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Air Quality, Human Capital Formation and the Long-term Effects of Environmental Inequality at Birth

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Air Quality, Human Capital Formation and the Long-term Effects of Environmental Inequality at Birth*

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Abstract

A growing body of literature suggests that pollution exposure early in life can have substantial long term effects on an individual's economic well-being as an adult, however the mechanisms for these effects remain unclear. I contribute to this literature by examining the effect of pollution exposure on several intermediate determinants of adult wages using a unique linked dataset for a large sample of individuals from two cohorts: an older cohort born around the 1970, and a younger cohort born around 1990. This dataset links responses to the American Community Survey to SSA administrative data, the universe of IRS Form 1040 tax returns, pollution concentration data from EPA air quality monitors and satellite remote sensing observations. In both OLS and IV specifications, I find that pollution exposure at birth has a large and economically significant effect on college attendance among 19-22 year olds. Using conventional estimates of the college wage premium, these effects imply that a 10 $\mu g/m^3$ decrease in particulate matter exposure at birth is associated with a \$190 per year increase in annual wages. This effect is smaller than the wage effects in the previous literature, which suggests that human capital acquisition associated with cognitive skills cannot fully explain the long term wage effects of pollution exposure. Indeed, I find evidence for an additional channel working through non-cognitive skill—pollution exposure at birth increases high school non-completion and incarceration among 16–24 year olds, and that these effects are concentrated within disadvantaged communities, with larger effects for non-whites and children of poor parents. I also find that pollution exposure during adolescence has statistically significant effects on high school non-completion and incarceration, but no effect on college attendance. These results suggest that the long term effects of pollution exposure on economic well-being may run through multiple channels, of which both non-cognitive skills and cognitive skills may play a role.

Keywords: air quality, human capital

JEL classification: J24, J31,Q51,Q52, Q53

*This paper is released to inform interested parties of research and to encourage discussion. The views expressed are those of the author and not necessarily those of the U.S. Census Bureau.

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1 Introduction

Over the last 40 years, ambient air quality in the United States has improved at a remarkable rate, even while economic growth has continued its upward trajectory. These improvements in air quality are widely attributed to policies enacted as part of the Clean Air Act of 1970 (CAA), and later amendments in 1977 and 1990 (Ross et al. (2012), Kahn (2001), EPA (2016)). These policies were enacted largely as a response to concerns about the (largely short-term) health effects of pollution exposure (Ahlers (2015)). What was not clear at the time of their passage, but is now an active area of investigation, is that pollution exposure, especially very early in life (and even while a child is in utero) can have substantial long term impacts not just on health, but on economic well being–labor market participation and wages–well into adulthood. Put another way, there may be substantial long term benefits to pollution reduction which were not readily apparent when policy-makers were designing the CAA and amendments.

In this paper, I leverage a novel linked data set which provides substantial advantages over the datasets used in the small but growing literature examining these long term economic impacts. Rather than examining the direct impact of pollution exposure in early life on adult wages, I focus on intermediate determinants of these wages, including both outcomes that likely work through cognitive skills (human capital formation in the form of college attendance) as well as outcomes that likely work through non-cognitive skills (high school non-completion and incarceration). As noted by Almond et al. (2017), there is a "missing middle" in the literature on the long term effects of pollution exposure at birth, both in the sense that intermediate pollution exposure is unobserved, and in the sense that, since intermediate outcomes, notably human capital accumulation, are unobserved, the mechanisms linking pollution exposure at birth, early life human capital and adult economic well being are mysterious. This study, then, takes up this mantle, by 1) directly examining how pollution exposure at birth affects human capital formation, with attention to pathways working through cognitive vs. non-cognitive skills and 2) leveraging linked survey and administrative data to separately examine how pollution exposure during adolescence affects potential pathways from early life exposure to adult economic well being.

This approach complements the recent work of Isen et al. (2016), who find that the sharp reduction in particulate pollution associated with the CAA 1970 had a large and positive effect on wages, and, among others, Bharadwaj et al. (2014) who present quasi-experimental evidence that pollution exposure at birth has a negative effect on primary school test scores. This paper adds to a growing literature that has examined the short and long term impacts of the environment in utero and at birth using quasi-experimental methods. Much of this literature in economics, motivated by concerns about "inequality at birth" (Currie (2011),

Ferrie et al. (2015)) initially focused on infant health and mortality (e.g. Chay and Greenstone (2003a), Chay and Greenstone (2003b),Currie et al. (2009), Currie and Walker (2011), Currie (2013)). However, this interest has broadened, largely as a result of increased data availability and innovations in quasi-experimental methods of identifying causal effects to include assessments of longer term health impacts, as well as at least some study of the impact of pollution exposure on educational attainment, and, as noted earlier, on wages. A related series of papers (e.g., Chang et al. (2014), Chang et al. (2016)) has also documented short term effects of pollution exposure on individual economic activity (e.g., productivity) among adults.

