Pollution, Education, and Their Genetic Interactions: Sources of Cognitive Improvements in the US Elderly Population

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Abstract

The age-sex adjusted prevalence of dementia in the United States has declined by more than 30% since 2000. However, the underlying sources of this cognitive improvement remain unclear. We examine this decline, focusing on two commonly hypothesized contributors to cognitive function: declining air pollution and rising education. Using the Health and Retirement Study (HRS) and quasi-experimental designs, we assess the importance of each for improvements in cognitive function since 2000. To form exogenous measures of exposure to small particulate matter (PM2.5), we consider large, exogenous changes in power plant emissions. We instrument for educational attainment by digitizing historical data on the timing of university openings across states. We find that both pollution and education affect cognition. These effects grow monotonically with age but show no systematic pattern by individuals' genetic predisposition for Alzheimer's disease. Pollution reductions and increased educational attainment together can more than fully explain the reduction in dementia prevalence over time.

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1 Introduction

Rising life expectancy and the aging of the United States population have focused considerable attention on Alzheimer's disease and related dementias (ADRD). ADRD is enormously costly, exceeding $350 billion in direct medical care costs in 2023 and roughly the same amount in informal care costs ("2024 Alzheimer's Disease Facts and Figures," 2024). Yet, while the number of people with ADRD has grown, the age-sex adjusted prevalence of dementia among elderly adults in the United States has fallen. Using data that we discuss in more detail below, the age-sex adjusted prevalence of dementia has declined by more than 30% since 2000. Given the high costs of cognitive impairment, these patterns have important implications for the health care system, labor markets, and the government budget.

The underlying sources of this decline are poorly understood. This is due in part to the fact that the etiology of ADRD itself is not well understood. A good deal of research has focused on the physiological precursors of dementia – the build-up of amyloid-beta plaques and neurofibrillary tangles in the brain (Kametani & Hasegawa, 2018).[1] However, it is not entirely clear how essential these changes are for cognitive impairment, and what social and environmental factors might lead to these changes (Livingston et al., 2020). A number of theories have been put forward to explain both amyloid and tau buildup and residual functioning given brain pathology, including cardiovascular health (Whitmer et al., 2005), air quality (Bishop et al., 2022; La Nauze & Severnini, 2025), education (Barcellos et al., 2025; Clouston et al., 2020), job type (Choi et al., 2022), and genetic predisposition. Correlations between these social and environmental factors and cognition are not hard to come by. For example, the link between education and cognition is

[1] These are often referred to as the "amyloid hypothesis" and "tau hypothesis." Even this is not universally accepted, however (Piller, 2024, 2025).

among the best known in the field. The causal story is not as clear.[2] In this paper, we explore the role of changes in air pollution and education in explaining changes in cognitive impairment since 2000. We consider these theories because air pollution and education have changed substantially over time and therefore have considerable scope for explaining reductions in cognitive impairment. For example, population-weighted small particulate matter (PM2.5) exposure in the United States has fallen by more than 68% since 1981, while the number of years of education has increased by 3.5 years since the early 20th century. We estimate the causal effect of each of these factors on cognitive status and use our estimates to assess the extent to which changes in air pollution and education can account for declines in dementia prevalence since 2000.

Our primary data source is the Health and Retirement Study (HRS), which includes detailed measures of cognition over time alongside a rich variety of individual characteristics. Education is asked about directly. Small particulate matter exposure has been estimated at very fine levels of geography, and we link such measures to people in the HRS. The HRS also includes a subsample of individuals for whom we have polygenic risk scores, allowing us to assess whether the effects of education and PM2.5 vary with individuals' genetic predisposition to ADRD.

The central empirical challenge is that neither pollution exposure nor education are exogenous to other factors that may drive cognitive impairment. Individuals may move in and out of an area because of pollution exposure, and, despite progress in recent years, a large literature shows that pollution has long been concentrated in areas that are low-income (Jbaily et al., 2022). This raises concerns about endogenous mobility or omitted variables biasing a naïve regression of cognition on pollution exposure. Similarly, individuals with more educational attainment may

[2] These analyses are motivated by a prevailing "use it or lose it" theory of cognition, which posits that individuals who use their brain more regularly – and therefore develop a certain amount of "brain reserve" – will be less prone to cognitive decline in old age (Stern 2012).

differ in unobservable ways from those with less educational attainment, for example in their exposure to early life nutritional conditions that might also influence late life cognition.

We address these concerns by constructing instruments for PM2.5 exposure and educational attainment and then linking these measures to individuals in our data. Our PM2.5 exposure instrument is calculated from changes in power plant emissions that arise from largely exogenous sources – e.g., EPA mandates on scrubber installation or changes in the type of fuel burned. Power plants are a particularly important source of PM2.5, accounting for 18% of the estimated PM2.5 exposure in the United States at the start of our sample period (Hernandez-Cortes et al., 2023).[3] We start by forming emissions output and total electricity generation at each plant in each year. Emissions will vary both because of technology and because of greater and lesser need for electricity. The latter is problematic for our estimates, as energy demand may be correlated with other factors that influence cognition. To purge energy demand from the estimates, we form a time series of emissions that fixes the megawatt (MW) output of each plant but allows the emissions rate per MW to vary over time. Using these constant-output emissions as inputs to a pollution transport model, we generate estimates of PM2.5 exposure from power plant changes for every Census block in the United States from 2000-2016 (Hernandez-Cortes et al., 2022; Tessum et al., 2017), which we link to individuals in the HRS.

Our education instruments combine the rich historical data in the HRS with digitized state-by-year data on the availability of higher education throughout the 20th century. Specifically, we use historical residence data in the restricted HRS to identify the state where individuals lived at age 10, and we link them to hand-collected measures of higher education availability in that state in the year they were 17, similar to the approach taken in other work (Currie & Moretti, 2003;

[3] The energy industry as a whole is a substantial contributor to overall PM2.5 exposure (McDuffie et al., 2021).

David Card, 1995; Doyle & Skinner, 2016; Fletcher & Noghanibehambari, 2024). Both of our instruments have strong first stages.

Our results demonstrate that both air pollution and education have significant effects on cognition. PM2.5 in the year of the survey has significant, negative effects on elderly adults' cognitive function, and these effects increase monotonically in age. A 1 standard deviation increase in PM2.5 exposure increases the probability that an individual has dementia by 1.5 percentage points, 17% of the sample mean. Perhaps surprisingly, past pollution exposure has little additional impact when controlling for current exposure. Using our genetics subsample, we show that these effects do not differ by individuals' genetic predisposition for ADRD.

Education also has significant effects throughout the distribution of cognitive ability. An additional year of schooling reduces the probability of dementia by 2.3 percentage points (26% of the sample mean) and mild cognitive impairment by 4.8 percentage points (26% of the mean). This effect also increases monotonically in age. Consistent with our pollution estimates, we find little heterogeneity in these effects by genetic predisposition for ADRD.

Evaluating the combined impact of recent improvements in air pollution and education, we find that changes in the two together are more than sufficient to explain all of the decline in dementia prevalence since 2000. In fact, all else equal, our point estimates imply a lower dementia prevalence in 2016 than we observe in the data. This suggests that other social phenomena – perhaps rising rates of obesity and associated cardiovascular risk factors – may push dementia rates upwards, against the trends of cleaner air and a better educated population. Studying these sources of cognitive decline is an important area for future work.

Our findings contribute to a few important strands of existing literature. First, we build on a robust literature on the effects of air pollution on human health. Much of the published literature

estimating the causal effects of pollution has focused on measures of physical health, such as mortality (Anderson, 2020; Currie & Neidell, 2005) and medical care utilization (Deryugina et al., 2019; Dominici et al., 2006; Komisarow & Pakhtigian, 2022; Sacks et al., 2022). By contrast, we focus on cognitive health, where, with a few recent exceptions (Bishop et al., 2022; Fukushima et al., 2024; La Nauze & Severnini, 2025; Von Hinke & Sørensen, 2023), causal links between air pollution and cognition are more scarce.

Our work also contributes to a broader literature regarding the effects of education on health. As with pollution, an extensive literature has explored the importance of education for individual health outcomes such as mortality, with mixed results (Cutler & Lleras-Muney, 2006; Galama et al., 2018). This work has focused largely on easily measured health outcomes (e.g., mortality) or health behaviors (e.g., smoking, lack of exercise, etc.). Less causal work explores a causal relationship between education and cognitive health, with one recent exception (Barcellos et al., 2025).

The rest of the paper proceeds as follows. Section 2 provides background on the hypothesized sources of cognitive decline and the role of social factors in cognitive impairment. Section 3 describes our data and estimation strategy. Section 4 presents information on our instruments. Section 5 details our results, while Section 6 outlines our estimates of the role of education and pollution in declining dementia prevalence since 2000. Section 7 concludes.

2 Cognition and Sources of Cognitive Decline

Cognition is a continuous metric, from excellent to poor. Generally, researchers group continuous measures of cognition into three categories: normal cognition, mild cognitive impairment, and dementia (Langa et al., 2020). The latter category is commonly termed Alzheimer's Disease and Related Dementias, or ADRD. We work with this discrete grouping, for

two reasons. First, the categories have been designed to match in-depth neuropsychological assessments conducted on a subsample of HRS respondents in the Aging, Demographics, and Memory Study (ADAMS) (Crimmins et al., 2011). Second, not everyone in the survey has a detailed cognitive score, but they do have category evaluations. For example, a person with severe dementia may not be able to complete a cognitive assessment. For this individual, all that is known is that the person has dementia, not the severity of the dementia.

Cognitive impairment is common in the elderly population. Of the population aged 65 and older, 72.4% have normal cognition, 18.6% have mild cognitive impairment, and 9% have dementia. The share with dementia rises from 3% for those aged 65-69 to 20% for those aged 80+.

Physiologically, there are two prevailing theories of cognitive decline. The first, known as the amyloid hypothesis, argues that cognitive decline arises because of the buildup of toxic plaques in the brain that are comprised of amyloid beta, a peptide of amino acids (Hardy and Allsop 1991; Selkoe 1991; Selkoe and Hardy 2016). A competing explanation, known as the tau hypothesis, is that cognitive decline arises because of changes within cells. Patients with cognitive decline – and more specifically those with Alzheimer's disease (AD) – often have neurofibrillary tangles comprised of tau protein within their brain cells; these tangles ultimately impede important functions of neurons in the brain, which may contribute to cognitive decline (Goedert, 1993; Kametani & Hasegawa, 2018).[4]

Researchers have devoted considerably less attention to social and environmental sources that may influence the buildup of either amyloid or tau plaques or that might explain cognitive function conditional on the amount of each, and most such analyses present correlations more than causal links.

[4] Because drugs targeting amyloid and tau have not been shown to be associated with large improvements, some researchers believe that additional theories are needed, though we do not explore that here.

