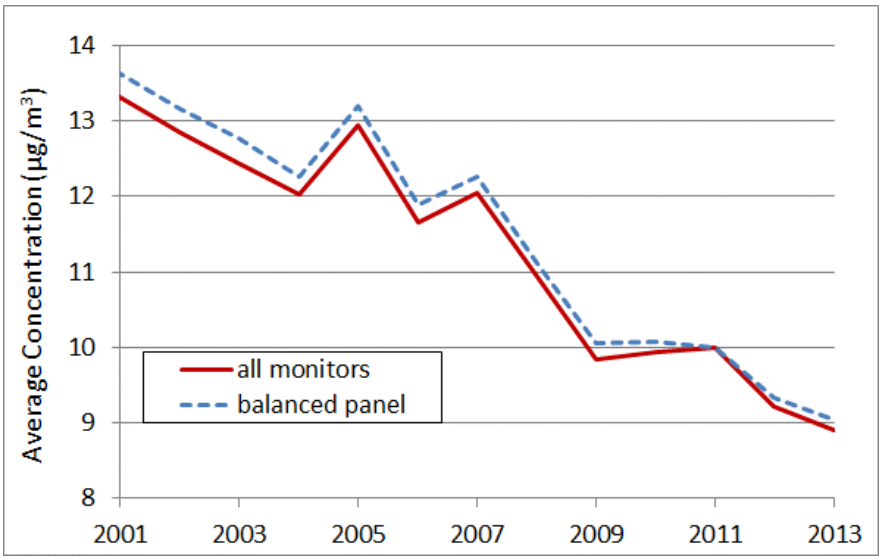
[11] A block group contains 600 to 3,000 residents on average (US Census).
[12] There are 927 CBSAs in the US, which are defined by the Office of Management and Budget as of one or more counties anchored by an urban center of at least 10,000 people plus adjacent counties that are socioeconomically tied to the urban center by commuting. For people living outside of CBSAs, we create an additional 50 state-specific, rural dummy variables.

initial national network of regulatory-grade $PM_{2.5}$ monitors was put into place. We use annual average $PM_{2.5}$ concentrations recorded at each of these monitors from 2001 through 2013. We use data from a balanced panel of 485 monitors that operated continuously through our study period to avoid measurement error that could be introduced if new monitors tend to be located in more or less polluted areas (Grainger and Schreiber, 2019).[13] In a sensitivity check, we instead use data from all 1,722 monitors.

We measure an individual's exposure to $PM_{2.5}$ in year t, $PM2.5_{i,t}$, based on concentrations at their residential address in that year. The CMS data include ZIP+4 Codes for each individual's sequence of addresses from 2004 to 2013.[14] We use this information to measure the individual's cumulative exposure to $PM_{2.5}$ incorporating changes in $PM_{2.5}$ experienced as a result of moving.[15] Individuals in our data live in 2.7 million distinct ZIP+4 Codes during 2004-2013. We use the latitude and longitude coordinates of each monitor and each ZIP+4 to assign the annual average concentration at each residence.[16] Specifically, we calculate the geographical distance between each ZIP+4 centroid and each monitor. Then, for each centroid-year combination, we calculate a weighted average of concentrations recorded at all monitors with the weights given by the square of the inverse distance.[17] Thus, as the distance from a ZIP+4 centroid to a monitor increases, the weight assigned to that monitor decreases.

[13] Following the literature, we drop individuals living in unmonitored counties. See Appendix A for details.
[14] ZIP+4 Codes are close to street addresses in terms of spatial precision: each code corresponds to a single mail delivery point such as a house, one floor of an apartment building, or one side of a street on a city block.
[15] 31% of individuals in our data move at least once, 17% move between counties and 10% move between states. These rates are similar to those reported by the Census Bureau for individuals aged 65 and above. We are unable to observe seasonal migration by people with more than one residence because we only observe the residential address on record with CMS. Fortunately, the scope for measurement error is small. Jeffery (2015) estimates that seasonal migrators only account for 2% to 4.1% of the Medicare population based on addresses on Medicare claims for primary care and emergency room visits.
[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] This method of interpolation, with weights given by the distance raised to a negative exponent, is a predominant method in the environmental economics literature.

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



Note: The figure reports the annual average concentrations of fine particulate matter based on place of residence for people age 65 and above on Medicare.

Figure II shows that annual average concentrations of $PM_{2.5}$ at the residences of the US Medicare population declined substantially during the 2000s, from over 13 µg/m^3 (micrograms per cubic meter of air) in 2001 to about 9 µg/m^3 in 2013. This is true regardless of whether we measure exposure using the balanced panel of monitors (the dashed line) or the full set of monitors (solid line).

We denote our measure of interest, the individual's average cumulative exposure to $PM_{2.5}$ from 2004 to year *t*, as $durPM_{i,t}$. We construct it by combining the described ZIP+4-specific annual $PM_{2.5}$ concentrations with individuals' residential ZIP+4 histories from 2004 to *t* according to: $durPM_{i,t} = \sum_{s=2004}^{t} \frac{PM2.5_{i,s}}{t-2004}$. Finally, we create a measure of the baseline $PM_{2.5}$ concentrations at the locations where individuals lived in 2004. We denote this measure as $basePM_i$ and construct it as the average concentration over the three years 2001 to 2003. These three years are the years that the EPA based its nonattainment designations on, as discussed in the next section.

III. Clean Air Act Regulation of $PM_{2.5}$

The Clean Air Act (CAA) of 1970 established national standards for concentrations of regulated air pollutants. The EPA designated counties containing monitors that exceeded these standards as nonattainment. States with nonattainment counties were required to coordinate with local regulators to bring those counties into compliance with the standards. States that failed to bring their counties into attainment faced penalties including loss of federal highway funds.

Due to its pernicious effects on human health, particulate matter has been subject to sustained and evolving federal regulation (US EPA 2005). Beginning in 1971, the EPA regulated total suspended particulates (TSP). In light of evidence that health effects were driven by the smallest particulates, the EPA replaced the TSP standard with a standard on PM_{10} (particulates smaller than 10 microns in diameter) in 1987 and a standard on $PM_{2.5}$ in 1997. Each new standard was followed by new nonattainment designations. These designations have the ability to affect pollution in both nonattainment and attainment counties because pollution travels across county boundaries. However, the designations for particulate matter have induced relatively larger pollution reductions in nonattainment counties. Prior research used the TSP standard (Chay and Greenstone 2005, Isen, Rossin-Slater, and Walker 2017) and the PM_{10} standard (Bento, Friedman, and Lang 2015) to create instruments for TSP and PM_{10} exposures, respectively. In this paper, we use the $PM_{2.5}$ standard to develop instruments for $PM_{2.5}$ exposures.

In 1997, the EPA set the regulatory standard for average annual $PM_{2.5}$ concentrations at 15.05 $\mu g/m^3$. In April 2003, state and local regulators were given a February 2004 deadline to provide $PM_{2.5}$ monitor data from the years 2001-2003, and to self-report any nonattainment monitors to the EPA, where nonattainment was defined by the monitor's three-year average $PM_{2.5}$ concentrations from 2001-2003. Based on these reports, the EPA formally defined each monitored county to be in

attainment or nonattainment in January 2005.[18] For counties with multiple monitors, the designations were based on the monitor with the highest three-year average from 2001-2003.

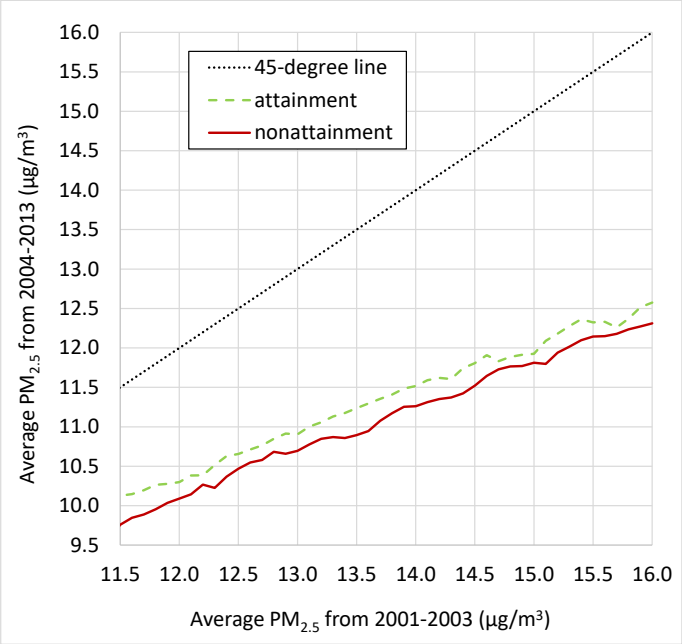
We define 2004 as the start of the regulatory period because local regulators learned which counties would be nonattainment between April 2003, when they received the EPA's request for data, and February 2004, when they were required to submit their status. EPA monitor data show $PM_{2.5}$ concentrations declining at a similar rate in both attainment and nonattainment counties prior to 2004, and then declining at a faster rate in nonattainment counties after 2004. These trends, shown in Appendix Figure C1, are analogous to the evidence that Chay and Greenstone (2005) first presented on the validity of using CAA regulation of PM as a quasi-experiment.

Figure III provides the intuition for how we use county nonattainment designations to isolate quasi-random variation in individuals' average $PM_{2.5}$ exposures from 2004-2013, conditional on baseline concentrations from 2001-2003.[19] The nonattainment and attainment lines plot the coefficients obtained by regressing the individual-level measure of decadal $PM_{2.5}$ exposure, $durPM_{i,2013}$, on indicators for 0.1 μg/m^3 bins of $basePM_i$ interacted with county attainment status, after absorbing CBSA dummies. Comparing the nonattainment and attainment lines with the 45-degree line shows that post-regulatory reductions in $PM_{2.5}$ were larger, on average, for individuals with larger baseline concentrations. This pattern is consistent with prior studies that used CAA regulatory standards to develop instruments for particulate matter exposures.

[18] Appendix Figure B1 shows the locations of attainment and nonattainment counties with air quality monitors. In 2005, 132 of the monitored counties containing approximately 27% of the US population were classified as nonattainment. Another 528 counties containing 43% of the US population were classified as attainment. The remaining counties lacked monitoring data and were designated "unclassifiable" and not subjected to additional regulation. Appendix Figure B2 shows the location of the monitors.

[19] As noted in Chay and Greenstone (2005), attainment status doesn't induce quasi-random variation in pollution levels, but rather quasi-random variation in changes in pollution. Equivalently, in our case, attainment status induces quasi-random variation in decadal pollution exposure, conditional on pre-regulatory baseline pollution.

FIGURE III: POST-REGULATORY $PM_{2.5}$ EXPOSURE 2004-2013, BY COUNTY ATTAINMENT STATUS AND PRE-REGULATORY EXPOSURE 2001-2003



Note: The nonattainment and attainment lines represent estimates from regressing individual exposure from 2004-2013 on indicators for 0.10 µg/m^3 bins of baseline exposure from 2001-2003 interacted with county attainment status. Additional covariates include CBSA dummies.

The key insight from Figure III is that the nonattainment line lies below the attainment line for all levels of average $PM_{2.5}$ from 2001-2003. This difference is statistically significant at the 1% level. This shows that when we compare individuals in the same CBSA who were in the same residential $PM_{2.5}$ bin for pre-regulatory exposure (2001-2003), those who lived in nonattainment counties were subsequently exposed to *lower* $PM_{2.5}$ during 2004-2013 than those in attainment counties. This follows from the incentives that regulators faced to target their mitigation efforts at nonattainment counties (Chay and Greenstone's 2005, Isen, Rossin-Slater, and Walker 2017). In addition, the vertical distance between the nonattainment and attainment lines decreases with baseline $PM_{2.5}$ concentrations from 2001-

2003.[20] This follows from the EPA policy in which a county's attainment status is linked to its dirtiest monitor, thus incentivizing local regulators to target pollution "hot spots" (Auffhammer, Bento, and Lowe 2009, Bento, Freedman, and Lang 2015).

IV. Estimating the Causal Impact of Decadal $PM_{2.5}$ on Dementia

We model how cumulative exposure to $PM_{2.5}$ over the decade from 2004 to 2013 affects the probability of an individual receiving a new dementia diagnosis. First, we consider a contemporaneous, decadal model where the decade is treated as a single time period. Second, we extend this framework to instead aggregate cumulative, year-specific impacts over the decade.

A. A Decadal Model of New Dementia Diagnoses

Let $y_{i,t}$ indicate whether individual *i* has received a dementia diagnosis by the end of year *t* and let $\Delta y_i = y_{i,2013} - y_{i,2004}$ denote the change in dementia status between 2004 and 2013. Because dementia has no cure, it is an absorbing state and, by definition, Δy_i is equal to zero for anyone with dementia in 2004. Therefore, we model whether individual *i* is *newly* diagnosed with dementia by the end of 2013, conditional on having not received a dementia diagnosis before the end of 2004.[21]

We model a new dementia diagnosis using a probit model where Δy_i^* denotes the latent propensity to become newly diagnosed with dementia,

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

and where an individual is diagnosed with dementia if their latent propensity is

[20] The scaling of the vertical axis in Figure III makes this trend hard to discern. It is easier to discern in Figure IV. Fitting a linear trend to the vertical distance between the nonattainment and attainment lines in Figure III reveals that a 1 $\mu g/m^3$ increase in baseline exposure is associated with a 0.02 $\mu g/m^3$ reduction in the vertical distance between the lines.

[21] We begin with a model of new diagnosis of dementia, which is standard in clinical research on dementia. In principle, we could instead begin with a model describing an individual's dementia status in both 2004 and 2013 to derive Equation (1) below. Such a model is shown in Appendix G. Our discussion of identification below explicitly accounts for the fact that the error in Equation (1) captures changes in unobserved dementia determinants, conditional on not having dementia in 2004.

positive, i.e., $\Delta y_i = 1[\Delta y_i^* > 0]$.

The parameter of interest, α_i, represents the causal effect of decadal exposure to $PM_{2.5}$ on Δy_i^*, holding all other factors constant.[22] All other factors that determine Δy_i^* are denoted by the error η_i. Following Angrist and Pischke (2009), we decompose η_i into a linear function of observable controls, $X_i, H_i, W_i, C_i, basePM_i$, and an error, e_i:

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

Combining the two previous equations yields our equation of interest:

$$(1)\ \Delta y_i^* = h\left(durPM_{i,2013}; \alpha_i\right) + \beta_x X_i + \beta_H H_i + \beta_W W_i + \beta_C C_{i,2013} + f(basePM_i; \beta_{basePM}) + e_i$$

In the simplest specification of this model, we specify $h\left(durPM_{i,2013}; \alpha_i\right) = \alpha\ durPM_{i,2013}$. In a more flexible specification of the decadal model, discussed in Section IV.B, we allow for non-linearities and heterogeneity along observable dimensions in the impact of $durPM_{i,2013}$ on the probability of a new diagnosis of dementia. In Section IV.C, we present a model that allows for additional non-linearity and heterogeneity with respect to the duration of exposure to $PM_{2.5}$.

We use α together with the other model parameters to recover the average marginal effect (AME) of changes in $durPM_{i,2013}$ on the probability of a new diagnosis, $Prob(\Delta y_i = 1)$. We discuss the controls, $X, H, W, C, basePM$, and the error, e, in the following paragraphs.