The literature closest to the current project has largely focused on the effects of exposure to various pollutants or other environmental hazards on early life human capital, typical embodied by test scores in elementary school. Several papers have focused on environmental outcomes at birth that may be substantially different than ambient air quality, such as radioactive fallout (Almond et al. (2009), Black et al. (2013)), exposure to lead through paint or soil (Aizer et al. (2016), Reyes (2014)) or proximity to toxic waste sites (Rau et al. (2015), Persico et al. (2016)). Only a few studies have examined how ambient air pollution exposure either contemporaneous (Lavy et al. (2014)) or in early life (Sanders (2012), Bharadwaj et al. (2014)) might affect educational outcomes in elementary or secondary school. One other study, Marcotte (2017) examines the separate effects of early life and contemporaneous exposure on early life human capital.

I contribute to the literature along a number of dimensions. First, in the context of long term effects of pollution exposure, college attendance has not been thoroughly studied, either as an outcome in and of itself or as a pathway through which pollution might affect other economic outcomes, while high school non-completion has not been studied at all, and incarceration has not been directly studied (although there is a literature on the connection between pollution exposure and crime).[1] Second, I introduce a novel data resource–the U.S. Census Bureau's data linkage infrastructure to the literature. This data linkage infrastructure allows me to construct a dataset which links survey responses from the 2005 through 2015 American Community Survey (ACS)[2] with administrative data from the Social Security Administration (SSA) on exact date and place of birth, and to the universe of Form 1040 tax returns from the Internal Revenue Service (IRS). The latter linkage provides a unique opportunity to control for observable characteristics of individuals' environment later in life (during their secondary education), an important set of confounding variables which would have been unobservable in previous studies. This linked dataset, and the broader linkage infrastructure provided by the US Census Bureau, provides a number of advantages over previously

[1] Only one other study, Peet (2015) addresses the effect of pollution exposure on college attendance, in a substantially different setting (the Philippines)

[2] ACS data are based on a sample and are subject to sampling variability. For more information see `https://www.census.gov/programs-surveys/acs/`

used datasets, and has the potential to be an important tool moving forward for this literature.[3]

Using this linked data, I examine the effect of exposure to pollution in utero and in early life on several intermediate determinants of adult wages: college attendance at ages 19–22, high school non-completion, and incarceration. I utilize two identification strategies: a modified version of the fixed effects strategy used in Bharadwaj et al. (2014), with the addition of controls for observable and unobservable adolescent environment characteristics, and an instrumental variables (IV) strategy similar to Chay and Greenstone (2003a) and Isen et al. (2016), using nonattainment designations that occurred after the enactment of the the Clean Air Act Amendments of 1990 (CAAA) as an instrument for pollution levels. Using this data and these strategies, I find strong evidence for an economically and statistically significant effect of particulate matter exposure, both in utero and during the first year of life, on college attendance at ages 19–22. Using these estimates, a back of the envelope calculation suggests that a 10 $\mu g/m^3$ decrease in TSP would increase wages by about \$175 per year, a wage effect less than three quarters of the baseline effect in Isen et al. (2016) (an effect I replicate using the linked ACS-SSA data). This suggests that a cognitive skills driven human capital channel, although important, cannot fully explain the wage effects. Indeed, I find that pollution exposure at birth has strong and consistent effects on high school non-completion and incarceration, which suggests that there may be an additional channel working through non-cognitive skills.

The remainder of the paper is organized as follows. Section 2 presents a conceptual model for thinking about the various pathways through which pollution exposure in early life can affect adult outcomes. Section 3 describes the data linkage process and the linked dataset used for estimation, and describes the identification strategies used. Section 4 replicates the Isen et al. (2016) long term wage effects using the linked ACS-SSA data for an older cohort. Section 5 presents results on the effect of pollution exposure on college attendance, and produces back of the envelope calculations of the implied wage premium due to air quality improvements as a result of the CAAA 1990. Section 6 examines the effects of pollution exposure on high school non-completion. Section 7 considers how pollution exposure affects incarceration. Section 8 concludes with a discussion of potentially fruitful future avenues of research on the long term effects of pollution exposure.