One widely noted fact is that air pollution is associated with worse cognition in older ages (Clifford et al., 2016; Zhang et al., 2023). Air pollution might affect cognition by carrying toxic metals directly to the brain or creating inflammation in the cardiovascular or digestive systems that damages the brain (Peeples, 2020). These theories are consistent with existing evidence that air pollution harms human health more broadly. A number of studies examine the effect of air pollution on mortality and show that a variety of pollutants ranging from PM2.5 to carbon monoxide increase mortality (Anderson, 2020; Beach & Hanlon, 2018; Chay & Greenstone, 2003; Currie & Neidell, 2005; Deryugina et al., 2019; Ebenstein et al., 2017) as well as morbidity (Deryugina et al., 2019; Dominici et al., 2006). Despite this robust literature linking pollution to human health, comparatively few papers focus on cognition. Those that do underscore that exposure to air pollution generally reduces individuals' cognition (Bishop et al., 2022; La Nauze & Severnini, 2025; Von Hinke & Sørensen, 2023).

However, there are limitations in these data. First, much of the recent literature relies on PM2.5 measurements from ground monitors, in some cases weighting individuals by their distance from monitors to deal with measurement error (Bishop et al., 2022; Deryugina et al., 2019). These are imperfect measures of PM2.5 because there is often a lot of space between monitors, and PM2.5 concentrations vary substantially even at a very local level (Clark et al. 2022). In contrast, we use detailed, Census-block estimates of PM2.5. Second, previous work on the effects of environmental sources of cognitive decline has relied on the use of claims-based indicators of decline, such as dementia diagnosis (Bishop et al., 2022). These measures are useful but limited, as ADRD is difficult to treat and thus often not formally diagnosed (Cutler et al., 2022).

Education is also strongly associated with cognition. Among people aged 70 and older, 4.7 percent of people with a college degree have dementia, compared to 15.7% among those with a

high school degree or less. This is often posited to be a result of "cognitive reserve": regular use of the brain throughout the life course may be protective against cognitive decline (Stern, 2012; Bonsang et al., 2012; Choi et al., 2022). While these descriptive findings are suggestive of a relationship, it is difficult to disentangle causal relationships in these settings from unobserved confounding. A notable exception is recent work from the United Kingdom showing that an additional year of education decreases the incidence of ADRD among the near-elderly (Barcellos et al., 2025). We build on this work by estimating the effect of education in an older population, where dementia prevalence is greater and the scope for education to influence cognition may be higher.

3 Data and Estimation Strategy

Our primary data source on cognition is the Health and Retirement Study (HRS). The HRS is a nationally representative, longitudinal survey of adults in the United States conducted biennially. It has been continuously administered since 1992 (Sonnega et al., 2014). We restrict our attention to individuals who are 65 and older, given low dementia prevalence prior to that age.[5]

We make use of several features of the HRS data. First, rich individual characteristics in the data allow us to observe a host of information about respondents. One of the most important individual characteristics we observe in the data is individuals' geography. We use restricted access HRS data that allow us to observe individuals' exact latitude and longitude in each year of the survey. As we describe in more detail below, this allows us to link individuals to measures of pollution exposure constructed at the Census block level. We also have information on the state the individual lived in when they were 10. We use this to match individuals to college availability

[5] Among those aged 55-64, for example, only 1.8% have dementia and only 9.5% have mild cognitive impairment.

at the time of typical college enrollment, as described below.

The HRS contains a cognition module with a battery of questions and exercises designed to measure individuals' cognition.[6] For individuals who do not have the cognitive capacity to respond to the survey, the HRS includes detailed data on functional limitations and interviewer assessments of cognition. We use a publicly available HRS data product that combines these scores, interviewer assessments, and functional limitations data into a single cognitive classification using the Langa-Weir classification algorithm (Langa et al., 2020). For each individual in each year, we note whether they fall into one of three classifications: normal cognition; cognitive impairment, no dementia (hereafter, "mild cognitive impairment"); and dementia. The set of variables used in the classification algorithm changed slightly in 2000 to include interviewer assessments of cognition. We therefore limit our analyses to years from 2000 on to ensure a consistent set of cognition measures.

Our sample size is generally large. We have nearly 83,000 observations on cognition, representing over 18,000 unique individuals. Figure 1 shows the trend in cognitive impairment in the sample. The age-sex adjusted prevalence of dementia and mild cognitive impairment have fallen steadily over time. From 2000-2020, the adjusted prevalence of dementia fell by approximately 5 percentage points, nearly a 40% reduction. Mild cognitive impairment has seen steady declines as well, falling by approximately 6.6 percentage points (approximately 31%) over the same time period.

At the same time, Figure 2b shows a marked rise in educational attainment by birth cohort, from roughly 10.5 years in 1910 to nearly 14 years in 1955. The 3.5-year increase represents an average increase of 34% in years of schooling.

[6] These include word recall, serial subtraction, and backwards counting exercises.

Genetic Data. We use genetic data collected by the HRS. The key measures for our analyses are polygenic scores (PGS), which are a summary measure of an individual's genetic predisposition for ADRD. For a given individual, the PGS is computed as a weighted sum of individual allele counts, with the weights given by the association between that allele and ADRD from a genome-wide association study (GWAS) (Ware et al., 2024). For example, an individual may have two copies of the APOE-4 gene, which would be included in their PGS weighted by the association between APOE-4 and ADRD.[7] These scores have been shown to predict cognitive decline in a variety of settings (Liu et al., 2023; Marden et al., 2016).

Genetic data are not available for everyone in the sample and have important limitations. Some people passed away before they consented to genetic information, and so their genetic information is not used.[8] Further, the construction of the polygenic scores relies on genome-wide associations studies (GWAS) conducted among those of European ancestry and may mischaracterize genetic risk for other groups (Ware et al., 2020). Our sample size for genetic information is roughly 54,000, representing almost 10,000 people – roughly half of the total sample. Owing this to this limitation, we run many of our analyses without the genetic data and present interactions with genetic predisposition to cognitive impairment as a subset of those analyses. We also dichotomize the population into those with above and below median polygenic risk for dementia.

*Pollution Data***.** We draw on public data to identify PM2.5 exposure from all sources

[7] In practice, many genes may be association with a given phenotype at varying levels of significance. We use a PGS restricted only to genotypes whose association with ADRD had a p-value of 0.01 or lower.
[8] The original cohorts in HRS were less likely to be asked to consent to genetic screens. Thus, the sample here is somewhat younger than our full sample.

(hereafter "total PM2.5"). Meng et al. (2019) estimate total PM2.5 for a gridded map of North America, with each grid cell measuring 0.01°x0.01° (roughly 1 km x 1 km). Their data are based on chemical transport modeling, satellite-based monitoring, and ground-based monitoring and are available from 1981-2016. We use the precisely identified latitude and longitude variables in the HRS to map individuals to their closest grid cell in this dataset.

Figure 2a shows the trend in population-weighted PM2.5 from the Meng et al. (2019) estimates. There is a large decline in PM2.5 over time, with a cumulative reduction of 69%. These declines have brought the national average below the Environmental Protection Agency's (EPA) health-based annual PM2.5 National Ambient Air Quality Standard (NAAQS) of 9 $\mu g/m^3$. At least part of this is from reduced pollution associated with power plants. PM2.5 from electricity generation fell 86% over this time period (Hernandez-Cortes et al., 2023).

Estimation

For the population as a whole, we wish to estimate equations of the form:

$$\text{Cognition}_{i,t} = \beta_1 \text{Education}_i + \beta_2 \text{TotalPM2.5}_{i,t} + \lambda_t + \lambda_{CZ} + X'_{i,t}\gamma + \varepsilon_{i,t} \quad (1)$$

In equation (1), β_1 is the impact of years of education on cognition, and β_2 is the impact of small particulate matter exposure on cognition. Education is generally fixed at older ages, but particulate matter exposure is not. We show equation (1) using contemporaneous pollution exposure, but in our empirical work we explore averages over some period of time.

Equation (1) controls for time fixed effects (λ_t), commuting zone fixed effects (λ_{CZ}),[9] and

[9] We follow the literature in using CZ fixed effects for our specification (Currie et al., 2023). Using CZs allows us to account for spatial correlation in pollution and the fact that changes in point sources of pollution in one area are likely to influence PM2.5 exposure in adjacent geographies. Moreover, because CZs approximate local labor markets, these fixed effects also ensure that our estimates compare individuals in the same labor market, which allows us to account for time-invariant differences across areas in economic circumstance that may influence cognition.

individual characteristics ($X'_{i,t}$), which include race, 5-year age-sex cells, marital status, and indicators for the first two waves of the survey.[10] $\varepsilon_{i,t}$ is the error term, which we cluster at the CZ level. We also use survey weights, so we depict the effect for the typical person.

As noted, we have genetic information for a subset of individuals. For this sample, we estimate model (1) including the polygenic risk score as a main effect and an interaction between below median polygenic risk score and each of education and pollution.

The central challenge in estimating equation (1) or its equivalent with genetic risk factor interactions is that pollution exposure and educational attainment are not random. For our pollution analyses, there are two primary threats to identification. The first is that richer people may move out of areas or not move into areas that are more polluted. In addition, pollution exposure is cyclical and thus may be correlated with other determinants of cognition. In the case of education, people who choose to stay in school longer may have other characteristics that lead them to have better cognition late in life. We address both of these using instrumental variables.

4 Instruments

Emissions Instrument

We develop an instrument for total PM2.5 exposure based on PM2.5 exposure resulting from exogenous changes in electricity generation at power plants. We start with data from the Environmental Protection Agency's Clean Air Markets Program Data (EPA CAMPD) on power plant emissions linked to smokestack characteristics from the US Energy Information Administration (EIA). A single power plant may contain several electricity generation units (EGUs), each with its own amount of emissions, fuel type, emissions control technologies, and

[10] Individuals who enroll in the HRS tend to be healthier than average. The dummy for the first few years in the surveys controls for this.

associated smokestacks. Our emissions data are at the EGU-fuel type-year level. For example, one observation includes the emissions output for a given EGU, the fuel type it uses, the characteristics of the smokestack to which it is linked, and other characteristics of the plant to which it belongs.

For each EGU-year, we observe emissions of various pollutants (nitrogen oxides, sulfur dioxide, carbon dioxide).[11] In addition, we observe the EGU's operating capacity (in megawatts), its heat input, and any associated smokestacks by which it emits pollutants. Finally, we observe the precise latitude and longitude of each EGU.[12] For each EGU and year, we follow previous literature and compute the EGU's emissions rate: the amount of emissions of pollutant *p* required to produce the EGU's level of output in a given year (Hernandez-Cortes et al., 2023). The emissions rate acts as a summary measure of the plant's emission control technologies. For example, if a plant installs a scrubber that allows it to emit less for a given amount of output, this will be reflected in a lower emissions rate. Over time, there were a variety of changes in emissions technology, driven by price changes for different inputs and by mandates.[13] We multiply the EGU-year emission rates by a fixed megawattage of output – taken from the second year we observe the EGU in the data – to produce the simulated total emissions with constant output in that year. This series is purged of any changes in demand for electricity over time.

Appendix Figure B.1 compares the predicted PM2.5 from electricity that results from using actual emissions versus our emissions measure stripped of cyclical variation in demand. The two series map closely to one another, suggesting that much of the decline in PM2.5 that has arisen

[11] We do not observe direct emissions of PM2.5. However, because nitrogen oxides and sulfur dioxide are two of the foremost precursory pollutants to the formation of secondary PM2.5 in the atmosphere, these data allow us to generate estimates of PM2.5 exposure using the pollution transport model described in Section 4.