In Section II, we defined the vectors of controls $X, H, W,$ and C. The vector X_i includes indicators for race and gender specific indicators for each integer age. H_i includes indicators for each unique combination of pre-existing clinical risk-factors for dementia (hypertension, diabetes, congestive heart failure, ischemic heart disease, and stroke) and a fourth-order polynomial function of individual medical

[22] Epidemiological "stress" models that consider life histories are discussed in Deaton and Paxson (1998).

spending in 2004. W_i includes Census block group variables describing the socio-economic characteristics of individuals living in individual i's neighborhood in 2004. Finally, $C_{i,2013}$ is a vector of indicators for each individual's 2013 CBSA.

The final control is a fourth-order polynomial function, $f(\cdot)$, of $basePM_i$. This controls for any residual effects of pre-regulatory sorting into more polluted neighborhoods by individuals who are more likely to receive a future dementia diagnosis. In addition, the inclusion of $f(basePM_i)$ means that α specifically measures how cumulative $PM_{2.5}$ exposure from 2004 to 2013 affects the probability of a new dementia diagnosis, conditional on pre-regulatory, baseline concentrations.

Finally, e_i, is an error term that represents any other determinants of a new dementia diagnosis that are not controlled for by a linear function of $X_i, H_i, W_i, C_{i,2013}$, and $f(basePM_i)$. The model imposes no assumption about the relationship between our variable of interest, $durPM_{i,2013}$, and e_i.[23] In fact, e_i most likely contains factors that would lead it to be correlated with $durPM_{i,2013}$, coming from (i) omitted variables, (ii) measurement error, and (iii) factors related to selection.

One example of an omitted variable in e_i that may be correlated with $durPM_{i,2013}$ is earlier-in-life exposure. While we don't specify the direct impact of earlier-in-life $PM_{2.5}$ exposure, we allow for earlier-in-life exposure to affect new dementia diagnoses and to be correlated with $durPM_{i,2013}$.[24] Another example is latent health. If individuals had sorted on unobserved health factors, including genetics, the error term and $durPM_{i,2013}$ may be correlated. Like earlier-in-life exposure, we will not specify the direct impacts of these latent health measures, but we do not rule out their presence in e_i.

Measurement error in $durPM_{i,2013}$ could also be present in e_i. All large-scale

[23] We make an assumption in Section IV.B regarding the independence of e_i and the vector of controls and instruments.
[24] Because we allow prior exposure to be an element of the error term, rather than explicitly model its impact, we cannot answer questions directly related to lifetime exposure. In our model, α captures the causal effect of later-in-life decadal pollution on the probability of a new dementia diagnosis, holding all else constant, including earlier-in-life exposure.

data on air pollution are based on ambient measures, such as satellite imaging or government monitors. While the regulatory-grade monitors that we use are well-validated, each one only measures pollution at a single place.[25] As a result, all available measures of pollution likely differ from what individuals actually breathe. This can arise from individual differences in indoor air, daily mobility, and activities, or from the interpolation between geography-based measures required to develop individual-level measures.

Finally, new dementia diagnoses are only measured for those individuals who survive until the end of the model's time period. This could induce a correlation between $durPM_{i,2013}$ and e_i among survivors if latent health that determines survival is (conditionally) correlated with latent health that affects the probability of a new dementia diagnosis.

B. Identification and Estimation

Relevant omitted variables, measurement error, and sample selection mean that estimating Equation (1) under the assumption that $durPM_{i,2013}$ and e_i are independent is unlikely to yield a consistent estimate of α. We use a two-pronged approach to overcome these challenges. First, to address omitted variables and measurement error in $durPM_{i,2013}$, we leverage the conditional variation in $durPM_{i,2013}$ across individuals that was induced by Clean Air Act regulations as described above. Second, to address selection based on survival, we employ a selection-correction approach.

i. Instrumenting for Pollution

As discussed in Section III, $PM_{2.5}$ regulations led to lower levels of $PM_{2.5}$ over

[25] The federal regulatory-grade monitors that we use for our analysis represent the best available information on ambient $PM_{2.5}$ in the US. Appendix B provides further information on EPA's approach to validating $PM_{2.5}$ measurements.

2004-2013 for people living in nonattainment counties relative to people in attainment counties in the same CBSA and the same levels of $PM_{2.5}$ over 2001-2003. The EPA solely relied on 2001-2003 to make its nonattainment designations. This is the essence of the quasi-experiment that we rely on to isolate conditionally exogenous variation in $durPM_{i,2013}$. More formally, we isolate this variation using a control-function approach with a vector of instruments, Z_i. The five elements of Z_i include an indicator for residing in a nonattainment county in 2004 and interactions between this indicator and $f(basePM_i)$. This set of instruments is designed to capture the between- and the within-county variation in decadal $PM_{2.5}$ induced by the CAA, as discussed in Section III. Our "first-stage equation" is given by:

$$(2)\ durPM_{i,2013} = \delta_Z Z_i + \delta_X X_i + \delta_H H_i + \delta_W W_i + \delta_C C_{i,2013} + f(basePM_i;\ \delta_{basePM}) + \varepsilon_i,$$

where the covariates other than Z_i are the same as in Equation (1).

We assume that (e_i, ε_i) is distributed jointly normal with mean zero and var(e_i) normalized to one, and is independent of the instruments, Z_i, and controls, $X_i, H_i, W_i, C_{i,2013}$, and $f(basePM_i)$.[26] Under this assumption, the order condition is satisfied, as the controls are exogenous and can serve as instruments for themselves, while the scalar $durPM_{i,2013}$ is treated as endogenous and is instrumented with Z_i.

We denote the residuals from an estimation of Equation (2) via OLS as $\hat{\varepsilon}_i$. Following Rivers and Vuong (1988), these residuals are then added as an additional control to Equation (1), which is then treated as a standard probit model and estimated using Maximum Likelihood.[27]

[26] While assuming joint normality is standard in this class of models, Rivers and Vuong (1988) note that it is actually stronger than the sufficient condition that e_i is normal and homoscedastic given ε_i, the instruments, Z_i, and controls, $X_i, H_i, W_i, C_{i,2013}, basePM_i$. We also assume that the technical assumptions of Rivers and Vuong hold, namely that the data are i.i.d. and the parameter vector lies in the interior of a compact, convex subset of Euclidean space.

[27] The Rivers and Vuong (1988) approach estimates a scaled version of the parameters in Equation (1) where the scaling

The equivalence of control-function estimation in linear models and two-stage least squares (2SLS) is well established (e.g., Hausman 1978). In non-linear models like ours, the estimators are not equivalent, but the intuition of 2SLS remains applicable. This gives rise to the term Two-Stage Conditional Maximum Likelihood (2SCML) that Rivers and Vuong (1988) use to describe the approach that we rely on. Our 2SCML approach requires the standard conditions for consistency of the 2SLS estimator, i.e., that the controls are exogenous, that the instruments, Z_i, are partially correlated with $durPM_{i,2013}$, and that the instruments, Z_i, are exogenous.[28]

The mean-independence assumption that guarantees exogeneity of the controls, i.e., $E\left[e_i|X_i, H_i, W_i, C_{i,2013}, f(basePM_i)\right] = E\left[\varepsilon_i|X_i, H_i, W_i, C_{i,2013}, f(basePM_i)\right] = 0$, is equivalent to the assumption that the functional forms specified in the decomposition of η and in Equation (2) are sufficiently flexible to capture the relationships between the controls and η_i and the controls and $durPM_{i,2013}$.[29] Three features of our research design support the credibility of this functional-form assumption. First, as discussed in Section II, our controls are extensive. Second, our model is saturated within some control vectors (e.g., integer-age-by-gender dummies and the full-factorial of baseline health conditions) and flexible in other control vectors (e.g., fourth order polynomial functions of medical spending and baseline pollution). Third, the estimated AMEs are relatively insensitive to adding additional interactions and additional flexibility in unsaturated control vectors.[30]

The first condition on the instruments, Z_i, (relevance) can be directly validated

factor depends on the variance of ε_i and the covariance between ε_i and e_i. While the unscaled coefficients can be recovered, this isn't necessary. As discussed in Wooldridge (2015), the scaled coefficients are sufficient for estimating the average structural function (Blundell and Powell 2013) and the AME of $durPM_{i,2013}$ on $Prob(\Delta y_i = 1)$.

[28] In a linear model, consistency requires that the controls and instruments are uncorrelated with the error. We are estimating a Probit model which requires the stronger assumptions of independence and normality.

[29] A necessary condition for Z_i to be a valid instrument for $durPM_{i,2013}$ is conditional independence, i.e., that Z_i, is independent of η_i, conditional on the controls. Combining this conditional independence assumption with the additional assumption that (e_i, ε_i) is mean independent of the controls is then sufficient for (e_i, ε_i) to be mean independent of both Z_i and the controls.

[30] See, for example, the discussions in Sections IV.C, IV.D, VI.B, and Appendix J.

with empirical testing, while the second condition (exogeneity) cannot be. A violation of the key identifying assumption of exogeneity would mean that some unobserved factor remaining in e_i causes individuals of the same age, race, sex, and baseline health who experienced the same residential $PM_{2.5}$ concentrations across 2001-2003 and lived in neighborhoods with the same socioeconomic conditions, nevertheless sorted into attainment versus nonattainment counties within the same CBSA on the basis of factors associated with different probabilities of receiving a new dementia diagnosis from 2005-2013 and yet did not have dementia prior to 2005. We follow prior studies and assume that nonattainment status is independent of measurement error in $PM_{2.5}$ exposure in counties that contain air pollution monitors (Chay and Greenstone 2005, Isen et al. 2017).

We consider the earlier-in-life exposure that, as previously discussed, is an element of e_i. The EPA nonattainment designations relied only on 2001-2003 concentrations and we include a flexible (fourth-order polynomial) function of $basePM_i$ (created using data from 2001-2003) in our empirical models. Thus, earlier-in-life exposure would bias our estimate of α only in the unlikely event that earlier-in-life exposure is not independent of nonattainment status conditional on baseline pollution and other controls. We provide support for the exclusion restriction assumption in Section VI by estimating a model that includes a measure of earlier-in-life exposure. While the coefficient on earlier-in-life exposure itself is uninformative for evaluating the 2SCML assumptions, the fact that the estimates of the AMEs are invariant to its inclusion suggests that the omission of earlier exposure is not biasing our estimated effect of interest.

To conclude, like 2SLS estimators, our key identifying assumption is that the error in Equation (1) is independent of our instrument, Z_i. This is likely to hold given our extensive set of controls and the sharply defined timeframe used by the EPA to make regulatory designations. We provide support for this assumption in Section VI.

ii. Addressing Selection on Mortality

Prior work has found that $PM_{2.5}$ causes mortality among seniors in the US (Di et al. 2017, Deryugina et al. 2019). For example, Deryugina et al. uses an instrumental-variables estimator to conclude that a one-day 1 $\mu g/m^3$ increase in $PM_{2.5}$ causes a 0.18% increase in mortality over three days. When we estimate the specification shown in Equations (1)-(2) but with decadal mortality as the dependent variable, we find that a 1 $\mu g/m^3$ increase in average $PM_{2.5}$ from 2004 through 2013 increases mortality by 2.47 pp, equivalent to 6% of the decadal mortality rate.[31]

These results, combined with the concern that unobserved aspects of health that determine survival may be correlated with unobserved aspects of health that determine dementia, suggest that sample selection may bias the estimates of Equations (1) and (2) when not accounting for selection on mortality. For example, suppose that unobserved aspects of health that determine survival are negatively correlated with unobserved aspects of health that determine dementia, i.e., sicker people who are more likely to die are also more likely to be diagnosed with dementia if they live. In this case, selection would bias downward the estimate of $PM_{2.5}$'s direct effect on dementia in the selected sample.[32] This would mean that our estimate of α when ignoring selection would capture both the causal effect of $PM_{2.5}$ on dementia (our object of interest) plus a compositional effect based on the set of survivors at the end of the decade.[33]

To address this selection issue, we obtain a selection-corrected estimate using a control-function approach (Heckman 1979, Heckman and Robb 1986). To implement this approach, we require an additional set of instruments.[34] In particular, the

[31] Appendix Table I1 provides the estimated effects of decadal $PM_{2.5}$ on mortality, i.e., an estimation of Equations (1) and (2) with mortality as the outcome in Equation (1).
[32] A less intuitive, but nonetheless possible, concern would be that the unobserved health determining survival was positively correlated with the unobserved health determining dementia, causing an upward bias in our estimate.
[33] Lee (2009) discusses this concept in detail in the context of a randomly assigned job-training program that affects whether individuals work and the level of their subsequent wages.
[34] In Appendix H, we show a Lee (2009) bounds approach that does not require these additional instruments, M_i, but does employ the CAA ones, Z_i, as described above.

relevance and validity conditions require that the additional instruments are correlated with decadal survival but are independent of the unobserved determinants of dementia. The medical literature provides such a set of diagnoses that affect survival but do not affect dementia: prior diagnoses of a subset of non-smoking related cancers, which are found to be unrelated to dementia outcomes (Driver et al. 2012, Ganguli 2015). To form the selection-correcting control function, we begin by estimating via Maximum Likelihood a probit model of decadal survival, S_i, with the same covariates as Equation (2) plus the vector of additional instruments, M_i. We do this by specifying a latent survival propensity

$$(3)\ S_i^* = \gamma_Z Z_i + \gamma_X X_i + \gamma_H H_i + \gamma_W W_i + \gamma_C C_{i,2013} + f(basePM_i; \gamma_{basePM}) + \gamma_M M_i + u_i$$

such that $S_i = 1[S_i^* > 0]$.