[3]The two other previously used data sources are the Panel Study of Income Dynamics, which is small and no longer representative of the current US population, and the Longitudinal Employer-Household Dynamics data used by Isen et al. (2016). Relative to these, the advantages of the linked data used here are: coverage (Isen et al. (2016)'s estimating sample includes only 24 states, compared to the nationally representative ACS), richer demographic information from the survey, a wider variety of outcomes observable (not just wages and labor force participation) and finer grained information on exact date of birth. Additionally, the linkage infrastructure provided by the Census Bureau offers the possibility of linking to other administrative and survey microdata, extending the possible set of questions that can be answered.

2 Conceptual Model

To clarify thought about how early life and later life pollution exposure might affect human capital formation, I present a simplified model of human capital accumulation, which is a modification of the model in Currie et al. (2013). This model links pollution exposure at various points in an individual's life to their human capital investments, and ultimately, to their wages as a working adult. This clarifies which relationships are of interest, and results in testable hypotheses. For simplicity, this model assumes a representative agent.

I begin with a Mincerian wage regression describing how wages relate to human capital:

$$W_A = f_w(H_A, X)$$

Where W_A is an individual's adult wage rate, H_A is her amount of adult human capital (which we will take as the amount of post-secondary education), and X represents time-invariant characteristics. Each individual's adult human capital, however, is also dependent both on the level of human capital investment in childhood, as well as accumulated environmental hazards.

For now, we assume that adult human capital takes the form:

$$H_A = f_A(H_L, H_E, P_L, X)$$

Where H_L is human capital accumulated in late childhood (i.e. high school completion), H_E is human capital accumulated in early childhood and P_L is pollution exposure in late childhood. Late-childhood human capital accumulation in turn depends on pollution exposure in later childhood, as well as parental investment which is related to early childhood human capital:

$$H_L = f_L(I(H_E), P_L)$$

Finally, early childhood human capital depends on both in-utero and early childhood pollution exposure, as well as fixed characteristics:

$$H_E = f_E(P_E, P_U, X)$$

Where P_U is in-utero exposure, and P_E is exposure after birth in early childhood.

This model then implies three different relationships between exposure over the lifespan and human capital. First, substituting and differentiating reveals that the relationship between in utero exposure and

adult human capital is:

$$\frac{\partial H_A}{\partial P_U} = \frac{\partial f_A}{\partial H_E}\frac{\partial H_E}{\partial P_U} + \frac{\partial f_A}{\partial H_L}\frac{\partial I}{\partial H_E}\frac{\partial H_E}{\partial P_U} = (\frac{\partial f_A}{\partial H_E} + \frac{\partial f_A}{\partial H_L}\frac{\partial I}{\partial H_E})\frac{\partial H_E}{\partial P_U}$$

Similarly, the effect of early life exposure on adult human capital is

$$\frac{\partial H_A}{\partial P_E} = \frac{\partial f_A}{\partial H_E}\frac{\partial H_E}{\partial P_E} + \frac{\partial f_A}{\partial H_L}\frac{\partial I}{\partial H_E}\frac{\partial H_E}{\partial P_E} = (\frac{\partial f_A}{\partial H_E} + \frac{\partial f_A}{\partial H_L}\frac{\partial I}{\partial H_E})\frac{\partial H_E}{\partial P_E}$$

Finally, the relationship between adult human capital and later childhood exposure is

$$\frac{\partial H_A}{\partial P_L} = \frac{\partial f_A}{\partial H_L}\frac{\partial H_L}{\partial P_L} + \frac{\partial f_A}{\partial P_L}$$

Note that the relationship between late childhood exposure and adult human is completely separate from the relationship between early childhood (or in utero) exposure and adult human capital. This is due to an assumption that exposure in the two periods is independent. If this is not the case (i.e. if there is some correlation between exposure in early childhood and exposure later in life), then the additional channel from early exposure to adult human capital, working through late childhood exposure, must be taken into account. Most other studies of the effect of early exposure on adult outcomes (e.g. Isen et al. (2016)) have lacked direct measurement of P_L, and have thus relied on research designs that compare individuals with otherwise identical later life experiences, with differences only occurring in the first years of life. On the other hand, if P_L can be directly observed (as is the case in this project), it is possible to separately identify each of the three effects above.

3 Data and Empirical Strategy

3.1 Linked Survey and Administrative Data

The investigation of the effects of pollution exposure at birth and in later childhood on human capital accumulation (specifically on college attendance) requires information on the location of birth, location in later childhood, and whether a college-aged student is attending college or university. To gather this information, I link responses to the American Community Survey with information on place and date of birth from the Social Security Administration and to address and income information from Form 1040 filings from the Internal Revenue Service. This linkage is performed using the Center for Administrative Records

Research and Applications (CARRA)'s Person Identification Validation System (PVS), which assigns unique Protected Identification Keys (PIKs) to records based on Social Security Numbers, date and place of birth, name, address and other personally identifiable information (PII).