[12] We thank Danae Hernandez-Cortes, Kyle Meng, and Paige Weber for sharing the linked emissions and stack characteristics data with us. For a complete description of the data and its construction, please see their paper (Hernandez-Cortes et al., 2023).

[13] For example, in 2011 the EPA finalized the Mercury and Air Toxics Standards (MATS), a set of regulatory controls requiring coal- and oil-fired power plants not using smokestack scrubbers to install them in order to reduce emissions. https://www.epa.gov/mats/cleaner-power-plants

from changes in emissions is due to falling emissions rates, not changes in aggregate electricity demand.

To translate these constant-output emissions to PM2.5 exposure, we use the Intervention Model for Air Pollution (InMAP) Source-Receptor Matrix (SRM).[14] The SRM is a set of matrices derived from the InMAP pollution transport model that characterize relationships between pollutant output at sources and changes in PM2.5 exposure at receptors. The inputs to InMAP are emissions data (noted above), location of the emissions sources, and details on the characteristics of the smokestacks from which pollutants are emitted. In our data, we observe detailed information on the smokestacks to which each EGU is linked, including height and diameter; the velocity at which pollutants are ejected; and the temperature at which they are emitted. The combined data – including emissions, EGU location, and an EGU's smokestack characteristics – give us all of the data inputs needed to estimate predicted PM2.5 using the SRM.

The SRM outputs gridded PM2.5 estimates for the lower 48 United States at a high level of spatial resolution - 1km x 1km in urban areas to 48km x 48km in rural areas (Tessum et al., 2017). We then use Census block shapefiles from the Integrated Public Use Microdata Series (IPUMS) to map the gridded estimates from the InMAP SRM to the Census blocks where HRS respondents live (Manson et al., 2023). To do so, we overlay the InMAP grid on the Census block map and compute the PM2.5 exposure in each Census block as a weighted average of the PM2.5 in the grid cells that intersect that block.

The resulting dataset contains estimated mean annual PM2.5 exposure arising from electricity generation in every Census block in the lower 48 United States from 2000-2016.

[14] A common approach in this literature is to instrument for PM2.5 exposure using changes in wind direction (Anderson, 2020; Deryugina et al., 2019). Pollution varies across space for many more reasons than just wind direction, however.

Because our HRS data are identified at the latitude and longitude level, we can identify respondents' Census blocks, allowing us to measure their pollution exposure at a fine geographic level.

There are a few key assumptions underlying our instrument. First, relevance requires that the instruments be related to total PM2.5. Panels (a) and (c) of Figure 4 plot the first-stage relationships between our instruments (x-axis) and endogenous variable (y-axis). Panel (a) is for pollution in the same year as the survey; panel (c) is for pollution in the current and four prior years. In general, our instruments are highly predictive, with an R^2 of 0.78 for both same-year and 5-year average PM2.5. In addition, we formally evaluate the strength of our instruments using the effective first-stage F-statistic of Montiel Olea & Pflueger (2013) and show for our main specifications that they exceed conventional thresholds (F = 42.6 for contemporaneous PM2.5 and F = 64.4 for 5-year PM2.5).

Further, the instruments must be as-if-randomly assigned (the independence assumption). To test this assumption, we first generate a measure of predicted dementia for each individual using a set of held-out variables that measure a variety of childhood health conditions prior to the age of 16.[15] These variables capture a host of early-life health conditions that may be predictive of dementia, and, importantly, are measured before individuals should be affected by our education instrument.

Our predicted measure of dementia is highly correlated with the true measure (Appendix Figure A.1). We then regress actual dementia and predicted dementia against the instruments. Panels (b) and (d) of Figure 4 show these relationships graphically along with their slopes. For

[15] Held-out measures include indicators for having the following conditions before age 16: measles, mumps, chicken pox, difficulty seeing, asthma, diabetes, respiratory disorders, speech impairment, allergic conditions, heart trouble, ear problems, epilepsy or seizures, headaches or migraines, stomach problems, high blood pressure, depression, drug or alcohol problems, and other psychological problems.

same-year PM2.5, we find a significant reduced-form relationship between our instrument and actual dementia, but little relationship for 5-year PM2.5. Importantly, we fail to reject a null hypothesis of zero relationship between our predicted measure and the instruments, suggesting that our instruments are well-balanced with respect to individual covariates predictive of dementia.

Third, the instruments must be monotonically related to the endogenous variable. In our first-stage estimates in Panels (a) and (c) of Figure 3, we show that this assumption holds on average. Finally, the instruments must only affect dementia through their effects on pollution. While this is untestable, we believe it a reasonable assumption in this setting.

Higher Education Instrument

To construct an instrument for educational attainment, we use data on the availability of higher education throughout the 20th century. We hand collect data from the Census Bureau's Statistical Abstract of the United States on the number of institutions of higher education in each state from 1916-1976 (U.S. Census Bureau, 2023).[16] The statistical abstracts include a wide variety of post-secondary institutions, including universities, liberal arts colleges, normal schools, and other professional schools. Because for many years we have biennial data, we interpolate the number of universities for the intervening years, taking the average of the two adjacent years.[17]

Using HRS data on where individuals lived at age 10, we match individuals to the availability of higher education based on that state and the year that they turned 17. This assumes that people did not move between those ages.[18] For each individual, we use the count of institutions

[16] The data will be posted online after the refereeing process is complete.
[17] From 1916-1962, the data are collected biennially. We also have data for 1963, 1965, 1967, 1968, 1970-1972, and 1974-1976. For any year where data is not collected, we compute the number of institutions as the average of the years immediately before and after.
[18] Interstate moving among children was low during this period, so we believe this to be a reasonable assumption (Card & Krueger, 1992).

of higher education in their state at age 17 as our instrument for educational attainment. The use of this spatial and temporal variation in the availability of post-secondary education is similar to other work in the literature (Currie & Moretti, 2003; David Card, 1995; Doyle & Skinner, 2016; Fletcher & Noghanibehambari, 2024). We experimented with using enrollment at age 17 as the instrument: for example, the share of people aged 15 to 24 in the state and year that the person turned 17 who were enrolled in higher education. However, data on enrollment by age are sparser, providing us with less year-over-year variation.

Panels (e) and (f) of Figure 3 shows tests of the validity of the education instrument. Education availability is strongly associated with educational attainment. Dementia prevalence falls with the number of higher education institutions, but predicted dementia prevalence based on health impairments at young ages does not.

5 Results

Table 1 shows summary statistics for the main estimation sample (column 1) and the genetics subsample (column 2). Average same-year PM2.5 exposure in our sample is about 9.76 $\mu g/m^3$, and the average individual in the sample has about 12.5 years of education. Approximately 72.4% of the person-years in the sample are classified as normal cognition, 18.6% are classified as having mild cognitive impairments, and 9% are classified as having dementia. Broadly speaking, our genetics subsample is similar to our full estimation sample. The key difference is that the genetics subsample is restricted to non-Hispanic white and non-Hispanic Black populations because of the data used to construct the polygenic scores. The genetics subsample is also slightly more educated and has slightly lower pollution exposure, likely because the genetics data were collected from 2006-2012.

Figure 3 shows the spatial distribution of changes in age-sex adjusted dementia prevalence (Panel A), PM2.5 (Panel B), and years of education (Panel C).[19] Age-sex adjusted dementia prevalence fell across all U.S. Census Divisions from 2000-2016, as did population-weighted average PM2.5 exposure. At the same time, the average number of years of education among those 65 and older increased across all divisions. Broadly speaking, these changes appear to be geographically correlated; the areas with the largest gains in education and reductions in pollution also have some of the largest reductions in dementia. Indeed, the correlation between the 2000-2016 change in dementia and the 2000-2016 change in PM2.5 at the Census division level is 0.39, and the same correlation for education is -0.77.

5.1 Effects of PM2.5 on Cognition

We first consider the effects of PM2.5 on cognition, ignoring the education information. We then do the same for education, and then combine the two. The combination is generally similar to the two independently, but the standard errors are somewhat smaller in the independent analysis.

Table 2 presents the results relating cognition and pollution. Panel A reports the effect of same-year PM2.5, and Panel B reports the effect of 5-year PM2.5 exposure. A 1 standard deviation increase in same-year PM2.5 (equal to about 3.02 $\mu g/m^3$ in our sample) increases the probability of dementia by about 1.5 percentage points. This is approximately 17% of the sample mean and is significant at the 10% level. We find corresponding reductions in normal cognitive function and mild cognitive impairment, but our estimates are noisy, and we are unable to reject no change in these measures. For all of our estimates, the first-stage F-statistic from Montiel-Olea & Pflueger (2013) is large and exceeds the conventional threshold of 10. Our estimates for five-year PM2.5

[19] For data identification purposes, HRS data cannot be shown at a level below the Census division. We report PM2.5 and education at the same area, so the figures are matching.

exposure for the full sample are similar in direction but are statistically indistinguishable from zero.

Similar to other work in this literature, we find that our OLS estimates understate the effects of PM2.5 on cognition (Deryugina et al., 2019). For dementia, the IV estimate is roughly 4 times the OLS estimate. A variety of factors may explain this result. For example, if PM2.5 is correlated with local economic activity that improves cognition (e.g., because incomes are higher), the OLS estimates would understate the effects of air pollution on cognition.

Exposure to small particulate matter may have different consequences for different groups. We therefore explore treatment effect heterogeneity in our IV estimates. We first examine different effects by age, as others have shown that life expectancy is an important predictor for the mortality effects of PM2.5 exposure (Deryugina et al., 2019). Figure 5 and Appendix Table A.1 show the estimates of equation (1) interacting pollution exposure with age bins (65-69, 70-79, and 80+). Consistent with previous work examining mortality (Deryugina et al., 2019), we find that the effects of PM2.5 on cognition rise monotonically with age. For individuals age 65-69, there is no statistically significant effect of same-year PM2.5 on the probability of dementia. However, a 1 standard deviation increase in PM2.5 increases the probability of dementia by 1.8 percentage points for 70-79 year-olds and by 2.4 percentage points for 80-89 year-olds. We find similar patterns for the effects of 5-year PM2.5, though our estimates have larger standard errors and are statistically indistinguishable from zero.

Table 5 and Panel (B) of Figure 5 show heterogeneity in the effects of PM2.5 by genetic predisposition for cognitive impairment. Our instruments remain strong when interacted with indicators for being above or below median polygenic score for ADRD. The effect of PM2.5 – both in the same year and over five years – does not vary for individuals above and below the

median polygenic risk score for ADRD. A 1 standard deviation increase in same year PM2.5 increases dementia risk by 1.5 percentage points for those with above median AD PGS, and 1.6 percentage points for those with below median AD PGS.

5.2 Effects of Education on Cognition

Table 3 shows the relationship between education and cognitive function. As shown in column (2), an additional year of schooling increases the probability of normal cognition by 7.1 percentage points, which is equivalent to about 10% of the sample mean. The improvements in cognition come from reductions in both dementia and mild cognitive impairment. An additional year of education decreases the probability of mild cognitive impairment by 4.8 percentage points, a reduction of 26%. Similarly, an additional year of education decreases the probability of dementia by 2.3 percentage points, equivalent to 26% of the sample mean. In all cases, we find that the IV estimates of the effect of education are considerably larger than the OLS estimates. For example, the effect of education on dementia is 27% greater in the IV estimate than in the OLS estimate. As others in the literature on the returns to schooling have noted, there may be several reasons for this. One possibility is that the individuals on the margin – that is, those who are induced to gain an extra year of education by the availability of more institutions of higher education – may have higher marginal returns to education than the average person.