In addition to the functional-form assumptions in Equation (3), we now assume that $(e_i, \varepsilon_i, u_i)$ is distributed jointly normal and is independent of the instruments, Z_i, the instruments, M_i, and controls, $X_i, H_i, W_i, C_{i,2013}$, and $basePM_i$. We define M_i to include indicators for baseline diagnoses of non-smoking-related cancers (leukemia, lymphoma, and cancers of the breast, prostrate, colon, rectum, and endometrium) from the CMS's Chronic Conditions Data Warehouse file. These seven cancers, which affect decadal survival, are assumed to be independent of latent features of health that affect the probability of a dementia diagnosis.[35] We then use the generalized residuals of Equation (3), denoted $\hat{v}_i$, to define an additional control

[35] A potential concern is that non-smoking related cancers, while not causing dementia, could be correlated with dementia through other omitted factors. For example, a competing-risks framework could lead to a negative correlation between non-smoking related cancers and latent health affecting dementia and lead to an upward-biased estimate of α in our selection-correction model. Such a framework would likewise suggest that estimating Equation (1) adding only the CAA-based control function would provide a downward-biased estimate. On this basis, one could interpret non-smoking related cancers as "imperfect instruments," as defined by Nevo and Rosen (2012), and use them to partially identify α. The estimated identification region would then simply be the interval between the two estimates.

that we include in Equations (1) and (2).[36]

To summarize, our estimation proceeds in three steps. The first step is to estimate Equation (3) via Maximum Likelihood and create the generalized residuals, $\hat{v}_i$. The second step is to include $\hat{v}_i$ as an additional control in Equation (2), estimate Equation (2) via OLS, and recover the residuals, $\hat{\varepsilon}_i$. The final step is to include functions of $\hat{\varepsilon}_i$ and $\hat{v}_i$ as additional controls in Equation (1). We show this version of Equation (1) that includes the additional controls in Equation (4), which we estimate via Maximum Likelihood:

$$(4)\quad \Delta y_i^* = h\left(durPM_{i,2013}; \alpha_i\right) + \beta_X X_i + \beta_H H_i + \beta_W W_i + \beta_C C_{i,2013} + f(basePM_i; \beta_{basePM}) + \beta_{CF} CF_i + \tilde{e}_i$$

where $\tilde{e}_i = e_i - \beta_{CF} CF_i$. CF_i denotes the control-function vector created with the generalized residuals from the estimation of Equation (3) and the residuals from the estimation of Equation (2). We set $CF_i = [\hat{\varepsilon}_i\ \hat{\varepsilon}_i^2\ \hat{v}_i\ \hat{v}_i^2]$.[37] Because we estimate $\hat{\varepsilon}_i$ and $\hat{v}_i$ in prior stages, we bootstrap standard errors over all three regressions, clustering at the Census block-group level to allow for spatial correlation in diagnoses.[38]

C. Allowing for Heterogeneity in Covariates

In the simplest specification of the decadal model, we specify $h\left(durPM_{i,2013}; \alpha_i\right) = \alpha\ durPM_{i,2013}$. However, we also estimate specifications that allow $durPM_{i,2013}$ to enter flexibly as a fourth-order polynomial and that allow

[36] Generalized residuals are defined as $\hat{v}_i = S_i\lambda\left(\widehat{S^*}\right) - (1 - S_i)\lambda\left(-\widehat{S^*}\right)$, where $\lambda(\cdot) = \phi(\cdot)/\Phi(\cdot)$, ϕ and Φ are the standard normal density and CDF, respectively, and $\widehat{S^*} = \hat{\gamma}_Z Z_i + \hat{\gamma}_X X_i + \hat{\gamma}_H H_i + \hat{\gamma}_W W_i + \hat{\gamma}_C C_{i,2013} + f(basePM_i; \hat{\gamma}_{basePM}) + \hat{\gamma}_M M_i$. By construction, S_i = 1 for all observations used in the estimation of Equations (1) and (2), therefore, for these observations, $\hat{v}_i = \lambda\left(\widehat{S^*}\right)$, simplifying to the familiar inverse Mills ratio used in Heckman (1979).

[37] In alternative specifications, e.g., Columns (3) and (2) of Table I, we consider a less flexible control function that only includes $\hat{\varepsilon}_i$ and $\hat{v}_i$, without their squares, as well as a version with only $\hat{\varepsilon}_i$, which controls for the type of endogeneity described in Section IV.B.i, but not selection on mortality.

[38] Our instruments vary within Census blocks across ZIP+4 codes. We alternatively cluster at the courser county level and find almost no impact on our results.

for interactions between $durPM_{i,2013}$ and the vectors X_i, H_i, W_i, and CF_i by specifying:[39]

$$(5)\ h\left(durPM_{i,2013};\alpha_i\right) = \alpha_1\, durPM_{i,2013} + \alpha_2 durPM_{i,2013}^2 + \alpha_3\, durPM_{i,2013}^3 + \alpha_4\, durPM_{i,2013}^4 + \alpha_X\, X_i\, durPM_{i,2013} + \alpha_H\, H_i\, durPM_{i,2013} + \alpha_W\, W_i\, durPM_{i,2013} + \alpha_{\mathrm{CF}}\, CF_i\, durPM_{i,2013}\ .$$

In this approach, the effect of $durPM_{i,2013}$ on the latent propensity to be newly diagnosed with dementia is allowed to vary flexibly with both the level of $durPM_{i,2013}$, and the levels of individual characteristics, neighborhood characteristics, and control function variables.[40,41]

D. Allowing $PM_{2.5}$'s Effect to Vary with Exposure Duration

The contemporaneous model described in Section IV.A and IV.B is both parsimonious and comparable to the existing literature on the impacts of pollution exposure on health outcomes. However, as the treatment is measured as the average $PM_{2.5}$ exposure from 2004-2013, and a dementia diagnosis can happen at any point between 2005 and 2013, there could be an aggregation bias if the data were systematically misaligned; for example, if the AME were driven by spatial correlation between dementia diagnoses in 2010 and pollution levels in 2013. The potential for misalignment due to temporal aggregation is a universal feature of research on pol-

[39] See Blundell and Powell (2003, 2004) for a discussion of estimating non-parametric, binary-response models with endogenous regressors.

[40] The fact that the effect of $durPM_{i,2013}$ on new dementia diagnosis is allowed to vary with $CF_i = [\hat{\varepsilon}_i\ \hat{\varepsilon}_i^2\ \hat{v}_i\ \hat{v}_i^2]$ means that this approach nests the correlated random-coefficients model of Garen (1984) with additional assumptions. Specifically, if there exist random coefficients that satisfy the linear conditional expectation assumption of Garen (1984), they will be accounted for in our analysis. Under these assumptions, we do not find evidence of bias coming from correlated random coefficients in one's sensitivity to pollution exposure.

[41] Following Rivers and Vuong (1988) and Wooldridge (2015), once the control function, CF_i, is included in Equation (4), $durPM_{i,2013}$ is independent of $\tilde{e}_i$ and, therefore, the non-linear functions of $durPM_{i,2013}$ in Equation (5) are also independent of $\tilde{e}_i$. And, as we had assumed that the controls are independent of $\tilde{e}_i$, the interaction terms in (5) are also independent of $\tilde{e}_i$. Adding the 115 additional functions of the single endogenous economic variable, $durPM_{i,2013}$, has little impact on the results as shown in Columns (4) and (5) of Table I.

lution and health due to the inability to measure pollution and health instantaneously.

In this section, we extend the analysis in three ways. First, we define the outcome measure to be a new dementia diagnosis during a single year $t = [2005,2013]$, thus avoiding the aggregation bias that could be introduced by misalignment of the data at the decadal level. Second, we only condition on surviving until the end of year *t*, thus incorporating the effects of $PM_{2.5}$ exposure on dementia for individuals who die prior to 2013. Finally, by estimating the model separately for each year, we allow for year-specific variation in all model coefficients. This additional flexibility allows the effect of nonattainment status on $PM_{2.5}$ exposure to evolve during the years after nonattainment designations were made. Moreover, it allows the effect of $PM_{2.5}$ exposure on the probability of a new dementia diagnosis to evolve with the duration of exposure. In principle, such differences could arise from biological mechanisms linking $PM_{2.5}$ to dementia, or from changes in the composition of people surviving from one year to the next.

Equations (6) and (7) describe the analogues to Equations (4) and (5), respectively, where $\Delta y_{i,t}^{*}$ now denotes the latent propensity to become newly diagnosed with dementia during year *t*. We estimate Equation (6) separately for each year 2005 to 2013 via Maximum Likelihood.

$$(6)\quad \Delta y_{i,t}^{*} = h\left(durPM_{i,t};\alpha_{i,t}\right) + \beta_{X,t}X_i + \beta_{H,t}H_i + \beta_{W,t}W_i + \beta_{C,t}C_{i,t} + f\left(basePM_i;\beta_{basePM,t}\right) + \beta_{CF,t}CF_{i,t} + \tilde{e}_{i,t}$$

$$\text{where}\quad \tilde{e}_{i,t} = e_{i,t} - \beta_{CF,t}CF_{i,t},$$

$$(7)\quad h\left(durPM_{i,t};\alpha_{i,t}\right) = \alpha_{1,t}\, durPM_{i,t} + \alpha_{2,t} durPM_{i,t}^{2} + \alpha_{3,t}\, durPM_{i,t}^{3} + \alpha_{4,t}\, durPM_{i,t}^{4} + \alpha_{X.t}\, X_i\, durPM_{i,t} + \alpha_{H,t}\, H_i\, durPM_{i,t} + \alpha_{W,t}\, W_i\, durPM_{i,t} + \alpha_{CF,t}\, CF_{i,t}\, durPM_{i,t},$$

and $CF_{i,t}$ denotes a control-function vector, $[\hat{\varepsilon}_{i,t}\ \hat{\varepsilon}_{i,t}^2\ \hat{v}_{i,t}\ \hat{v}_{i,t}^2]$, created with the generalized residuals from the estimation of Equation (8) (the analogue to Equation (3)) and the residuals from the estimation of Equation (9) (the analogue to Equation (2)):

$$(8)\quad S_{i,t}=1\big(\gamma_{Z,t}Z_i+\gamma_{X,t}X_i+\gamma_{H,t}H_i+\gamma_{W,t}W_i+\gamma_{C,t}C_{i,t}+f\big(basePM_i;\gamma_{basePM,t}\big)+\gamma_{M,t}M_i+u_{i,t}>0\big),$$

$$(9)\quad durPM_{i,t}=\delta_{Z,t}Z_i+\delta_{X,t}X_i+\delta_{H,t}H_i+\delta_{W,t}W_i+\delta_{C,t}C_{i,t}+f\big(basePM_i;\delta_{basePM,t}\big)+\hat{v}_{t,i}+\varepsilon_{t,i}.$$

We begin by estimating Equation (8) via Maximum Likelihood on the full sample of individuals. The survival outcome, $S_{i,t}$, now indicates whether individual *i* is still alive through the end of year *t* and has not previously received a dementia diagnosis. We then estimate the year-*t*-specific pollution Equation (9) via OLS. This equation includes the generalized residuals from the survival function, $\hat{v}_{i,t}$. Equations (9) and (6) are estimated using the subset of people who are still alive through the end of year *t* and had not been diagnosed with dementia prior to year *t*.

We then use the year-t-specific parameter vector, $\hat{\alpha}_t$, to calculate AME_t, the average effect of a marginal increase in $PM_{2.5}$ exposure from 2004 through year *t* on the probability of receiving a new dementia diagnosis during year *t*. We additionally calculate the cumulative effect of $PM_{2.5}$ exposure from 2004 through year *t* on new dementia diagnoses during that period according to,

$$(10)\quad cumulativeAME_t=\sum_{s=2005}^{t}\left(\frac{pop_s}{pop_{2005}}\right)AME_s,$$

by summing the year-specific average marginal effects, after weighting them by their corresponding shares of the original population to account for attrition due to

dementia and death.[42] Finally, we bootstrap standard errors on $cumulativeAME_t$ by repeating estimation of Equations (6)-(10) after resampling from the original population one thousand times with replacement and clustering at the Census block-group level.

V. Results

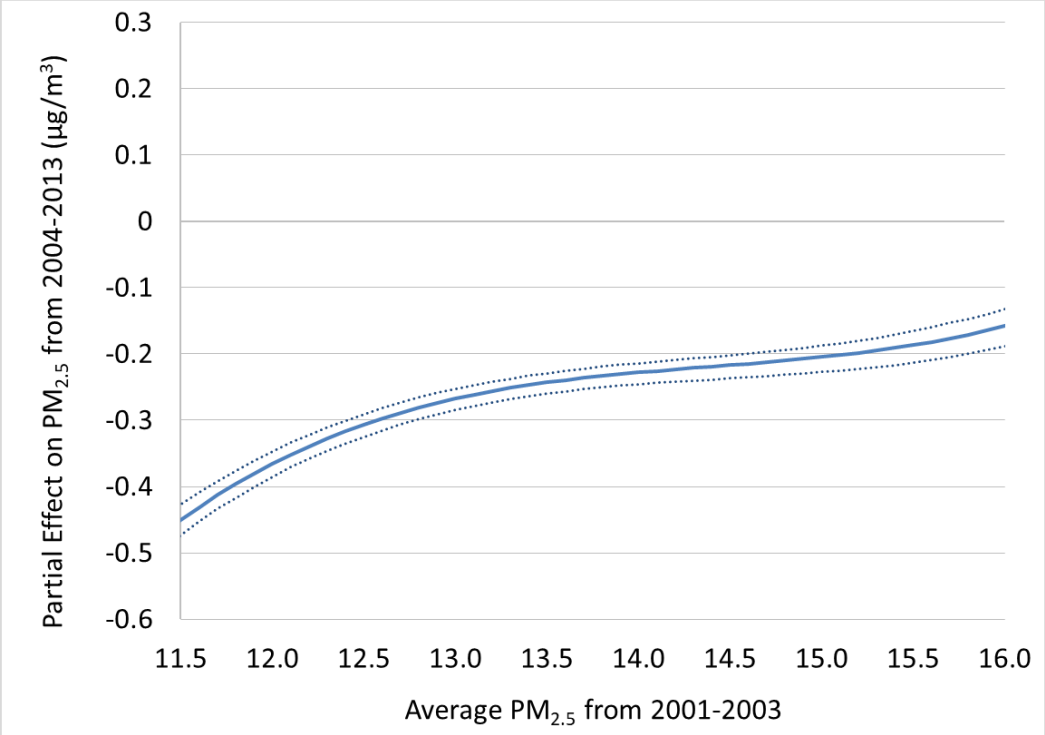
A. $PM_{2.5}$ Regulations Created Conditional Differences in Subsequent $PM_{2.5}$

The identifying variation for our estimator comes from the fact that the EPA's nonattainment designations created quasi-random differences in $durPM_{i,t}$ for $t = [2005,2013]$, conditional on $basePM_i$ and the additional controls in Equations (2) and (9). Figure IV shows this identifying variation for the year 2013. Specifically, it uses the coefficients on the instruments from the year-2013 version of Equation (9) to plot the estimated partial effect of nonattainment on $durPM_{i,2013}$ across levels of $basePM_i$. Similar figures plotting the estimated partial effect for $t = [2005,2012]$ versions of Equation (9), as well as the decadal version in Equation (2), are shown in Appendix I1. Intuitively, the partial effects are negative, showing that nonattainment status reduced pollution. In addition, as permitted (but not determined) by our construction of Z_i, the partial effects vary with baseline $PM_{2.5}$. This yields within-county identifying variation in $durPM_{i,t}$ in all years.[43] The first-stage partial R^2 of the identifying instruments is 0.047 and the F statistic is 489 for the regression underlying Figure IV, suggesting that any finite sample bias is negligible. The size of the F statistic reflects the number of observations (approximately 1 million) and number of Census block group clusters (approximately 140 thousand).

[42] For example, we multiply the estimated AME in 2009 by 0.65 because 65% of the original year-2005 sample survives to the end of 2009. This adjusts for the progressive decline in sample size due to dementia and mortality.

[43] While nonattainment status caused reductions in $durPM_{i,t}$ at all levels of $basePM_i$, these reductions are larger at lower baseline levels.

FIGURE IV: ESTIMATED PARTIAL EFFECT OF NONATTAINMENT ON $PM_{2.5}$ EXPOSURE 2004-2013, BY BASELINE CONCENTRATIONS 2001-2003



Note: The figure shows the average effect of the county-level nonattainment designation on the average individual-level conditional change in $PM_{2.5}$ concentrations over the period 2004-2013. The zero line represents individuals living in attainment counties at the same baseline $PM_{2.5}$ concentration and holding all else in Equation (9) constant. The dotted lines denote 95% confidence bands constructed from 1,000 bootstrap replications, with clustering at the Census block group.

B. The Effect of $PM_{2.5}$ on Dementia

We find that a 1 $\mu g/m^3$ increase in average $PM_{2.5}$ concentrations starting in 2004 increases the probability of receiving a new dementia diagnosis before the end of 2013 by an average of 2.15 percentage points (pp). To illustrate the importance of various aspects of our identification strategy, we present the AME of cumulative $PM_{2.5}$ exposure over the decade on new dementia diagnoses from six specifications described in Section IV.

The first column of Table I begins with a simple, associative model of decadal $PM_{2.5}$ and dementia diagnosis over the decade. The next four columns retain the

contemporaneous, decadal specification and incrementally address potential confounders that may underlie this association, as previously discussed. The final column presents our preferred specification that aggregates year-by-year marginal effects over the decade while addressing all of the potential confounders described in Section IV. In all cases, the AMEs are scaled to represent percentage point (pp) changes in the probability of receiving a new dementia diagnosis by the end of 2013.

TABLE I—AVERAGE MARGINAL EFFECT OF CUMULATIVE $PM_{2.5}$ ON THE PROBABILITY OF A NEW DEMENTIA DIAGNOSIS

	(1)	(2)	(3)	(4)	(5)	(6)
(1 μg/m^3 increase in decadal $PM_{2.5}$)	0.629***	0.124	1.545***	2.283***	2.384***	2.151***
	(0.058)	(0.105)	(0.536)	(0.565)	(0.568)	(0.846)
individual & neighborhood covariates		x	x	x	x	x
$PM_{2.5}$ control function			x	x	x	x
survival control function				x	x	x
polynomial functions and interactions					x	x
heterogeneity by exposure duration						x
F-statistic on $PM_{2.5}$ instruments			496	498	498	165 to 489
number of individuals: dementia function	1,179,094	1,179,094	1,179,094	1,179,094	1,179,094	989,751 to 2,293,270
Chi-square statistic on survival instruments				3,813	3,813	1,166 to 2,274
number of individuals: survival function				2,439,904	2,439,904	2,439,904

Note: The outcome is scaled to equal 100 if an individual was diagnosed with dementia and 0 otherwise. By 2013, 20% of the individuals in our sample who were alive in that year had been diagnosed with dementia. In Column (1) the covariates are $PM_{2.5}$ and CBSA dummies. Column (2) adds covariates for baseline health in 2004, individual demographics, demographics for the individual's Census block group, and pre-regulatory (2001-2003) $PM_{2.5}$ levels at their residence. Column (3) adds a control function for $PM_{2.5}$. Column (4) adds a control function for survival. Column (5) adds additional polynomial functions of covariates. Column (6) reports a cumulative decadal AME that aggregates year-specific AMEs, along with ranges for the year-specific F-statistics, Chi-square statistics, and sample sizes. Year-specific estimates are reported in Table I5. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels using robust standard errors clustered at the block group. Standard errors in Columns (3) through (6) are bootstrapped using 1,000 repetitions.

Column (1) in Table I shows the result from a simple associative model of decadal $PM_{2.5}$ and dementia diagnosis over the decade. The only covariates are CBSA

dummies. The result indicates that a 1 μg/m^3 increase in average decadal $PM_{2.5}$ is associated with a 0.63 pp higher probability of receiving a dementia diagnosis between 2005 and 2013.

Column (2) then additionally includes the observed characteristics represented by X_i, H_i, W_i and $f(basePM_i)$ in Equation (1). Adding these covariates reduces the conditional association between measured decadal $PM_{2.5}$ and dementia over the decade to 0.12 pp. Thus, most of the within-CBSA association between measured $PM_{2.5}$ and new dementia diagnoses can be explained by people with observably higher baseline risks of dementia living in more polluted neighborhoods. Notably, 99% of the decline that occurs as we move from Column (1) to Column (2) can be explained by the inclusion of X_i, H_i, and W_i. When all of these covariates are included, adding $f(basePM_i)$ only reduces the AME for $PM_{2.5}$ exposure from 2004-2013 by 1%. This shows that our extensive measures of individual demographics, baseline health, and neighborhood characteristics explain almost all of the heterogeneity that contributes to any association between neighborhood $PM_{2.5}$ in 2001-2003 and new dementia diagnoses.

Column (3) adds the $PM_{2.5}$ control function to address measurement error in pollution exposure, or any residual differences driven by sorting. The resulting order-of-magnitude increase in the AME relative to Column (2) is unsurprising. First, our extensive set of geographic controls could potentially exacerbate the effect of any measurement error in pollution. Second, while the bias introduced by measurement error is ambiguous in general, prior studies have consistently found that instrumenting for (shorter-term) measures of air pollution exposure results in order-of-magnitude increases in estimates for its effects on other morbidities and mortality among older adults (see, for example, Schlenker and Walker 2016, Deschênes,

Greenstone, and Shapiro 2017, and Deryugina et al. 2019).[44]

Column (4) adds the survival control function to address selection on mortality.[45] Controlling for selection on survival increases the AME to 2.28 pp, a 48% increase relative to Column (3). This increase is consistent with classic selection bias caused by positively correlated latent health: individuals who were more likely to die were also more likely to develop dementia.[46]

Column (5) shows the AME from our specification shown in Equations (4)-(5) that allows for additional parametric flexibility in the covariates.[47] This only increases the AME to 2.38 pp, which is about a 4% increase relative to Column (4).[48]

The final AME shown in Column (6) shows the cumulative AME at the end of the decade as shown in Equations (6) through (10). This model differs from the model underlying the AME in Column (5) in three potentially important ways. First, it limits aggregation bias that could be introduced by the misalignment of the data at the decadal level. Second, it incorporates the effects of $PM_{2.5}$ exposure on dementia for people who die during the decade, almost doubling the number of observations used in estimation. Finally, it allows the effect of $PM_{2.5}$ exposure on the probability of a new dementia diagnosis to evolve with the duration of exposure, as shown in Equation (6).

This cumulative AME indicates that a 1 $\mu g/m^3$ increase in average $PM_{2.5}$ increases the cumulative probability of a new dementia diagnosis by the end of 2013 by 2.15 pp. Comparing this cumulative AME against the results from the more parsimonious model in Column (5) indicates that the three notable differences between

[44] These studies find that instrumenting for air pollution increases their estimates for its effects on morbidity and mortality by factors ranging from 6 to 20. The twelve-fold increase in our Table I estimates sits near the middle of this range.
[45] The average marginal effects of the survival instruments are reported in Appendix Table I2.
[46] We build on this result and develop a partial-identification approach to exploring the role of selection on survival in Appendix H.
[47] Appendix Table I3 reports the full results from this specification. Appendix Table I4 compares the AME for $PM_{2.5}$ from this specification to the AMEs that we estimate for other dementia risk factors that were included as covariates in the model. Note that we do not consider the coefficients on risk factors other than decadal $PM_{2.5}$ to reflect a causal relationship.
[48] When we run this specification using a linear-probability model, we find an AME of 2.16 pp that is statistically significant the 1% level.

the two approaches yields only a small difference in the economic magnitude of their estimated effects (0.23 pp).

To provide context for the AME of 2.15pp, a 1 μg/m^3 change is equivalent to 9.1% of the average person's exposure between 2004 and 2013 and 59% of a standard deviation. A 2.15 pp change in the dementia diagnosis rate is a 11% increase relative to the diagnosis rate among people in our sample who survive to the end of 2013. To provide an age-based comparison to this statistic, the dementia diagnosis rate in 2013 was 2.2 pp higher among 80-year-old women compared with 79-year-old women (Figure I).[49]

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

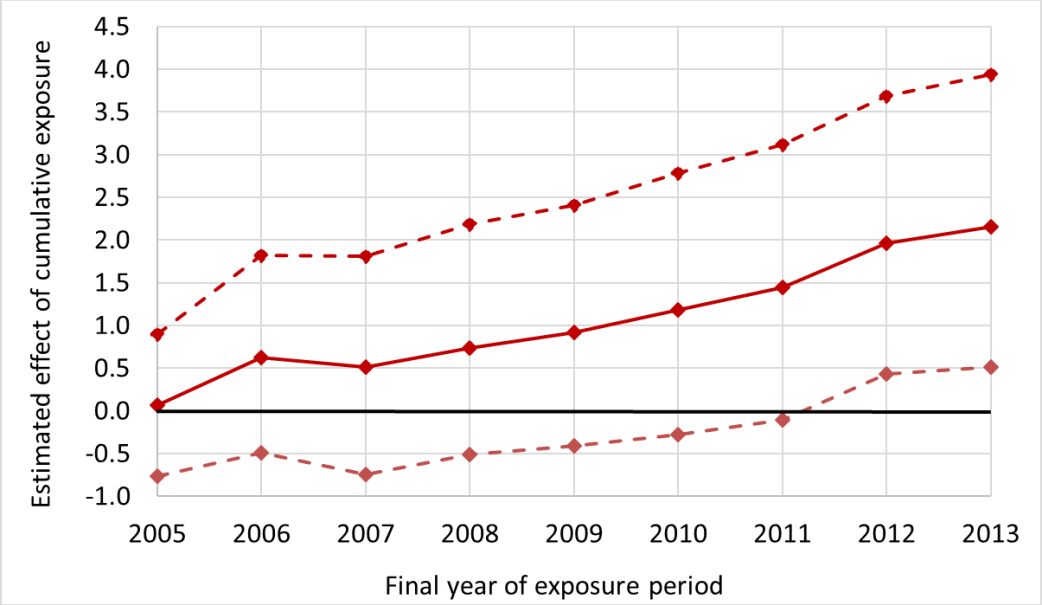


Figure V shows how our estimates of the cumulative AME evolve over time,

[49] To compare these results to earlier medical literature, Gatz et al. (1997) finds that approximately 74% of Alzheimer's disease cases are heritable using twin pairs. We impose the additive separability assumption underlying that statistic and perform a back-of-the-envelope calculation to see how much variation in new dementia diagnoses could be explained by decadal $PM_{2.5}$ exposures after age 65 in our sample. Specifically, we use a linearized and additively separable version of our decadal model to calculate $((AME^2\ Var(durPM))/(Var(\Delta y)) \approx 1\%$, where AME=0.0238 (this number is multiplied by 100 when discussed in the text), Var(durPM)=2.8812, and Var(Δy)=0.1572. We thank an anonymous referee for this suggestion.

along with 95% bootstrapped confidence intervals. The underlying year-specific AMEs are presented in Appendix Table I5. While the year-specific AMEs are imprecisely estimated, the AME for 2005 is close to zero and, starting in 2008, the year-specific AMEs are positive in each year, which is reflected in the increasing cumulative AME shown in Figure V. In addition, the year-specific AMEs are generally increasing in the duration of exposure. When we weight the year-specific AMEs by the surviving share of the baseline population to account for attrition, as shown in Equation (10), the resulting weighted year-specific AMEs become similar in magnitude. This similarity is reflected in the approximately-linear trend in cumulative AME point estimates shown in Figure V, although visual inspection of the confidence intervals suggests that we lack the statistical power to rule out a nonlinear function.

C. Heterogeneity in Effects

The results shown in Column (6) of Table 1 average over considerable heterogeneity in the marginal effects of $PM_{2.5}$ exposure. Interestingly, the cumulative AME's tend to be larger among individuals who experienced lower levels of $PM_{2.5}$. To illustrate this, we divide individuals into terciles by their baseline residential exposures during 2001-2003. Individuals in the top tercile of baseline exposure (above 14.2 $\mu g/m^3$) experienced a cumulative AME of 1.91 pp. In comparison, individuals in the middle tercile (whose baseline exposures were between 12.4 and 14.2 $\mu g/m^3$) experienced an AME of 2.10 pp. Individuals with baseline exposures below 12.4 $\mu g/m^3$ experienced an AME of 2.45 pp. For those in the top, middle and bottom terciles who survived through 2013, average exposures from 2004-2013 were 12.46, 11.14 and 9.24 $\mu g/m^3$, respectively. These results highlight that the effects of $PM_{2.5}$ on dementia persist well below the current US regulatory threshold of 12 $\mu g/m^3$ of annual average concentrations.

The estimates also show heterogeneity across individual characteristics. For example, the cumulative AME is larger for individuals whose exposures we observe at older ages (e.g., 1.13 pp for people born in 1938 for whom we observe quasi-random variation in exposure from age 66 up to age 75, compared with 2.34 pp for people born in 1928 for whom we observe variation in exposure from age 76 up to age 85). Conditional on age at exposure, the AME is higher for women compared with men (e.g., 2.57 pp for women born in 1928 compared with 2.03 pp for men born in 1928). Conditional on age and sex, the AME is higher for individuals with more clinical risk factors for dementia at the start of the decade (e.g., 2.24 pp for women born in 1928 with no baseline clinical risk factors compared with 2.61 pp for women born in 1928 who had been diagnosed with ischemic heart disease and hypertension at baseline). Finally, when we condition on $PM_{2.5}$ exposure, the AME is higher among individuals denoted by CMS as "Black or African-American" compared with "non-Hispanic White" (e.g., 0.21 pp higher among women born in 1928 whose baseline exposure to $PM_{2.5}$ was within a one-unit window of the sample median of 13.4 $\mu g/m^3$).[50]

VI. Main Validation Tests and Additional Sensitivity Analysis

A. Main Validation Tests

Table II presents three validation tests of our estimator. First, we assess the assumption that our nonattainment instrument is independent of earlier-in-life measures of $PM_{2.5}$, conditional on baseline $PM_{2.5}$ exposure and the other covariates. Specifically, we examine whether the AME shown in Table I, Column (6) changes when we add measures of earlier-in-life exposures, specifically average annual $PM_{2.5}$ in 1999 and 2000.[51] These are the first two years that the US EPA had a

[50] Average decadal $PM_{2.5}$ exposures in our estimation sample were 6% higher for Black or African-American individuals compared with non-Hispanic White individuals who survived through 2013.
[51] In the years of 1999 and 2000, 86% of our balanced panel of monitors were in operation.

national network of $PM_{2.5}$ monitors and the first two years that researchers can obtain administrative data describing the Medicare population. Thus, this validation test exhausts the available data. For the 23% of our sample that were under age 65 in those years and not yet enrolled in Medicare, we assign 1999 and 2000 $PM_{2.5}$ exposures based on the location where we first observe these individuals living upon enrolling in Medicare. While this assignment is imperfect, low short-term migration rates among this age group limit the scope for error. For example, the year 2000 Census of Population reports that 77% of people aged 65-69 lived in the same residence as they did five years ago.

TABLE II—VALIDATION TESTS

	(1)	(2)	(3)	(4)
Probit model average marginal effect	2.151***	2.246***	1.754**	-0.167
(1 μg/m^3 increase in decadal $PM_{2.5}$)	(0.846)	(0.929)	(0.704)	(0.283)
modification to main specification				
control for $PM_{2.5}$ in 1999 and 2000		x		
control for other regulated air pollutants			x	
placebo outcome = dementia in 2004				x
F-statistic on $PM_{2.5}$ instruments	165 to 489	147 to 492	146 to 350	620
number of individuals: dementia function	989,751 to 2,293,270	989,751 to 2,293,270	989,751 to 2,293,270	2,734,032
Chi-square statistic on survival instruments	1,166 to 2,274	1,166 to 2,274	1,168 to 2,277	
number of individuals: survival function	2,439,904	2,439,904	2,439,904	

Note: The first column repeats our main result from Table I, Column (6) for comparison. The next three columns report results from alternative specifications that are designed to test the identifying assumptions that underlie our main specification. Asterisks indicate statistical significance at the 10% (*), 5% (**), and 1% (***) levels based on robust standard errors clustered at the Census block group. See the note to Table I and main text for further details.