We next examine heterogeneity in the effects of education by age. To do so, in Figure 5 we re-estimate the main specification, interacting our instruments with age bins. While these estimates are instructive, we note that the interacted instruments have F-statistics well below conventional thresholds (Appendix Table A.2). As with our PM2.5 estimates, the effect of education on the probability of dementia rises monotonically with age. For individuals age 65-69,

our estimate of the effect of education on cognition is statistically indistinguishable from zero. However, an additional year of education reduces the probability of dementia by 3.2 percentage points for 70–79-year-olds and by 4.6 percentage points for those 80 years and older. This pattern reverses for mild cognitive impairment.

We next consider whether the effects of education may vary by genetic predisposition for Alzheimer's disease, with the results shown in Figure 5 and Table 5. As with exposure to pollution, we find little variation in the effects of education by individuals' genetic risk. As above, we interpret these results cautiously, as our interacted instruments are weak by conventional thresholds for F-statistics.

5.3 Comparing Sources of Cognitive Decline

Finally, we consider the effects of education and PM2.5 jointly. Table 4 shows the results. As in our interacted estimates for education, we interpret these more cautiously, as our instruments are weak by conventional thresholds.

Despite the concerns about instrument relevance, one result is striking: including both instruments jointly has little effect on our point estimates. In our joint estimation, the effect of education on normal cognition and mild cognitive impairment is persistent, increasing the probability of normal cognition by 7.1 percentage points and reducing mild cognitive impairment by 4.8 percentage points. Our estimated effect of education on dementia is nearly identical to our separate estimation but is slightly noisier and insignificant. Likewise, we estimate that a 1 standard deviation increase in PM2.5 exposure increases the probability of dementia by approximately 1.6 percentage points, very close to the estimate from pollution included independently (1.5 percentage points). Taken together, these findings suggest that both education and PM2.5 are

consequential social and environmental sources of cognitive function but operate largely independently from one another.

5.4 Robustness

We conduct several robustness checks of our results. Appendix Table A.3 and A.4 show our first set of results. Columns (1) and (2) shows the OLS and IV estimates from our primary specification noted above. The remaining columns show various robustness checks. First, we re-estimate the main findings controlling for state-year time trends to rule out the possibility that secular trends in states that become disproportionately more educated or less polluted are driving our results. In column (3) of each table, we show that our results are robust to including these controls; in the case of pollution, our point estimates are larger, suggesting that secular trends push against our findings.

Next, we include controls for same-day meteorological conditions to rule out the possibility that our estimates are driven by transient changes in the environment the day of the interview. To do so, we use the HRS Date of Interview file to identify the exact date of each interview. We then link individuals to EPA data that allows us to measure day-to-day changes in meteorological conditions (e.g., wind speed, temperature, etc.) and pollutants (e.g., PM2.5, sulfur dioxide, etc.).[20] In column (4) of each table, we show that our estimates are robust to including controls for same-day conditions that include a daily air quality index, daily temperature, and daily PM2.5. Indeed, the effect of same day weather conditions is small and statistically insignificant. Our results are not driven by the environment on the exact day that individuals are interviewed.

[20] These data are available from the EPA: https://aqs.epa.gov/aqsweb/airdata/download_files.html.

Third, for our education analysis, we consider an alternative measure of education, whether the individual received any college. In Appendix Table A.5, we show that our estimates using this variable are similar in direction, but are much noisier and our instruments weaker; we therefore use years of education as our primary specification.

6 Accounting for Changes in Cognition since 2000

We use the estimates from Section 5 to quantify the contribution of PM2.5 and education to declines in dementia prevalence since 2000. We draw the PM2.5 data from our population-weighted totals in Figure 2, and the education distribution from the HRS. We focus our pollution results on changes in same-year PM2.5 because we find no significant effects of long-run exposure to PM2.5 in our main analysis.

Figure 6 shows observed and predicted dementia prevalence under varying scenarios. In navy, we show predicted dementia prevalence held fixed at 2000 levels. The red series shows observed changes in the age-sex adjusted prevalence of dementia. The remaining three lines show various alternative scenarios. In yellow, we show predicted dementia prevalence adding only the gains from reducing same-year PM2.5. In green, we do the same adding only the gains from increasing education levels. The two are roughly the same magnitude. In purple, we show the gains from adding both.

As is clear from the figure, reductions in PM2.5 and increases in education are sufficient to explain over 100 percent of the decline in age-sex adjusted dementia prevalence. Both series of predicted prevalence end at approximately the same value as observed dementia prevalence in our data. All else equal, we estimate that absent these reductions in PM2.5 and increases in education, dementia prevalence would be approximately 25% higher.

When we combine the effect of PM2.5 and education, we estimate predicted dementia prevalence lower than observed dementia rates. This suggests that other forces increasing dementia prevalence may counteract the gains from cleaner air and rising education.

7 Conclusion

As the number of individuals with cognitive impairment continues to rise, so too will the importance of policies designed to help improve cognitive function. In this paper, we offer new evidence examining the sources of the United States' striking decline in age-sex adjusted dementia prevalence. We study the effect of rising education levels and falling air pollution levels on cognition. We do so by combining detailed geographic, historical, and cognition data from the HRS with instruments that we construct for educational attainment and pollution exposure to estimate the causal effects of these hypothesized contributors to cognitive function.

We reach three primary conclusions. First, reductions in air pollution and increases in education have both increased cognitive function. A 1 standard deviation reduction in air pollution reduces dementia rates by approximately 17%, while an additional year of schooling reduces dementia rates by approximately 26%. These estimates are robust to including a variety of fixed effects, individual-level controls, time trends, and controls for day of survey meteorological conditions. Second, the effects of education and PM2.5 on dementia increase monotonically with age but exhibit little variation across genetic predisposition for Alzheimer's disease. Third, increases in education and declines in PM2.5 independently account for nearly all of the decline in dementia prevalence in the United States from 2000-2016. In fact, assuming these effects are additive suggests that dementia rates should be even lower than what we observe in the data, suggesting that other forces are pushing dementia rates up.

Our analysis is limited in a few important ways. First, our education analysis only allows us to assess the importance of education early in life, rather than throughout the life course. Future work should examine the importance of continuing education and different activities in later life as contributors to 'cognitive reserve' that protects against the onset of cognitive decline. Second, we have limited ability to examine effects for certain subgroups of interest. For example, we lack the power to estimate effects for certain racial and ethnic subgroups, and we are restricted to examining individuals' pollution exposure only where they live, as opposed to where they work. Each of these issues is important for future work.

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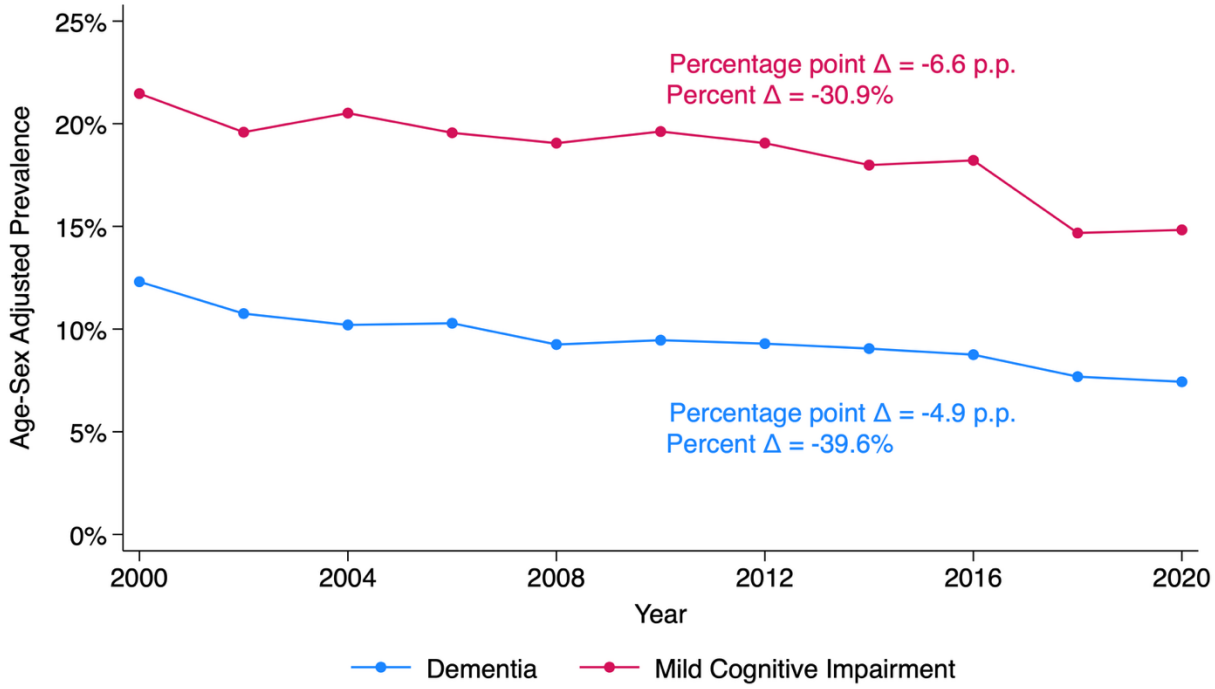
Tables and Figures

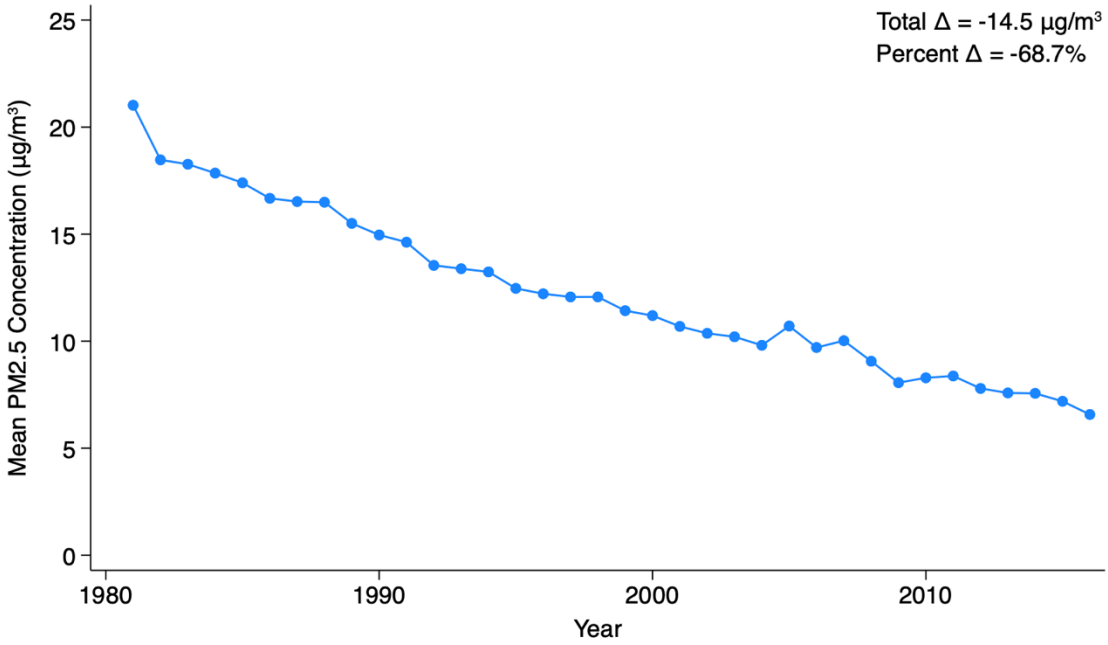
Figure 1. Age-Sex Adjusted Trends in Cognitive Impairment, 2000-2020