If the exclusion restrictions on Z_i are valid, then adding controls for earlier-in-life $PM_{2.5}$ should not change the estimated AME of cumulative exposure over the decade. Column (2) shows that this augmented specification yields an AME of 2.25 pp. This is similar to the AME of 2.15 pp from our main specification (repeated in Column (1) for convenience). This similarity reinforces the validity of the instrument and is consistent with the EPA's nonattainment designation criteria, which relied solely on $PM_{2.5}$ concentrations in 2001-2003.

The specification in Column (3) tests whether our results are confounded by the

model's omission of air pollutants that may be co-generated with $PM_{2.5}$. Specifically, we add measures of exposure to PM_{10}, ozone, nitrogen dioxide, sulfur dioxide, and carbon monoxide. Each measure is constructed following the same procedures that we used to construct measures of cumulative $PM_{2.5}$. When we control for these ancillary pollutants, the cumulative AME for $PM_{2.5}$ remains large and precisely estimated.

Additionally, we test for sorting based on unobserved risk factors, such as genetics, that may contribute to dementia and be correlated with $PM_{2.5}$. In principle, sorting on unobserved risk factors could bias the estimator if, prior to our study period, people at a lower unobserved risk for dementia sorted themselves into neighborhoods that were more or less likely to be designated as nonattainment in the future, even conditional on baseline neighborhood $PM_{2.5}$ and the other controls. While we cannot directly test this sorting hypothesis in our main estimation sample, we can test it indirectly by extending the sample to include the people who were excluded because they were diagnosed with dementia prior to 2005. In other words, if individuals sorted themselves into future nonattainment areas based on unobserved dementia risk then we would expect to see a conditional relationship between dementia rates in 2004 and $PM_{2.5}$ exposure over the subsequent decade.[52] We test this hypothesis using a placebo specification that replaces the outcome in Equation (4) with an indicator for a dementia diagnosis in 2004. Including everyone alive in 2004, with or without dementia, increases our sample size to 2.7 million. Column (4) shows that the estimated AME is negative, close to zero, and estimated relatively precisely. This provides supporting evidence that the exclusion restriction is unlikely to be violated by initial differences in unobserved dementia risk, including unobserved genetic factors.

[52] Intuitively, under the hypothesis that people sorted into future nonattainment areas based on unobserved dementia risk, some people would have been diagnosed with dementia prior to 2005 and been dropped from our estimation sample, while others would have been diagnosed after 2005 and been included in our estimation sample.

B. Additional Sensitivity Analysis

The effect of $PM_{2.5}$ on dementia persists when we use (1) different measures of dementia such as the use of prescription drugs for the symptoms of Alzheimer's disease rather than claims-based diagnosis codes; (2) different samples that include people who select into managed care plans known as Medicare Advantage; (3) monitor-level attainment indicators rather than county-level indicators; (4) different approaches to measuring $PM_{2.5}$ exposure including expanding the set of monitors to include those not present for the entire study period, (5) a limited sample of individuals who live close to a monitor, and (6) controls for baseline pollution exposure that are even more flexible than the fourth-order polynomial function described above. We present and discuss these results in Appendix J.

Finally, we estimate models for placebo health outcomes. We examine five chronic conditions that are not known or suspected to be caused by air pollution but share similarities with dementia in terms of how they affect the body, how they are diagnosed, and how diagnosis rates are correlated with age, race, and gender. These are glaucoma, fibromyalgia, breast cancer, prostate cancer, and peripheral vascular disease.[53] Appendix Table J5 shows that we fail to reject the null hypothesis of zero effect at the 10% significance level for each of these placebos. We elaborate on these models and results in the appendix.

Our criteria for selecting placebos excluded illnesses that have previously been linked to air pollution. When we instead ignore these criteria and repeat estimation for each of the 15 most common chronic conditions among the Medicare population including those linked to pollution exposure, we find positive effects of $PM_{2.5}$ at the 5% level for two diseases besides dementia: chronic obstructive pulmonary disease (COPD) [AME = 1.79, p=0.002] and chronic kidney disease [AME = 1.15,

[53] Glaucoma is a progressive disorder with nerve degeneration that is strongly associated with age; fibromyalgia affects mood and behavior and can be difficult to diagnose; breast cancer and prostate cancer can be slow to progress and have gender-specific diagnosis rates; and peripheral vascular disease is associated with reduced blood circulation.

p=0.038].[54] These results could be interpreted as "reverse placebo tests" in the sense that positive findings may be expected based on prior cohort studies that found that long-term exposure to $PM_{2.5}$ is associated with these diseases (e.g., Guo et al. 2018).

VII. Conclusion

Dementia's global social costs continue to grow with the aging populations of many countries, causing the World Health Organization to label it a "public health priority" and the US Centers for Disease Control to describe it as a "public health crisis." Because no medical preventions or cures exist, policy discussions have focused on investment in research and health infrastructure and modifying behaviors related to smoking, diet, and exercise. Our findings reveal that air quality regulations provide another lever to policy makers to reduce the prevalence of dementia.

Beyond these policy implications, our results provide guidance for additional research on the causes and consequences of dementia. Our study establishes a causal link between long-term, later-in-life exposure to $PM_{2.5}$ and dementia, yet the precise mechanisms and causal pathways remain unknown. Research can investigate how the presence of small particulates in the brain alters cognitive function and relates to Alzheimer's disease specifically, and whether the effects differ across chemical composition, genotypes, comorbidities, stages of life, or other factors. Likewise our results can help guide efforts to study the broader link between air pollution, cognitive decline and financial decision making. Such insights can shed light on the economic costs of impaired cognition as well as the value of various approaches to mitigate these costs, whether through the provision of long-term care and long-term care insurance, support for family caregivers, financial decision support, and medical technologies.

[54] According to the Centers for Medicare and Medicaid Services (2012) the top 15 conditions ranked from most prevalent to least prevalent are high blood pressure, high cholesterol, ischemic heart disease, arthritis, diabetes, heart failure, chronic kidney disease, depression, COPD, Alzheimer's disease, atrial fibrillation, cancer, osteoporosis, asthma, and stroke.

Data Availability Statement. The Medicare data used in this study are available through the Centers for Medicare and Medicaid Services but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. The code and other data underlying this research is available on Zenodo at https://doi.org/10.5281/zenodo.7196076.

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Review of Economic Studies

Hazed and Confused: The Effect of Air Pollution on Dementia

By *Kelly C. Bishop, Jonathan D. Ketcham, and Nicolai V. Kuminoff*

ONLINE APPENDIX

Table of Contents

A. Sample Construction and Summary Statistics

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

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

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

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

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

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

FIGURE A1: SAMPLE SIZES AND TRANSITIONS FROM 2004-2013

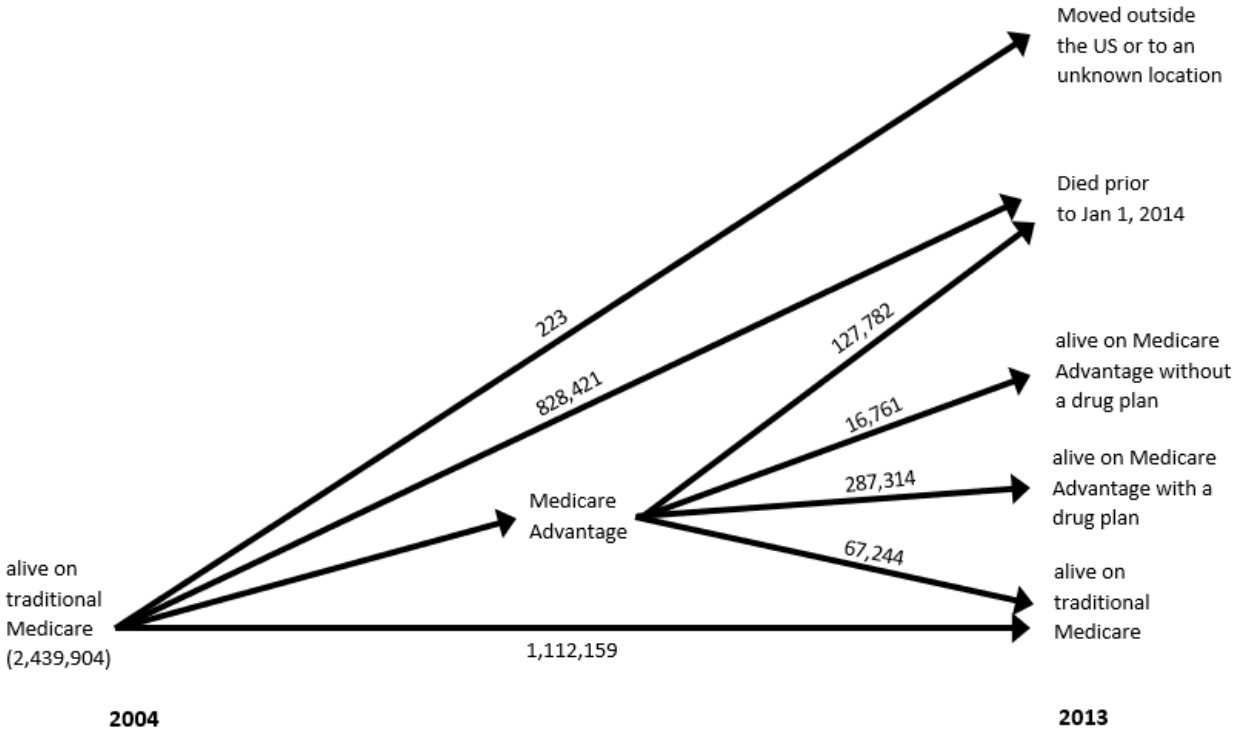


TABLE A1: SUMMARY STATISTICS FOR MEDICARE BENEFICIARY SAMPLES

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

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

B. Locations of Air Pollution Monitors and Nonattainment counties

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

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

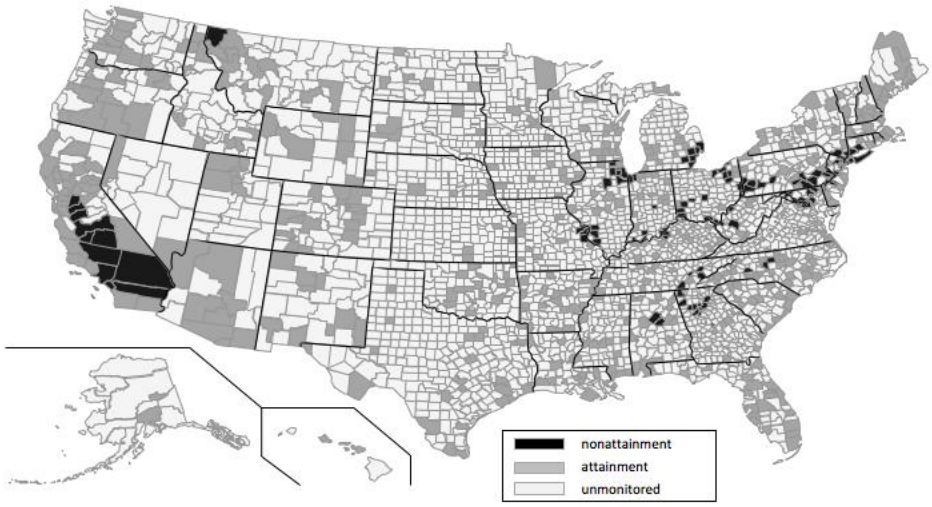


Figure B2 shows the locations of $PM_{2.5}$ monitors. This figure was generated using the Environmental Protection Agency's AirData Air Quality Monitor app: https://www.epa.gov/outdoor-air-quality-data/interactive-map-air-quality-monitors.

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

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

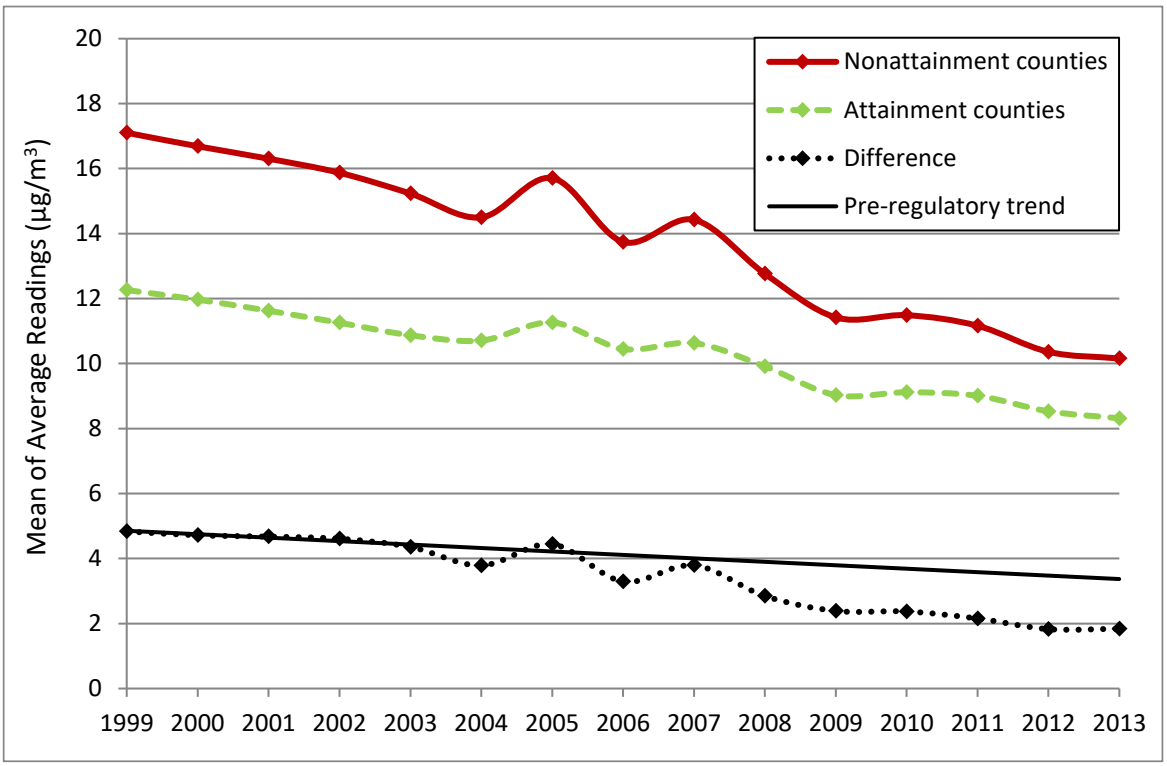
C. Annual $PM_{2.5}$ Concentrations by County Attainment Status