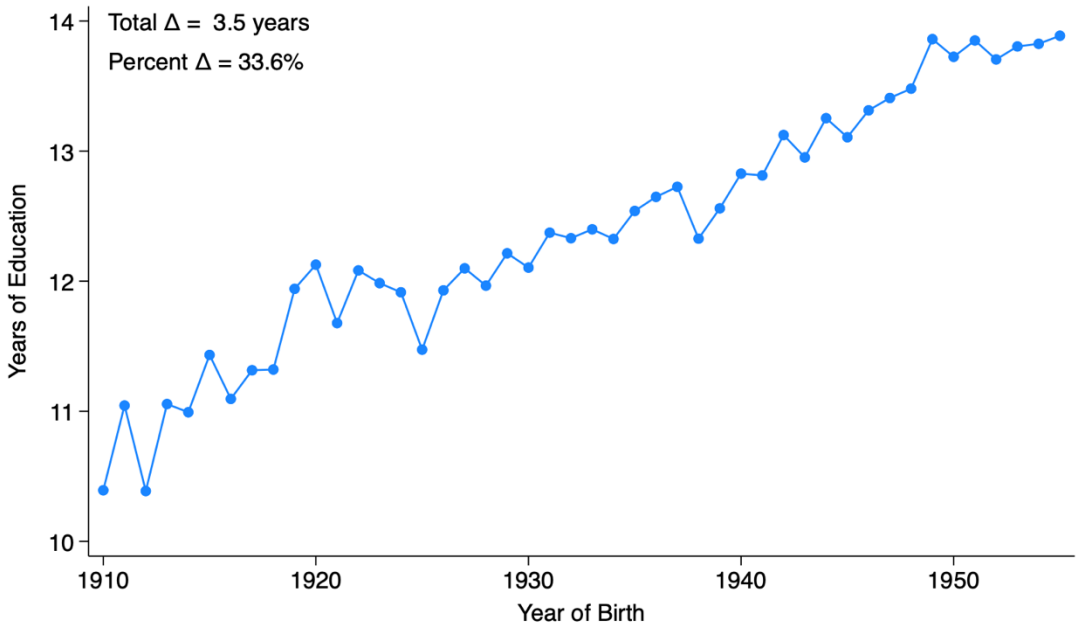
Notes: Figure shows the change in the age-sex adjusted prevalence of mild cognitive impairment (red) and dementia (blue), using publicly available HRS data from Langa et al. (2020).

Figure 2. Trends in PM2.5 Concentration and Educational Attainment

(a) PM2.5 Concentration (μg/m^3)



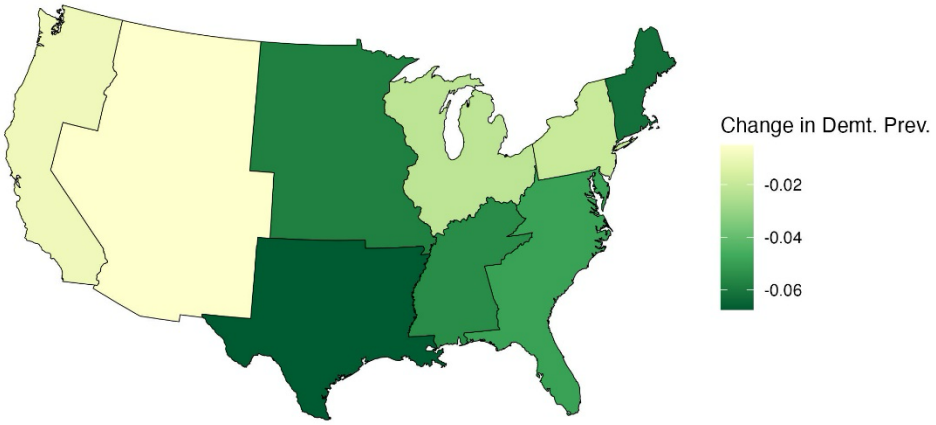
(b) Educational Attainment by Birth Cohort



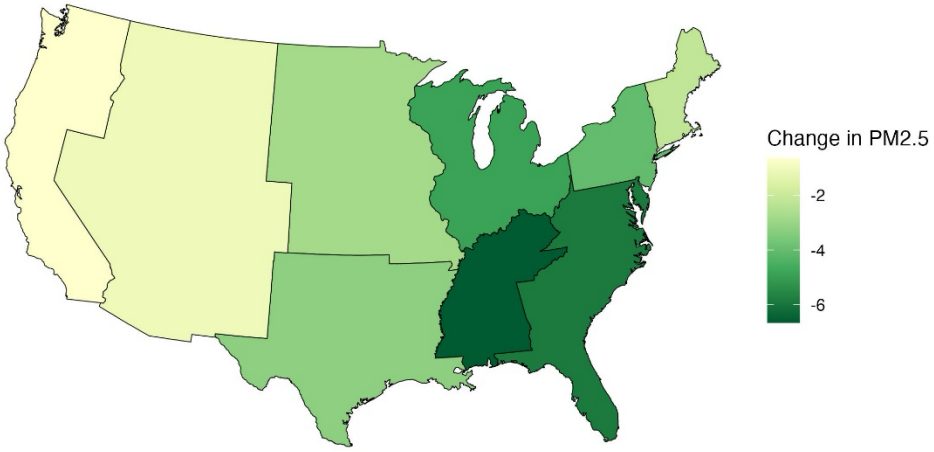
Notes: Panel A shows the decline in population-weighted PM2.5 concentration (μg/m^3) from 1981-2016 in the United States using data from Meng et al. (2019). Estimates are weighted by county population. Panel B shows educational attainment by birth cohort using data from the Health and Retirement Study.

Figure 3. Spatial Distribution of Changes in Dementia, PM2.5, and Education

(a) Age-Sex Adjusted Dementia Prevalence



(b) PM2.5



(c) Years of Education

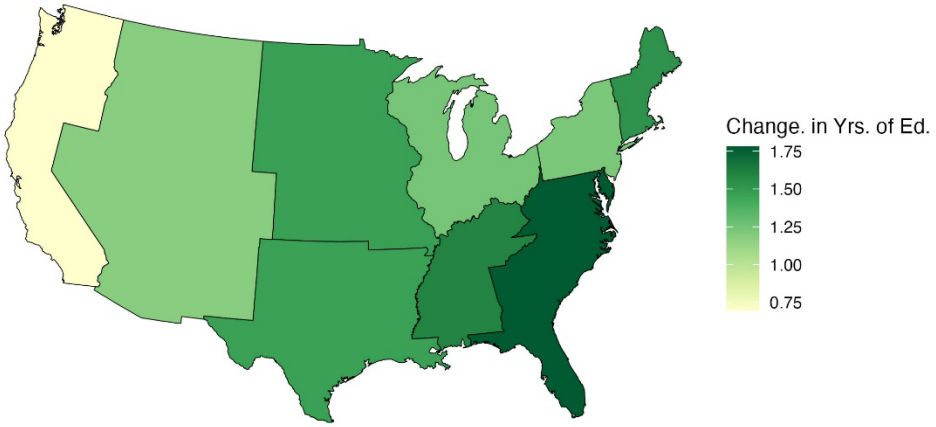
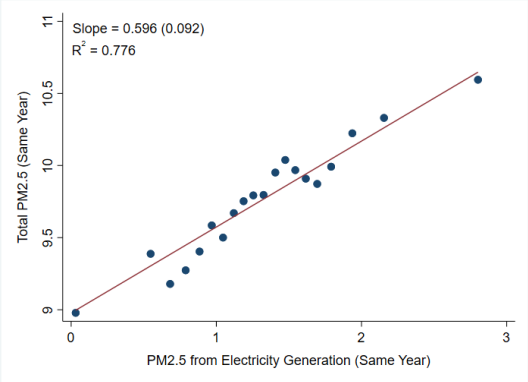
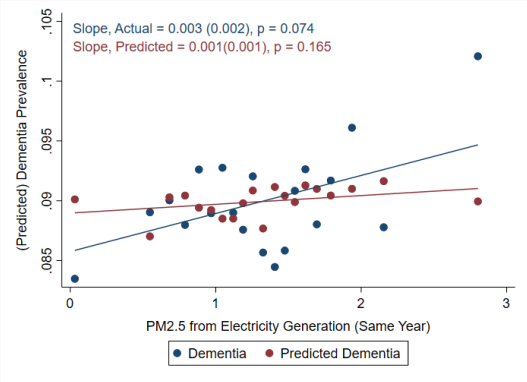


Figure 4. First Stage, Reduced Form, and Instrument Balance

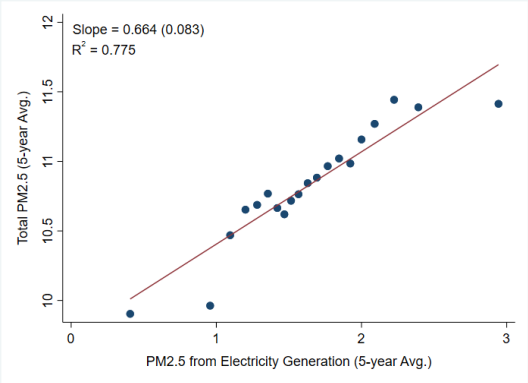
(a) First-Stage: Same Year PM2.5



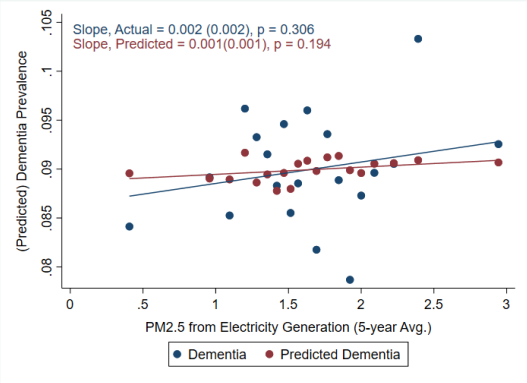
(b) Reduced Form and Balance: Same-Year PM2.5