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

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

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

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

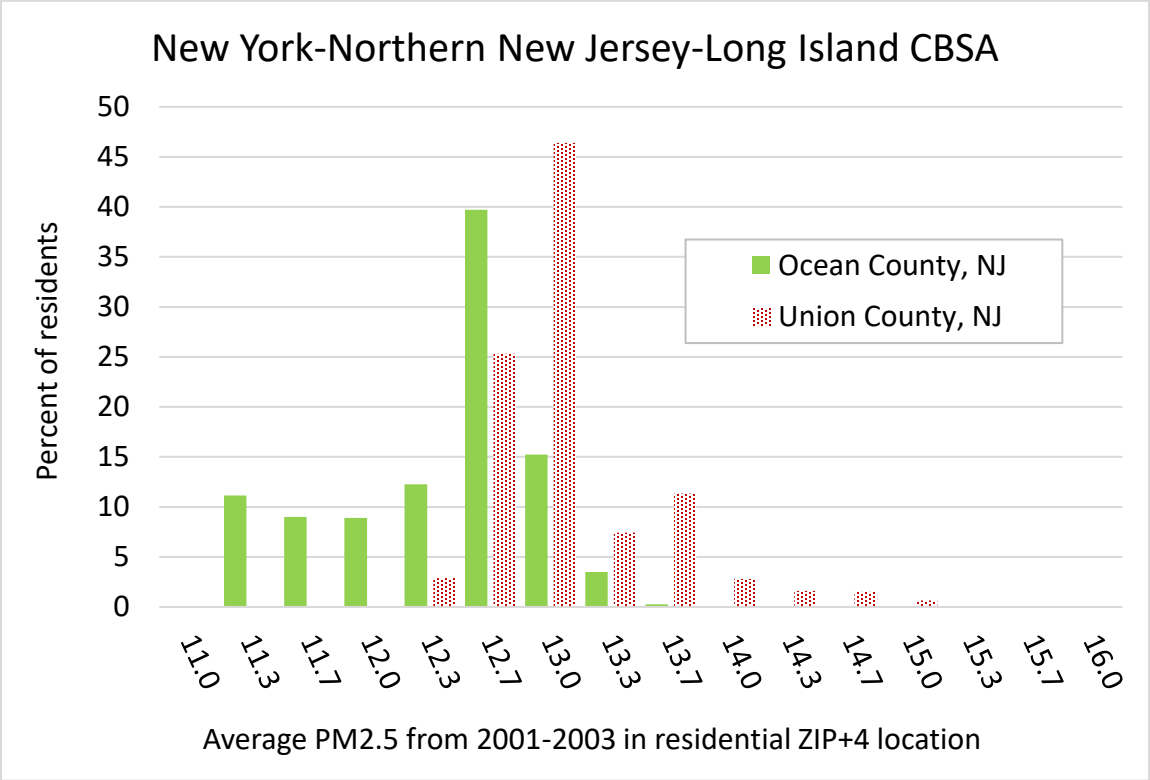


Note: The figure reports annual average concentrations of $PM_{2.5}$. Measurements are taken from air quality monitors in counties designated in 2005 as attainment or nonattainment with the federal standard based on monitor readings from 2001-2003. The nonattainment line is a simple average over monitors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

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

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

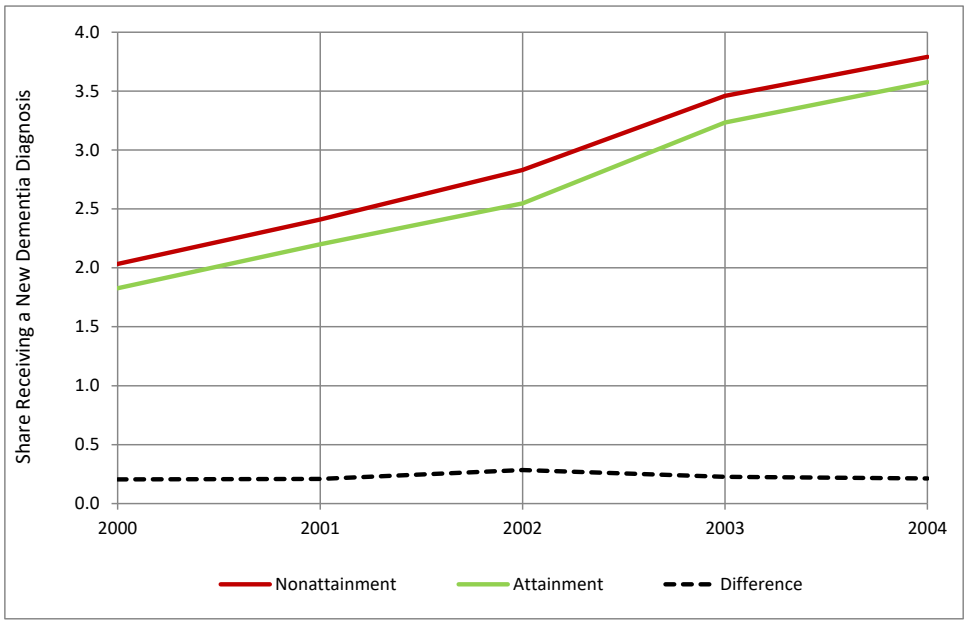
FIGURE D1: WITHIN-CBSA VARIATION IN NONATTAINMENT STATUS BY BASELINE $PM_{2.5}$ LEVELS



E. Trends in New Dementia Diagnoses Prior to 2005

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

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



F. $PM_{2.5}$ Exposure and Migration

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

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

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

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

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

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

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

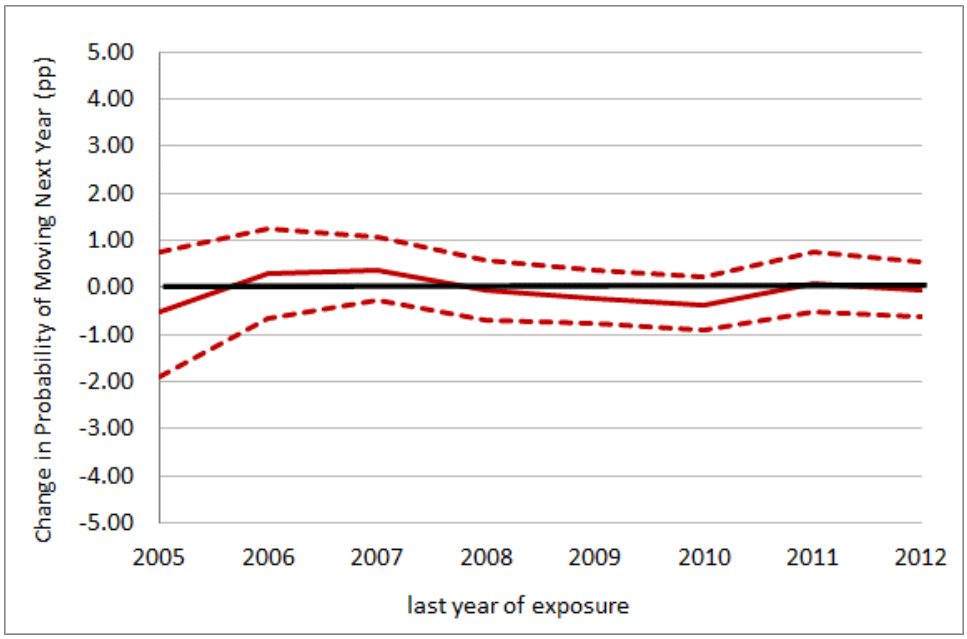


Figure F1 shows our 2SCML estimates for π and 95% confidence bands. The point estimates range from -0.52 pp to 0.38 pp and the confidence bands include zero in all years. For reference, the share of people moving each year ranges from a low of 3.97 pp in 2007 to a high of 5.23 pp in 2005.

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

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

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

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

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

G. A Model of a Dementia-Diagnosis Production Function

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

H. Using Partial Identification to Address Selection on Mortality

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

This allows us to write:

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Results

TABLE H1—AVERAGE MARGINAL EFFECTS ALLOWING FOR SELECTION ON SURVIVAL

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

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

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

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

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

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

I. Additional results referenced in Sections III and IV

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

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

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

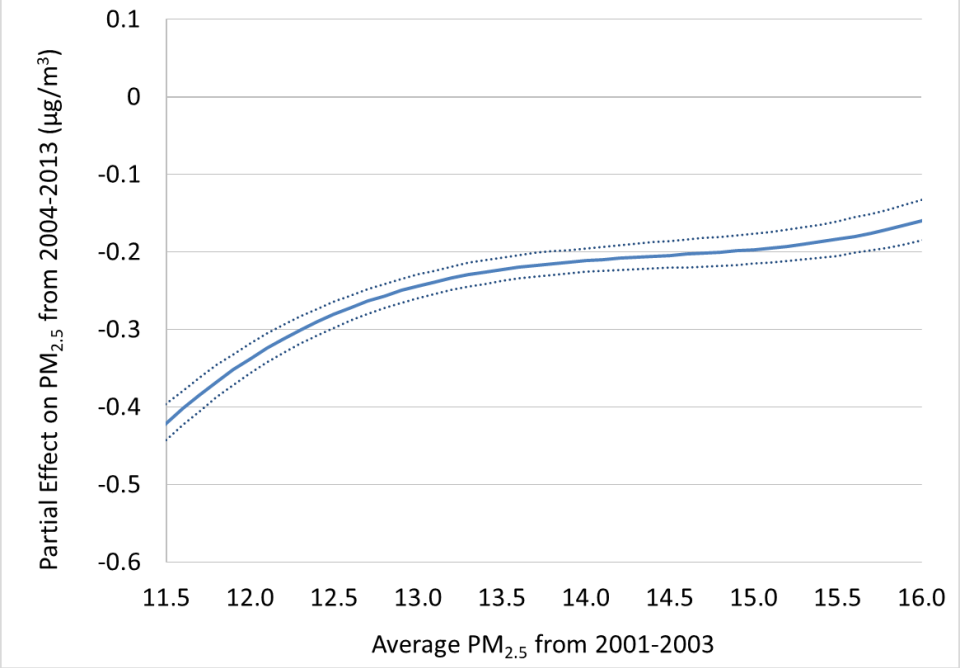
Note: The dependent variable equals 100 if an individual died on or before December 31, 2013 and 0 otherwise. The columns are numbered to correspond to the specifications in the corresponding columns of Table I in the main text. Col (1) is a probit model with CBSA-specific intercepts. Col (2) adds all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

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

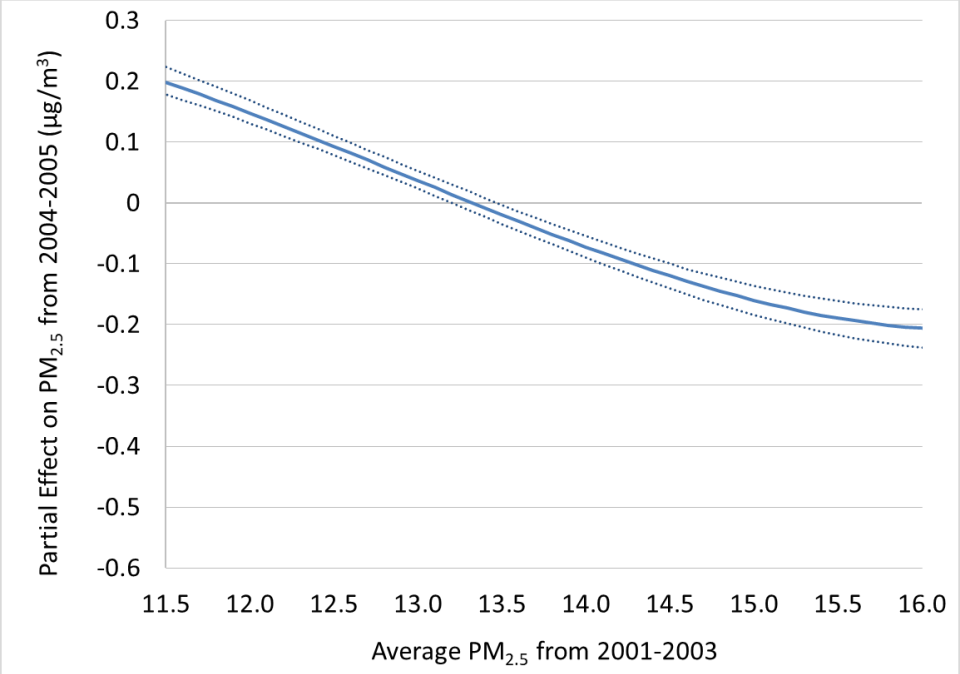
2. *Duration-Specific Partial Effects of Nonattainment on $PM_{2.5}$ Exposure*

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

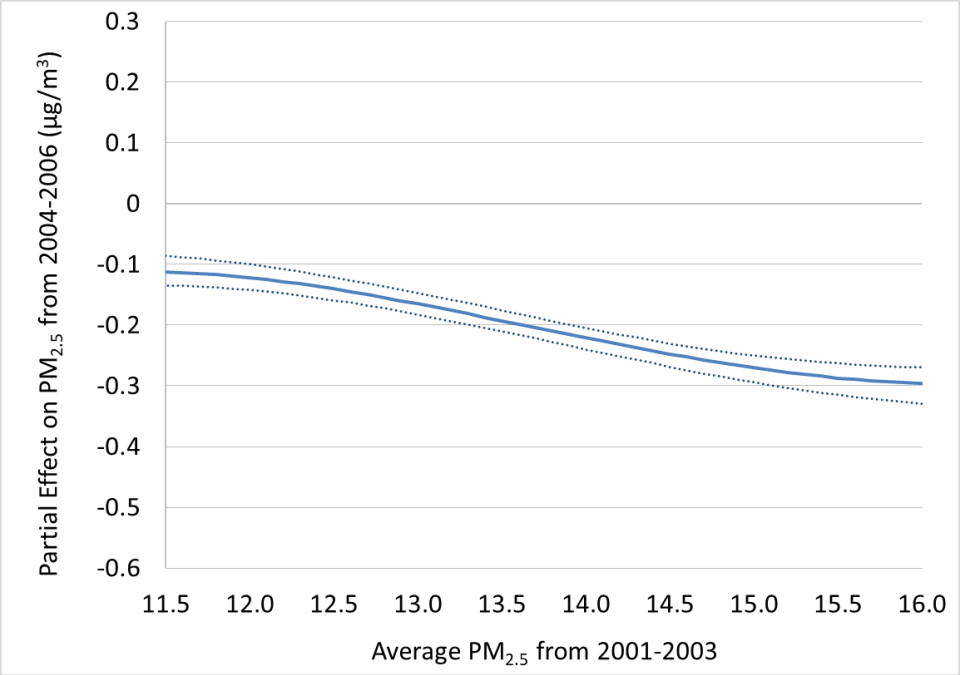
FIGURE I1— ESTIMATED PARTIAL EFFECTS OF NONATTAINMENT (NA) ON $PM_{2.5}$ EXPOSURE



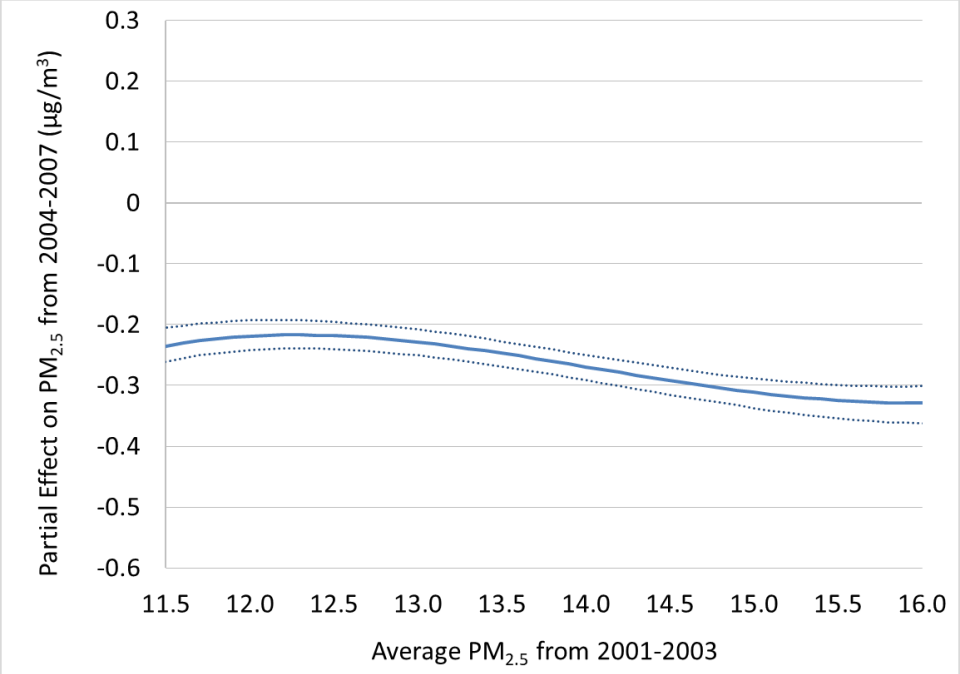
Panel i: Estimated Partial Effect of NA on Decadal $PM_{2.5}$ Exposure, 2004-2013



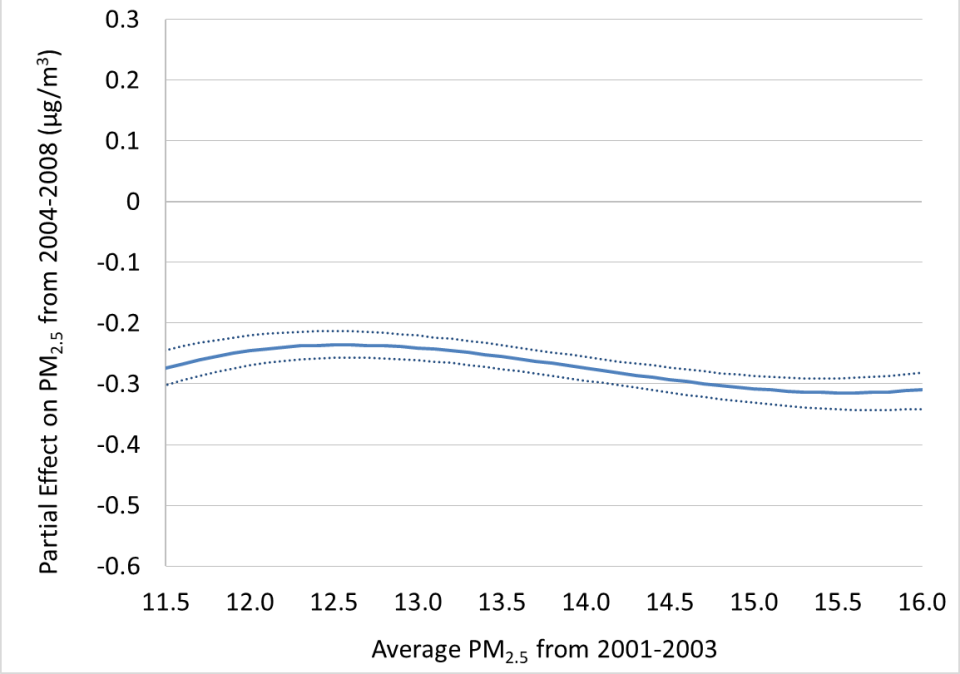
Panel ii: Estimated Partial Effect of NA on $PM_{2.5}$ Exposure, 2004-2005



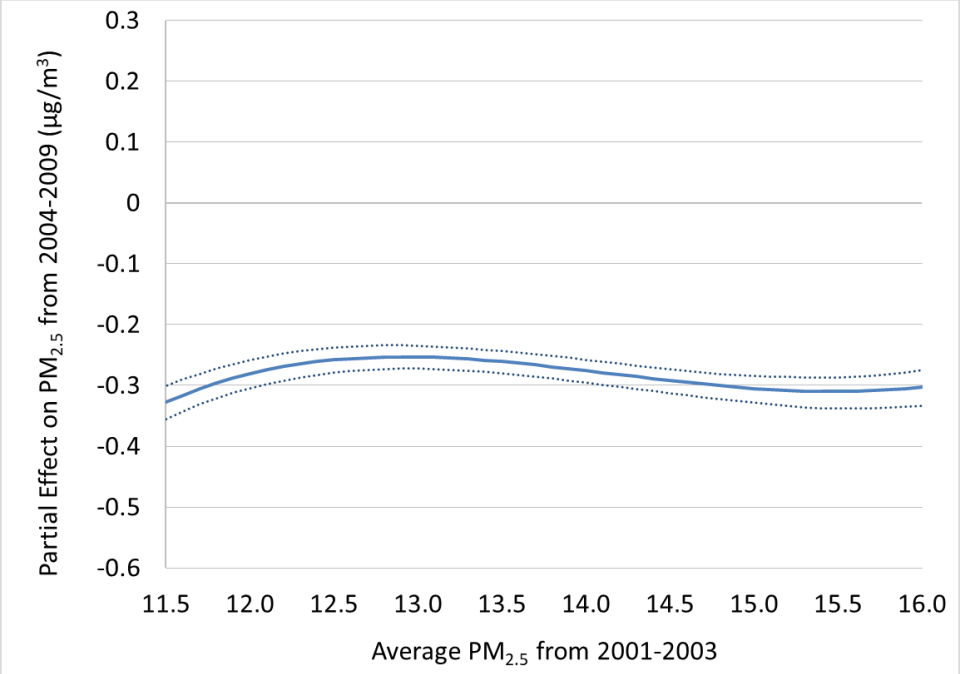
Panel iii: Estimated Partial Effect of NA on $PM_{2.5}$ Exposure, 2004-2006



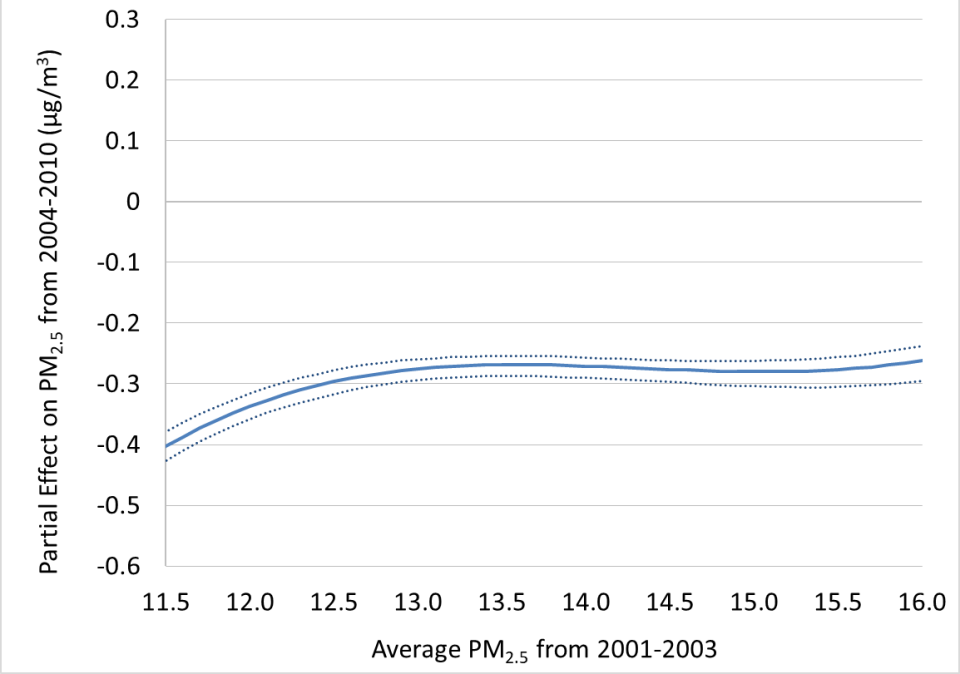
Panel iv: Estimated Partial Effect of NA on $PM_{2.5}$ Exposure, 2004-2007



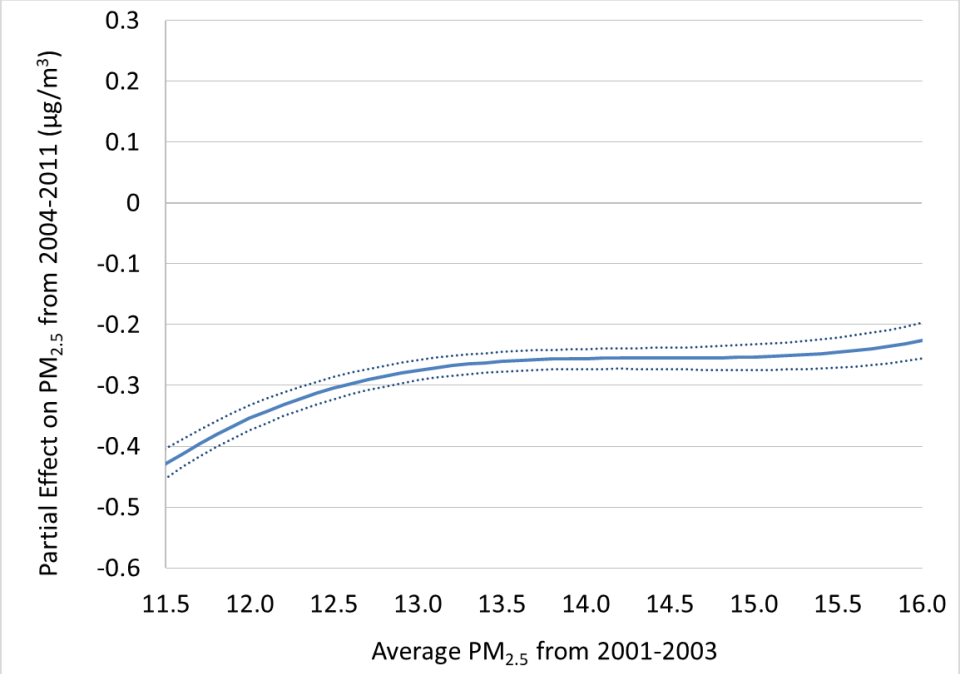
Panel v: Estimated Partial Effect of NA on $PM_{2.5}$ Exposure, 2004-2008



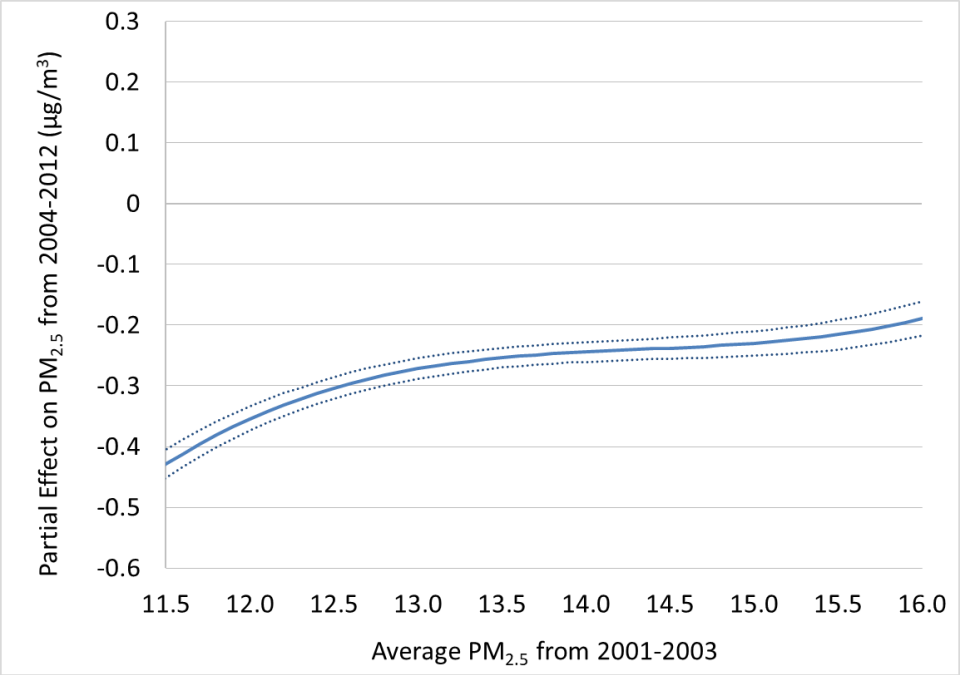
Panel vi: Estimated Partial Effect of NA on $PM_{2.5}$ Exposure, 2004-2009



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



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



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

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

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

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

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

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

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

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

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

TABLE I3: RESULTS FROM THE DECADAL 2SCML SPECIFICATION

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
$PM_{2.5}$ (1 µg/m^3) (Decadal, 2004-2013)	39.484	25.849	-12.319	90.670
$(PM_{2.5})^2$	4.680	2.932	-0.485	10.526
$(PM_{2.5})^3$	-0.443	0.169	-0.785	-0.149
$(PM_{2.5})^4$	0.012	0.004	0.005	0.019
$PM_{2.5}$ (1 µg/m^3) interacted with:				
2004 Gross Medicare Expenditures ($10,000)				
expenditures	1.422	0.968	-0.460	3.307
expenditures^2	-0.825	0.375	-1.569	-0.116
expenditures^3	0.110	0.048	0.018	0.204
expenditures^4	-0.004	0.002	-0.008	0.000
2004 Census Block Group Demographics				
median household income / 1000	-0.003	0.007	-0.015	0.011
per capita income / 1000	0.005	0.011	-0.016	0.026
median year built	-0.010	0.006	-0.023	0.002
median house value / 1000	-0.001	0.001	-0.003	0.000
average house value / 1000	0.000	0.000	-0.001	0.001
median gross rent / 1000	-0.005	0.019	-0.044	0.031
% over 65	-2.129	0.932	-3.948	-0.289
% white	1.009	0.933	-0.715	2.931
% black	-0.255	1.146	-2.374	2.016
% hispanic	-4.659	1.030	-6.661	-2.544
% 9th through 12th	-2.922	2.514	-7.542	1.940
% high school graduate	-4.996	1.863	-8.708	-1.250
% some college	-4.735	1.841	-8.414	-1.409
% associate degree	-2.661	2.374	-7.167	2.445
% bachelor's degree	-7.192	1.907	-11.229	-3.296
% graduate degree	-7.006	2.114	-11.090	-2.992
% owner occupied	-2.534	1.011	-4.495	-0.543
% renter occupied	-1.245	1.083	-3.457	0.763

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

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
$PM_{2.5}$ (1 μg/m^3) interacted with:				
Chronic conditions in 2004				
H	0.243	0.261	-0.279	0.745
S	0.659	1.201	-1.591	3.203
S, H	0.561	0.734	-0.853	2.064
D	1.449	0.691	0.156	2.837
D, H	1.308	0.481	0.336	2.231
D, S	1.222	3.301	-5.412	7.599
D, S, H	4.250	1.318	1.788	6.811
I	0.555	0.459	-0.365	1.411
I, H	1.181	0.338	0.543	1.785
I, S	1.950	1.540	-0.894	5.132
I, S, H	1.643	0.786	0.155	3.128
I, D	2.729	1.086	0.673	4.798
I, D, H	1.949	0.544	0.840	3.033
I, D, S	7.245	3.878	-0.828	15.051
I, D, S, H	2.749	1.137	0.495	5.005
C	4.532	1.520	1.770	7.546
C, H	2.438	0.923	0.619	4.157
C, S	0.334	5.631	-10.370	11.557
C, S, H	1.522	1.959	-2.669	5.234
C, D	5.325	3.179	-0.322	11.770
C, D, H	3.731	1.290	1.139	6.253
C, D, S	-14.273	14.365	-39.912	17.850
C, D, S, H	4.367	2.823	-1.022	10.229
C, I	3.374	1.333	0.687	5.861
C, I, H	3.310	0.858	1.617	5.004
C, I, S	2.938	3.168	-3.593	9.318
C, I, S, H	3.442	1.266	0.937	5.960
C, I, D	4.838	2.609	-0.188	9.968
C, I, D, H	4.815	1.161	2.355	7.138
C, I, D, S	2.502	6.684	-10.026	15.451
C, I, D, S, H	4.525	1.584	1.203	7.558