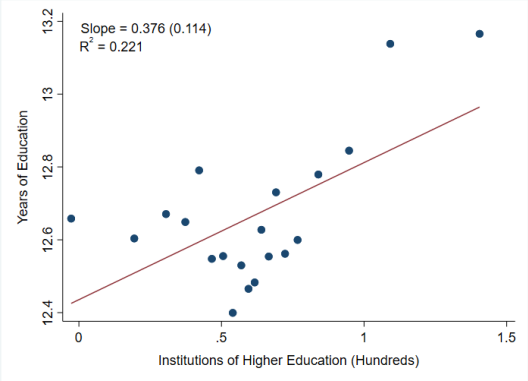
(c) First-Stage: 5-Year PM2.5



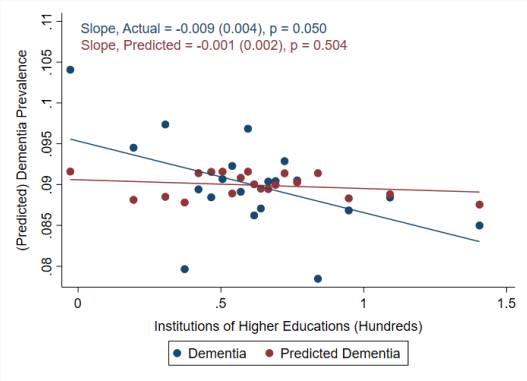
(d) Reduced Form and Balance: 5-Year PM2.5



(e) First-Stage: Years of Education



(f) Reduced Form and Balance: Years of Education

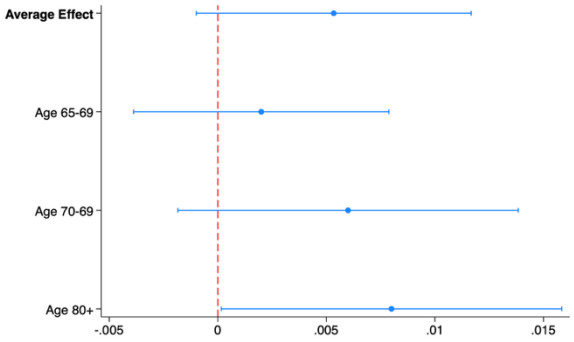


Notes: Figures show tests of IV assumptions. Panels (a), (c), and (e) show the first-stage relationship between our instrument (x-axis) and endogenous variable (y-axis). The slope of the first-stage relationship is shown in the top left corner, with standard errors in parentheses. Panels (b), (d), and (f) show first show the reduced form relationship between our instrument and observed dementia (blue). Next, these panels show the relationship between our instrument and a predicted measure of dementia using held-out measures of childhood health from the HRS (red). The slope and standard error of each of these relationships is shown in the top left corner.

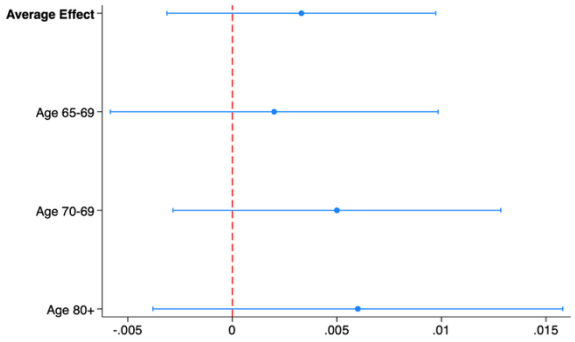
Figure 5. Effects of PM2.5 and Education on Dementia by Age and Genetics

(a) Heterogeneity by Age

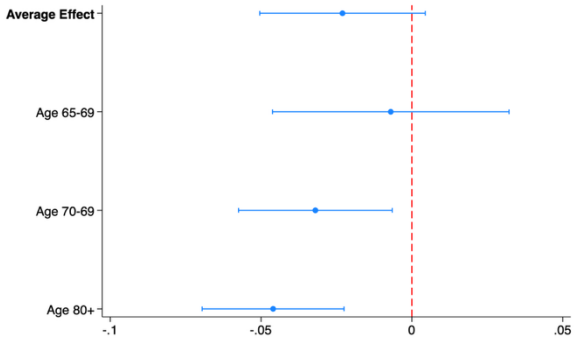
Same-Year PM2.5



5-Year PM2.5

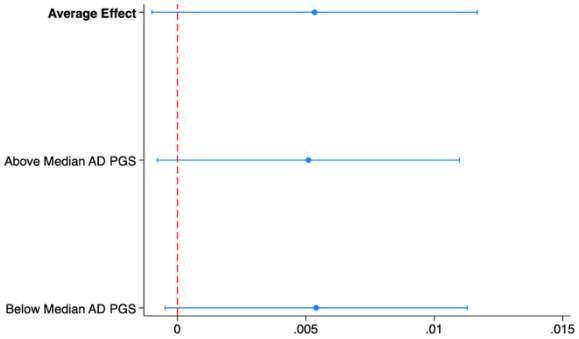


Years of Education

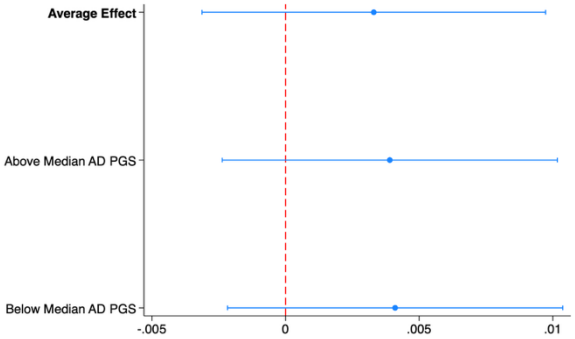


(b) Heterogeneity by Genetic Predisposition for ADRD

Same-Year PM2.5



5-Year PM2.5



Years of Education

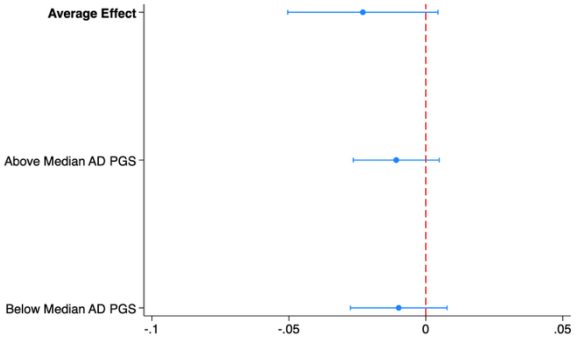
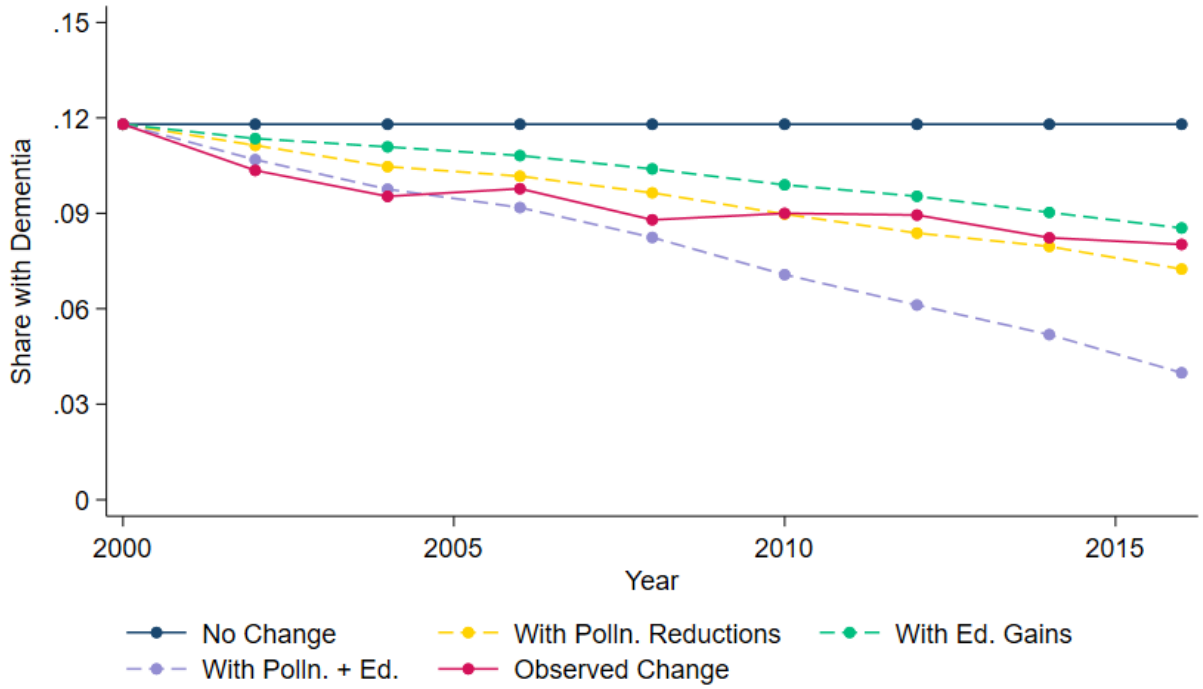


Figure 6. Counterfactual Changes in Dementia



Notes: Figure shows observed changes in age-sex adjusted dementia prevalence (red line) compared to various counterfactual scenarios. Navy line shows fixed age-sex-adjusted dementia prevalence from 2000 onwards. Yellow line shows prevalence accounting for reductions in same-year PM2.5 exposure, computed by scaling year-over-year changes in PM2.5 by IV estimate in Table 2. Green line shows prevalence accounting for improvements in education, computed by scaling year-over-year changes in educational attainment by IV estimates in Table 3. Purple line shows prevalence accounting for improvements in education and same-year PM2.5 combined, assuming additive effects.

Table 1. Summary Statistics

	Full Estimation Sample (1)	Genetics Subsample (2)
Demographics		
Age	75.044	74.555
	(7.619)	(7.354)
Female	0.573	0.573
	(0.495)	(0.495)
Non-Hispanic White	0.864	0.918
	(0.343)	(0.275)
Non-Hispanic Black	0.086	0.082
	(0.280)	(0.275)
Non-Hispanic Other Race	0.014	0.000
	(0.118)	(0.000)
Hispanic	0.036	0.000
	(0.187)	(0.000)
Pollution Exposure		
Same-Year PM2.5	9.760	9.439
	(3.017)	(2.832)
5-Year PM2.5	10.829	10.506
	(3.251)	(3.081)
Education		
Years of Education	12.668	13.026
	(2.949)	(2.673)
HS or Less	0.567	0.530
	(0.496)	(0.499)
Some College	0.214	0.228
	(0.410)	(0.419)
College or More	0.219	0.242
	(0.414)	(0.429)
Cognition		
Normal Cognition	0.724	0.783
	(0.447)	(0.412)
CIND	0.186	0.166
	(0.389)	(0.372)
Dementia	0.090	0.052
	(0.286)	(0.221)
N (Person-Years)	82,908	54,333
Unique Individuals	18,290	9,861

Table 2. Effect of PM2.5 on Cognition

	Normal Cognition		CIND		Dementia	
	OLS	IV	OLS	IV	OLS	IV
	(1)	(2)	(1)	(2)	(1)	(2)
Panel A. Same-Year						
Total PM2.5	0.00138	-0.00422	-0.00277**	-0.00112	0.00138	0.00534*
	(0.00171)	(0.00630)	(0.00134)	(0.00590)	(0.00086)	(0.00323)
Effective F-Statistic		42.587		42.587		42.587
R^2	0.212	0.180	0.083	0.064	0.139	0.114
Panel B. 5-Year						
Total PM2.5	0.00026	-0.00234	-0.00212	-0.00095	0.00186**	0.00330
	(0.00163)	(0.00599)	(0.00129)	(0.00564)	(0.00081)	(0.00328)
Effective F-Statistic		64.375		64.375		64.375
R^2	0.212	0.181	0.083	0.064	0.139	0.114
Age-Sex Cells	Y	Y	Y	Y	Y	Y
Year Fixed Effects	Y	Y	Y	Y	Y	Y
CZ Fixed Effects	Y	Y	Y	Y	Y	Y
Mean	0.724	0.724	0.186	0.186	0.090	0.090
SD	0.447	0.447	0.389	0.389	0.286	0.286
N	82908	82908	82908	82908	82908	82908
Unique Individuals	18290	18290	18290	18290	18290	18290

Table 3. Effect of Education on Cognition

	Normal Cognition		CIND		Dementia	
	OLS	IV	OLS	IV	OLS	IV
	(1)	(2)	(3)	(4)	(5)	(6)
Years of Education	0.039***	0.071***	-0.021***	-0.048***	-0.018***	-0.023*
	(0.001)	(0.015)	(0.001)	(0.013)	(0.001)	(0.014)
Age-Sex Cells?	Y	Y	Y	Y	Y	Y
Year Fixed Effects?	Y	Y	Y	Y	Y	Y
Year Age 17 Fixed Effects?	Y	Y	Y	Y	Y	Y
CZ Fixed Effects?	Y	Y	Y	Y	Y	Y
Dependent Variable Mean	0.724	0.724	0.186	0.186	0.090	0.090
Dependent Variable SD	0.447	0.447	0.389	0.389	0.286	0.286
Effective F-Statistic		10.97		10.97		10.97
R^2	0.243	0.176	0.091	0.039	0.162	0.136
N	82908	82908	82908	82908	82908	82908
Unique Individuals	18290	18290	18290	18290	18290	18290

Table 4. Comparing the Effects of PM2.5 and Education on Cognition

	Normal Cognition		CIND		Dementia	
	OLS	IV	OLS	IV	OLS	IV
	(1)	(2)	(1)	(2)	(1)	(2)
Panel A. Same-Year						
Total PM2.5	0.00080	-0.00448	-0.00261**	-0.00141	0.00181**	0.00588**
	(0.00146)	(0.00608)	(0.00121)	(0.00601)	(0.00075)	(0.00282)
Years of Education	0.03896***	0.07150***	-0.02125***	-0.04795***	-0.01771***	-0.02355
	(0.00122)	(0.01863)	(0.00103)	(0.01375)	(0.00139)	(0.01481)
F-Statistic		4.519		4.519		4.519
R^2	0.243	0.176	0.091	0.039	0.162	0.135
Panel B. 5-Year						
Total PM2.5	-0.00048	-0.00404	-0.00190*	-0.00029	0.00238***	0.00433
	(0.00130)	(0.00598)	(0.00112)	(0.00568)	(0.00077)	(0.00318)
Years of Education	0.03898***	0.07243***	-0.02125***	-0.04794***	-0.01773***	-0.02449
	(0.00122)	(0.01915)	(0.00103)	(0.01406)	(0.00139)	(0.01539)
F-Statistic		4.330		4.330		4.330
R^2	0.243	0.174	0.091	0.039	0.162	0.135
Age-Sex Cells	Y	Y	Y	Y	Y	Y
Year Fixed Effects	Y	Y	Y	Y	Y	Y
CZ Fixed Effects	Y	Y	Y	Y	Y	Y
Year Age 17 Fixed Effects	Y	Y	Y	Y	Y	Y
Mean	0.724	0.724	0.186	0.186	0.090	0.090
SD	0.447	0.447	0.389	0.389	0.286	0.286
N	82908	82908	82908	82908	82908	82908
Unique Individuals	18290	18290	18290	18290	18290	18290

Table 5. Effects of PM2.5 on Cognition by Genetic Predisposition for ADRD

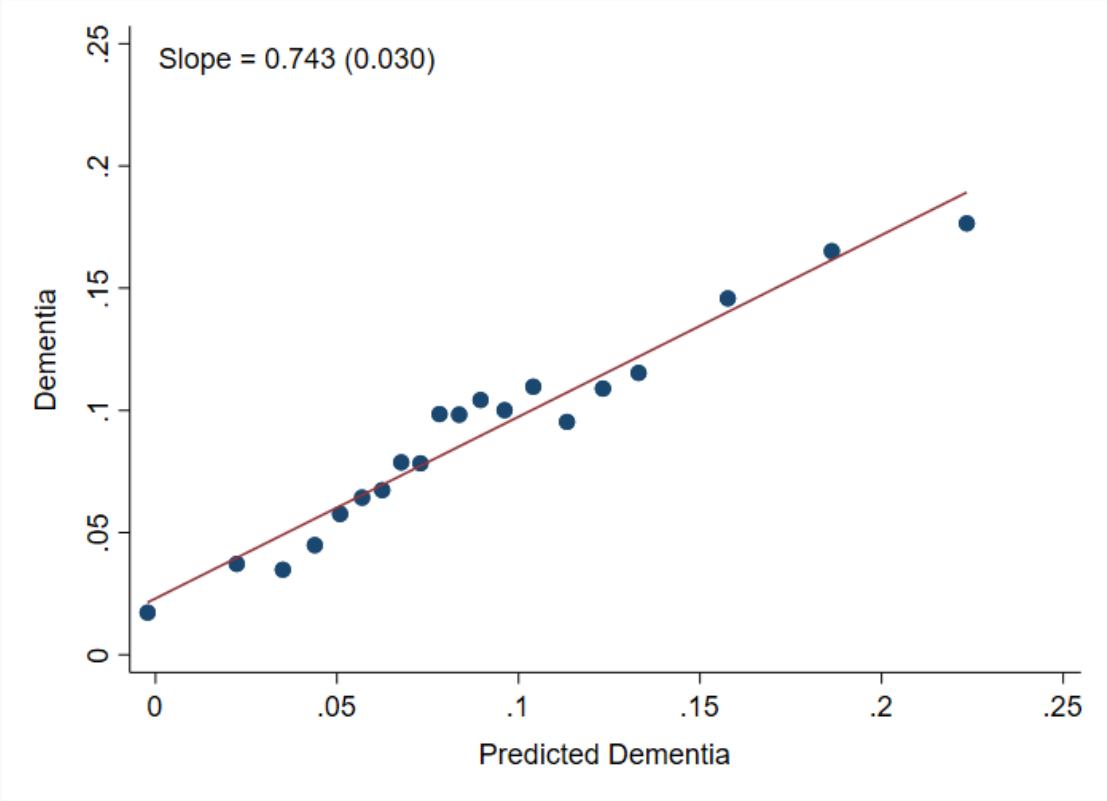
	Normal Cognition		CIND		Dementia	
	OLS	IV	OLS	IV	OLS	IV
	(1)	(2)	(1)	(2)	(1)	(2)
Panel A. Same-Year PM2.5						
Above Median AD PGS	0.0043*	-0.0082	-0.0034*	0.0031	-0.0009	0.0051*
	(0.0025)	(0.0074)	(0.0020)	(0.0069)	(0.0009)	(0.0030)
Below Median AD PGS	0.0033	-0.0108	-0.0024	0.0054	-0.0008	0.0054*
	(0.0026)	(0.0077)	(0.0020)	(0.0070)	(0.0009)	(0.0030)
AD PGS	-0.024***	-0.0299***	0.014***	0.0191***	0.010***	0.0108***
	(0.0047)	(0.0060)	(0.0039)	(0.0048)	(0.0021)	(0.0028)
KP F-Stat		31.442		31.442		31.442
R^2	0.185	0.154	0.096	0.073	0.109	0.086
Panel B. 5-Year PM2.5						
Above Median AD PGS	0.0028	-0.0083	-0.0029	0.0044	0.0001	0.0039
	(0.0022)	(0.0076)	(0.0018)	(0.0072)	(0.0008)	(0.0032)
Below Median AD PGS	0.0019	-0.0105	-0.0020	0.0064	0.0001	0.0041
	(0.0023)	(0.0076)	(0.0019)	(0.0071)	(0.0008)	(0.0032)
AD PGS	-0.028***	-0.0294***	0.017***	0.0186***	0.011***	0.0108***
	(0.0062)	(0.0061)	(0.0049)	(0.0048)	(0.0033)	(0.0028)
KP F-Stat		37.121		37.121		37.121
R2	0.185	0.154	0.096	0.073	0.109	0.086
Panel C. Years of Education						
Above Median AD PGS	0.0373***	0.0566***	-0.0252***	-0.0457***	-0.0121***	-0.0108
	(0.002)	(0.018)	(0.001)	(0.014)	(0.001)	(0.008)
Below Median AD PGS	0.0359***	0.0548***	-0.0243***	-0.0449***	-0.0116***	-0.0099
	(0.002)	(0.018)	(0.001)	(0.014)	(0.001)	(0.009)
AD PGS	-0.0255***	-0.0262***	0.0136***	0.0118***	0.0119***	0.0144***
	(0.005)	(0.005)	(0.004)	(0.004)	(0.002)	(0.003)

KP F-Stat		3.60		3.60		3.60
R^2	0.215	0.173	0.109	0.068	0.128	0.106
Age-Sex Cells	Y	Y	Y	Y	Y	Y
Year Fixed Effects	Y	Y	Y	Y	Y	Y
CZ Fixed Effects	Y	Y	Y	Y	Y	Y
Mean	0.783	0.783	0.166	0.166	0.052	0.052
SD	0.412	0.412	0.372	0.372	0.221	0.221
N	54344	54333	54344	54333	54344	54333
Unique Individuals	9865	9861	9865	9861	9865	9861

Online Appendix for Sources of Cognitive Improvements among Elderly Adults
Cutler and Peterson (2025)

Appendix A

Figure A.1. Actual versus Predicted Dementia



Notes: Figure shows observed dementia (y-axis) plotted against predicted dementia (x-axis). Predicted dementia is estimated using held-out measures of individuals' childhood health from before the age of 16. Held-out measures include indicators for having the following conditions before age 16: measles, mumps, chicken pox, difficulty seeing, asthma, diabetes, respiratory disorders, speech impairment, allergic conditions, heart trouble, ear problems, epilepsy or seizures, headaches or migraines, stomach problems, high blood pressure, depression, drug or alcohol problems, and other psychological problems.