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

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
$PM_{2.5}$ (1 μg/m^3) interacted with:				
Age				
76	-0.228	0.700	-1.641	1.182
77	0.488	0.663	-0.844	1.803
78	0.347	0.672	-1.008	1.645
79	0.967	0.685	-0.455	2.197
80	1.109	0.688	-0.236	2.382
81	0.620	0.707	-0.898	1.949
82	0.629	0.753	-0.890	2.106
83	0.346	0.770	-1.333	1.779
84	0.650	0.828	-1.070	2.173
85	1.698	0.875	-0.144	3.389
86	1.828	0.925	-0.157	3.613
87	2.424	0.998	0.348	4.318
88	2.826	1.115	0.569	4.847
89	1.798	1.224	-0.755	4.044
90	3.597	1.361	0.828	6.265
91	4.834	1.500	1.740	7.719
92	3.909	1.626	0.391	6.940
93	5.231	1.808	1.449	8.681
94	6.661	1.982	2.513	10.393
95	4.594	2.132	0.227	8.305
96	5.789	2.350	0.650	10.188
97	7.989	2.579	2.766	12.553
98	4.890	2.831	-1.017	10.233
99	7.660	3.038	1.315	13.517
100 and over	8.984	3.274	1.805	15.021
control functions				
cf_pm2.5	1.267	0.367	0.534	1.962
$(cf\_pm2.5)^2$	-0.168	0.093	-0.334	0.030
cf_survival	-11.117	3.412	-17.540	-3.984
$(cf\_survival)^2$	2.403	0.878	0.540	4.042

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

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
$PM_{2.5}$ (1 μg/m^3) interacted with:				
Age x male				
76	-0.109	1.110	-2.218	2.034
77	-0.918	1.033	-2.901	1.112
78	-0.479	1.039	-2.510	1.492
79	-1.991	1.049	-3.995	0.207
80	-2.531	1.044	-4.611	-0.440
81	-0.416	1.028	-2.385	1.768
82	-1.294	1.065	-3.293	0.757
83	0.312	1.043	-1.700	2.494
84	-0.764	1.045	-2.848	1.282
85	-1.030	1.041	-2.975	1.019
86	0.327	1.073	-1.688	2.435
87	-0.084	1.080	-2.239	2.177
88	-0.720	1.100	-2.972	1.499
89	0.408	1.168	-2.013	2.639
90	-0.227	1.198	-2.585	2.051
91	-0.515	1.279	-2.963	2.065
92	-1.124	1.325	-3.798	1.404
93	-0.657	1.465	-3.533	2.147
94	-0.705	1.678	-3.937	2.478
95	2.402	1.822	-1.191	5.974
96	-0.632	2.200	-4.909	3.764
97	-1.789	2.615	-6.992	3.343
98	2.141	3.329	-4.198	8.830
99	2.470	4.055	-5.530	10.321
100 and over	-2.415	3.005	-8.430	3.353
individual demographics				
male	1.645	0.808	-0.058	3.128
White	-1.077	0.737	-2.510	0.338
Black or African American	-0.975	0.866	-2.697	0.677
Asian	-1.023	0.787	-2.563	0.513
Hispanic	-2.852	0.782	-4.502	-1.330

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

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
cf_survival	74.934	38.070	-6.040	146.613
$(cf\_survival)^2$	-32.118	9.822	-50.539	-11.592
cf_pm2.5	-22.404	4.816	-32.406	-13.454
$(cf\_pm2.5)^2$	3.888	1.129	1.623	6.061
Chronic conditions in 2004				
H	1.006	2.872	-4.556	6.641
S	25.583	13.155	-2.052	49.909
S, H	30.668	8.086	14.282	46.116
D	0.793	7.647	-14.363	15.759
D, H	3.220	5.291	-7.071	14.309
D, S	46.609	36.678	-24.743	119.243
D, S, H	6.776	14.671	-21.305	34.500
I	2.247	5.075	-7.195	12.233
I, H	-1.926	3.761	-8.770	5.195
I, S	18.031	17.179	-16.960	49.854
I, S, H	23.884	8.773	6.994	41.027
I, D	-8.003	12.063	-31.620	14.825
I, D, H	3.651	6.048	-8.165	16.141
I, D, S	-41.323	43.638	-132.063	46.768
I, D, S, H	27.623	12.761	2.537	52.483
C	-20.615	16.852	-54.720	11.104
C, H	0.132	10.286	-19.045	20.810
C, S	44.545	63.352	-84.456	163.363
C, S, H	40.346	21.784	-1.952	86.917
C, D	-9.535	35.429	-81.698	52.420
C, D, H	4.764	14.389	-23.253	33.639
C, D, S	235.527	162.408	-124.224	527.102
C, D, S, H	31.650	32.111	-36.656	93.936
C, I	-6.339	14.881	-34.375	23.194
C, I, H	-4.033	9.580	-22.052	14.570
C, I, S	19.125	34.838	-47.472	89.692
C, I, S, H	24.788	14.238	-2.820	52.601
C, I, D	-4.186	28.891	-59.831	52.782
C, I, D, H	-1.275	12.918	-26.061	25.325
C, I, D, S	64.062	73.935	-79.146	202.917
C, I, D, S, H	39.926	17.723	5.197	76.292

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

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
2004 Gross Medicare Expenditures ($10,000)				
expenditures	-8.341	10.659	-29.167	12.259
expenditures^2	5.482	4.131	-2.187	13.784
expenditures^3	-0.748	0.523	-1.803	0.232
expenditures^4	0.033	0.022	-0.009	0.075
Age				
76	8.930	7.770	-6.168	24.592
77	7.297	7.326	-7.101	22.315
78	15.123	7.483	0.466	29.949
79	15.648	7.597	1.726	30.990
80	22.663	7.598	9.035	37.284
81	33.472	7.874	18.559	50.206
82	41.253	8.319	24.868	57.412
83	53.219	8.542	37.905	71.564
84	55.971	9.212	38.384	75.077
85	54.606	9.699	36.112	75.018
86	62.368	10.215	43.225	83.703
87	63.677	11.032	42.569	86.685
88	68.490	12.357	45.467	92.894
89	89.353	13.592	64.143	117.638
90	77.938	15.127	47.727	108.520
91	72.873	16.613	42.018	108.464
92	90.433	18.024	56.684	128.590
93	84.583	20.067	47.948	126.895
94	80.763	21.952	39.800	127.324
95	112.809	23.698	70.727	161.665
96	111.373	26.117	62.279	167.530
97	95.170	28.771	42.532	155.848
98	139.612	31.415	82.014	203.274
99	119.898	34.031	55.085	187.834
100 and over	118.523	36.441	50.700	194.638

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

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
Age x male				
76	0.030	12.290	-24.162	22.733
77	9.217	11.410	-13.508	31.248
78	4.558	11.630	-18.078	27.070
79	19.901	11.707	-3.474	42.810
80	24.186	11.628	1.334	47.623
81	-0.170	11.492	-24.485	21.816
82	11.756	11.755	-10.489	34.176
83	-7.170	11.617	-31.609	14.877
84	5.771	11.691	-16.046	29.412
85	7.740	11.560	-14.559	30.072
86	-7.441	11.886	-31.957	14.554
87	-4.438	12.058	-29.330	19.385
88	2.303	12.239	-21.791	26.867
89	-9.780	13.018	-34.658	17.078
90	-3.227	13.295	-29.142	23.396
91	-3.664	14.169	-32.538	23.925
92	7.056	14.648	-22.047	36.095
93	0.372	16.258	-32.113	32.857
94	-1.296	18.393	-35.859	33.604
95	-36.027	20.098	-75.501	2.717
96	-2.124	24.304	-49.600	46.066
97	16.116	29.169	-41.674	72.660
98	-36.966	36.434	-110.867	32.419
99	-40.442	44.344	-126.756	46.923
100 and over	9.610	33.451	-53.474	75.826

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

OUTCOME: NEW DEMENTIA DIAGNOSIS

	probit coefficient (x 100)	Robust bootstrap standard error	95% Confidence Interval	
individual demographics				
male	-19.128	8.986	-35.928	-0.288
White	15.615	7.915	0.607	31.268
Black or African American	23.995	9.475	5.794	43.434
Asian	9.355	8.418	-7.172	26.376
Hispanic	40.919	8.409	25.348	58.641
2004 Census Block Group Demographics				
median household income / 1000	0.003	0.074	-0.148	0.148
per capita income / 1000	-0.069	0.120	-0.297	0.164
median year built	0.089	0.070	-0.050	0.222
median house value / 1000	0.001	0.010	-0.018	0.022
average house value / 1000	0.001	0.004	-0.008	0.010
median gross rent / 1000	0.121	0.213	-0.260	0.555
% over 65	15.774	10.136	-3.654	36.214
% white	-4.145	10.315	-24.675	15.135
% black	13.415	12.939	-13.480	37.991
% hispanic	54.232	11.384	29.783	76.231
% 9th through 12th	36.212	28.489	-20.266	89.031
% high school graduate	39.746	20.932	-2.056	80.630
% some college	28.153	20.694	-9.611	68.211
% associate degree	-3.357	26.527	-61.196	47.685
% bachelor's degree	50.085	21.488	5.327	93.467
% graduate degree	49.560	23.686	2.762	94.380
% owner occupied	15.949	11.045	-4.658	37.178
% renter occupied	20.582	11.842	-1.127	45.274
$PM_{2.5}$ (1 µg/m3) baseline (2001-2003)	-36.492	8.987	-54.736	-18.638
$(PM_{2.5})^2$	2.556	0.912	0.681	4.275
$(PM_{2.5})^3$	-0.094	0.040	-0.168	-0.012
$(PM_{2.5})^4$	0.001	0.001	0.000	0.003

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

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

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

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

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

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

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

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

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

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

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

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

$R^2 = 0.961$

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

OUTCOME: SURVIVAL TO 2013

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

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

OUTCOME: SURVIVAL TO 2013

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

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

OUTCOME: SURVIVAL TO 2013

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

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

OUTCOME: SURVIVAL TO 2013

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

5. Association between Dementia and its Clinical Risk Factors

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

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

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

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

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

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

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

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

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

J. Sensitivity analyses referenced in Section VI

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

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

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

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

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

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

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

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

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

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

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

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

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

2. Sensitivity Analysis: Medicare Advantage

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

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

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

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

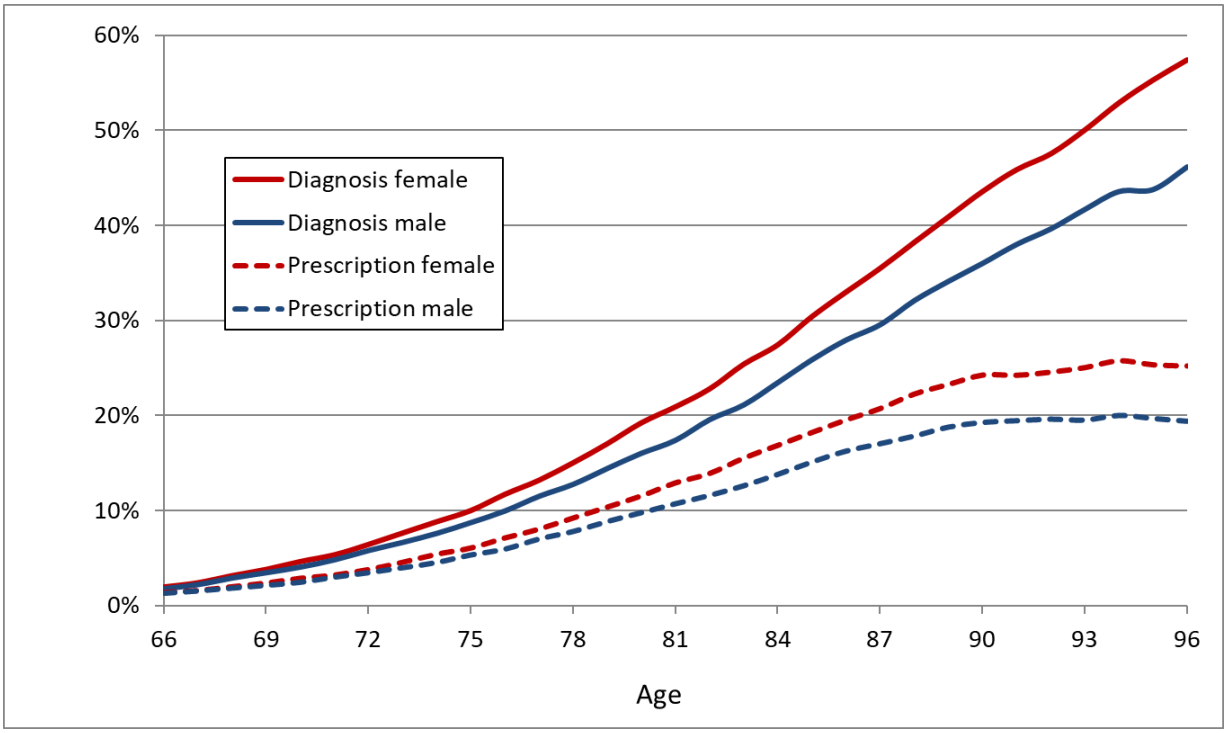


Table J2 contrasts the results shown in Column (5) of Table I that we reported for the TM population with ancillary estimates that include people from the MA population on drug plans. As

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

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

TABLE J2—AVERAGE MARGINAL EFFECTS INCLUDING MEDICARE ADVANTAGE ENROLLEES

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

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

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

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

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

3. Sensitivity Analysis: Alzheimer's and Other Dementias

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

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

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

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

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

4. Sensitivity Analysis: Distance to Nearest Monitoring Station

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

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

TABLE J4—AVERAGE MARGINAL EFFECTS CONDITIONAL ON DISTANCE TO MONITOR

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

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

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

5. *Placebo Outcomes*

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

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

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

TABLE J5—AVERAGE MARGINAL EFFECTS FOR PLACEBO SPECIFICATIONS

	Dementia	Glaucoma	Fibro-myalgia	Breast cancer	Prostate cancer	Peripheral vascular disease
1 μg/m^3 increase in decadal $PM_{2.5}$	1.707***	-0.424	-0.603	-0.008	-0.121	0.788
	(0.559)	(0.590)	(0.540)	(0.237)	(0.255)	(0.645)
F-statistic on instruments for $PM_{2.5}$	496	448	476	474	472	483
number of individuals: outcome equation	1,179,094	997,106	1,104,693	1,162,565	1,164,689	1,115,560
dependent variable mean	20	18	18	3	4	25

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

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