Appendix Table A.1. Effect of PM2.5 on Cognition by Age

	Normal Cognition		CIND		Dementia	
	OLS	IV	OLS	IV	OLS	IV
	(1)	(2)	(1)	(2)	(1)	(2)
Panel A. Same-Year PM2.5						
Average Effect	**0.00138**	**-0.00422**	**-0.00277****	**-0.00112**	**0.00138**	**0.00534***
	(0.00171)	**(0.00630)**	**(0.00134)**	**(0.00590)**	**(0.00086)**	**(0.00323)**
Age 65-69	0.005***	-0.001	-0.004***	-0.002	-0.001	0.002
	(0.002)	(0.006)	(0.001)	(0.006)	(0.001)	(0.003)
Age 70-79	-0.001	-0.006	-0.001	0.000	0.002**	0.006*
	(0.002)	(0.007)	(0.001)	(0.006)	(0.001)	(0.004)
Age 80+	0.001	-0.006	-0.004**	-0.002	0.003**	0.008**
	(0.002)	(0.007)	(0.002)	(0.007)	(0.002)	(0.004)
KP F-Stat		14.308		14.308		14.308
R^2	0.212	0.181	0.084	0.065	0.139	0.114
Panel B. 5-Year PM2.5						
Average Effect	**0.00026**	**-0.00234**	**-0.00212**	**-0.00095**	**0.00186****	**0.00330**
	(0.00163)	**(0.00599)**	**(0.00129)**	**(0.00564)**	**(0.00081)**	**(0.00328)**
Age 65-69	0.004**	0.000	-0.004**	-0.002	0.000	0.002
	(0.002)	(0.007)	(0.001)	(0.007)	(0.001)	(0.004)
Age 70-79	-0.001	-0.005	-0.001	0.000	0.002***	0.005
	(0.002)	(0.007)	(0.001)	(0.007)	(0.001)	(0.004)
Age 80+	-0.000	-0.004	-0.004**	-0.002	0.004***	0.006
	(0.002)	(0.008)	(0.001)	(0.007)	(0.001)	(0.005)
KP F-Stat		17.987		17.987		17.987
R2	0.212	0.181	0.084	0.065	0.139	0.115
Age-Sex Cells	Y	Y	Y	Y	Y	Y
Year Fixed Effects	Y	Y	Y	Y	Y	Y
CZ Fixed Effects	Y	Y	Y	Y	Y	Y
N	82908	82908	82908	82908	82908	82908
Unique Individuals	18290	18290	18290	18290	18290	18290

Appendix Table A.2. Effect of Education on Cognition by Age

	Dependent Variable					
	Normal Cognition		CIND		Dementia	
	OLS	IV	OLS	IV	OLS	IV
	(1)	(2)	(3)	(4)	(5)	(6)
Years of Education						
Average Effect	**0.039*** **	**0.071*** **	**-0.021*** **	**-0.048*** **	**-0.018*** **	**-0.023***
	(0.001)	**(0.015)**	**(0.001)**	**(0.013)**	**(0.001)**	**(0.014)**
Age 65-69	0.035***	0.068***	-0.027***	-0.061***	-0.008***	-0.007
	(0.002)	(0.017)	(0.001)	(0.018)	(0.001)	(0.020)
Age 70-79	0.042***	0.076***	-0.027***	-0.044**	-0.015***	-0.032**
	(0.002)	(0.017)	(0.001)	(0.016)	(0.002)	(0.013)
Age 80+	0.038***	0.070***	-0.010***	-0.024	-0.028***	-0.046***
	(0.001)	(0.020)	(0.002)	(0.015)	(0.002)	(0.012)
Age-Sex Cells?	Y	Y	Y	Y	Y	Y
Year Fixed Effects?	Y	Y	Y	Y	Y	Y
Year Age 17 Fixed Effects?	Y	Y	Y	Y	Y	Y
CZ Fixed Effects?	Y	Y	Y	Y	Y	Y
Mean	0.724	0.724	0.186	0.186	0.090	0.090
SD	0.447	0.447	0.389	0.389	0.286	0.286
KP F Stat		3.88		3.88		3.88
R^2	0.244	0.176	0.094	0.053	0.168	0.125
N	82908	82908	82908	82908	82908	82908
Unique Individuals	18290	18290	18290	18290	18290	18290

Appendix Table A.3. Robustness – Effect of Same-Year PM2.5 on Cognition

	Effect of PM2.5 on Cognition			
	(1)	(2)	(3)	(4)
Panel A. Normal Cognition				
Total PM2.5	0.0014	-0.0042	-0.0010	-0.0053
	(0.0017)	(0.0063)	(0.0081)	(0.0064)
Same-Day PM2.5				-0.0010
				(0.0006)
DV Mean (SD)	0.724 (0.447)	0.724 (0.447)	0.724 (0.447)	0.724 (0.447)
R^2	0.212	0.180	0.18	0.18
Panel B. CIND				
Total PM2.5	-0.0028**	-0.0011	-0.0073	-0.0006
	(0.0013)	(0.0059)	(0.0073)	(0.0059)
Same-Day PM2.5				0.0005
				(0.0005)
DV Mean (SD)	0.186 (0.389)	0.186 (0.389)	0.186 (0.389)	0.186 (0.389)
R^2	0.083	0.064	0.06	0.06
Panel C. Dementia				
Total PM2.5	0.0014	0.0053*	0.0083**	0.0059*
	(0.0009)	(0.0032)	(0.0040)	(0.0033)
Same-Day PM2.5				0.0005
				(0.0005)
DV Mean (SD)	0.090 (0.286)	0.090 (0.286)	0.090 (0.286)	0.090 (0.286)
R^2	0.139	0.114	0.11	0.11
Age-Sex Cells	Y	Y	Y	Y
Year Fixed Effects	Y	Y	Y	Y
CZ Fixed Effects	Y	Y	Y	Y
State Time Trends			Y	
Same-Day Conditions				Y
Effective F-Statistic		42.587	42.587	42.587
N	82908	82908	82908	82908
Unique Individuals	18290	18290	18290	18290

Appendix Table A.4. Robustness – Effect of Education on Cognition

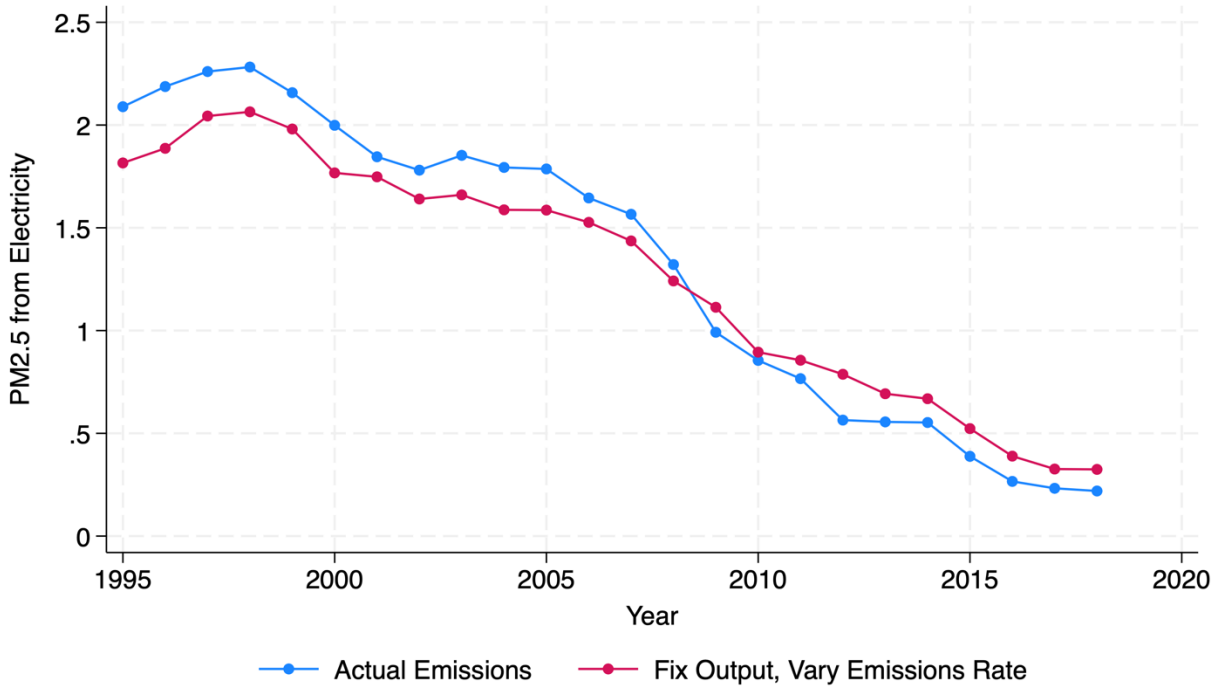
	Effect of Education on Cognition			
	(1)	(2)	(3)	(4)
Panel A. Normal Cognition				
Years of Education	0.0390***	0.0713***	0.0732***	0.0708***
	(0.00115)	(0.01491)	(0.01613)	(0.01509)
Same-Day PM2.5				-0.0008
				(0.00069)
DV Mean (SD)	0.724 (0.447)	0.724 (0.447)	0.724 (0.447)	0.724 (0.447)
R^2	0.243	0.176	0.17	0.18
Panel B. CIND				
Years of Education	-0.0213***	-0.0480***	-0.0501***	-0.0481***
	0.00095761	0.012989362	0.012375559	0.013032041
Same-Day PM2.5				0.0004
				(0.00061)
DV Mean (SD)	0.186 (0.389)	0.186 (0.389)	0.186 (0.389)	0.186 (0.389)
R^2	0.091	0.039	0.03	0.04
Panel C. Dementia				
Years of Education	-0.0177***	-0.0233*	-0.0230*	-0.0227*
	(0.00135)	(0.01369)	(0.01340)	(0.01354)
Same-Day PM2.5				0.0004
				(0.00045)
DV Mean (SD)	0.090 (0.286)	0.090 (0.286)	0.090 (0.286)	0.090 (0.286)
R^2	0.162	0.136	0.14	0.14
Age-Sex Cells	Y	Y	Y	Y
Year, Year Age 17 Fixed Effects	Y	Y	Y	Y
CZ Fixed Effects	Y	Y	Y	Y
State Time Trends			Y	
Same-Day Conditions				Y
Effective F-Statistic		10.974	10.974	10.974
N	82908	82908	82908	82908
Unique Individuals	18290	18290	18290	18290

Appendix Table A.5. Effect of Education on Cognition, Using Any College (0/1) as Endogenous Variable

	Normal Cognition		CIND		Dementia	
	OLS	IV	OLS	IV	OLS	IV
	(1)	(2)	(1)	(2)	(1)	(2)
Any College	0.148***	0.549***	-0.097***	-0.370***	-0.050***	-0.179
	(0.006)	(0.170)	(0.004)	(0.132)	(0.004)	(0.113)
Age-Sex Cells?	Y	Y	Y	Y	Y	Y
Year Fixed Effects?	Y	Y	Y	Y	Y	Y
Year Age 17 Fixed Effects?	Y	Y	Y	Y	Y	Y
CZ Fixed Effects?	Y	Y	Y	Y	Y	Y
Mean	0.724	0.724	0.186	0.186	0.090	0.090
SD	0.447	0.447	0.389	0.389	0.286	0.286
Effective F-Statistic		8.87		8.87		8.87
R^2	0.215	0.008	0.083	-0.040	0.142	0.075
N	82908	82908	82908	82908	82908	82908
Unique Individuals	18290	18290	18290	18290	18290	18290

Appendix B. Data Appendix

Appendix Figure B.1. PM2.5 from Electricity Under Different Emissions Scenarios



Notes: Figure shows the estimated change in PM2.5 from electricity generation in the United States from 1995-2018. The blue series shows estimated changes in PM2.5 using true electricity generating unit (EGU) emissions as inputs to the InMAP model to generate PM2.5 estimates (Tessum et al., 2017). The red series shows estimated changes in PM2.5 using the emissions stripped of demand variation as inputs to the InMAP model. To strip the emissions of demand variation, we fix a given EGU's output at its second year of operation and allow the rate at which it emits a given pollutant to vary over time. For each year and pollutant, we then compute the emissions stripped of demand variation as the product of the time-invariant output and the time-varying pollutant-specific emissions rate.