The American Community Survey is a large scale, nationally representative survey which was designed to replace the functionality of the decennial Census long form on a more frequent time scale (annually instead of decennially). The ACS asks a number of questions about schooling, current enrollment, and incarceration and labor market outcomes.[4] I examine two distinct groups of respondents to the ACS: an older cohort born around the passage of the Clean Air Act of 1970, and a younger cohort born around the passage of the Clean Air Act Amendments of 1990. Due to data limitations, I lack important information about the experiences of the older cohort between birth and adulthood. I will thus use the linked data for the older cohort to replicate and extend the main results from Isen et al. (2016) and then use the richer data from the younger cohort to examine potential mechanisms for this effect.

I focus on two human capital outcomes for the younger cohort: college attendance and high school non-completion. The groups of interest in these two outcomes are slightly different. I focus on "college aged" individuals (aged 19–22 at the time of survey) who are potential college students (i.e. have graduated from High School and have not already received a college degree) when analyzing college attendance, and on individuals aged 16–24 when analyzing high school non-completion.[5] In addition to these human capital outcomes, I will also consider the effect of pollution exposure on several other outcomes that may be related to human capital acquisition, including labor force participation (among non-college attenders) and incarceration. The ACS provides rich demographic information about these individuals, which allows me to examine heterogeneity in the effect of exposure across race, ethnicity and family income. There are 5,636,272 respondents aged 16–24 at survey response in the 2005 through 2015 ACS, of whom 4,868,217 can be assigned a PIK (a PIK rate of 86.3 percent). Note that previous work using PIK-based data linkage has shown that PIK assignment may be non-random.[6] To alleviate this concern, I present re-weighted results (weighted by the inverse probability of receiving a PIK) to complement the baseline results, which are weighted only by ACS 1-year final sampling weights. This re-weighting does not substantively change any of the subsequent results (see Appendix B for more details).

[4]The ACS does not contain a direct question about incarceration, but does include information on whether an individual was in a correctional facility at survey response. Thus, my "incarceration" outcome is defined as an indicator for whether an individual was in correctional facility group quarters.

[5]This wider age range is used since these individuals are above the age of required school attendance (16 in many states), and are born around the enactment of the Clean Air Act Amendments of 1990, which provides the quasi-experimental variation used in the IV results.

[6]In particular, immigrants, young people and non-whites are less likely to receive PIKs.

To obtain information about the date and place of birth, I link these 4,868,217 individuals in the younger cohort (and 5,793,127 in the older cohort) to the SSA Numident by PIK. The Numident is the central database of all individuals who have been assigned a social security number (since PIKs correspond to SSNs on a 1-to-1 basis, all individuals with PIKs appear on the Numident). The Numident contains fields for exact date of birth,"place of birth", "state of birth", as well as a flag for whether an individual was born outside the United States. The place of birth field, filled out as part of an application for a social security number, can either be a city name or a county (or county equivalent). Although the state of birth field contains consistently defined two-character state abbreviations, the place of birth field is in practice a messy string variable, containing misspelled, inconsistently abbreviated and truncated place names. This makes assigning county of birth difficult.

To address this problem, I use a fuzzy string matching algorithm, using the optimal string alignment (OSA) distance, to match the place of birth variable to the official list of census-defined places and counties.[7] The algorithm works as follows. 1) I first attempt to find all exact string matches between the Numident place of birth field and the list of all census-designated places, blocked by state of birth. 2) I then attempt to find all exact matches between the remaining unmatched entries and the list of county names, blocked by state. 3) I then find all "fuzzy matches" with an OSA distance of 1 between the remaining unmatched entries and the list of places. 4) I then find all "fuzzy matches" with an OSA distance of 1 between the remaining unmatched entries and the list of counties. I then repeat steps 3 and 4, iteratively increasing the OSA distance. If an entry cannot be matched with an OSA distance of 5, I consider that entry to be "unmatchable" and drop the observation from the sample. This process allows me to identify the county of birth for 3,243,256 of the linked ACS-Numident responses for the younger cohort (74.2 percent of this linked sample for which the country of birth field in the Numident is the United States).[8]

The linked ACS-Numident dataset can be used to locate individuals in both the older and younger cohorts at the beginning of their lives (county of birth), and when they are respond to the survey, but provide little information about where they reside between these two points. To fill in this lack of information for the younger cohort, I supplement the linked ACS-Numident data with information on location and income from IRS form 1040 filings. Again, using PIKs as a unique identifier, I link each respondent to the universe of tax returns filed from 2000–2014. I assign location in High School as their most common filing location (at the county-tract-block level) for tax returns filed when they were aged 14–17. For individuals who have

[7] OSA distance is an extension of the Damerau-Levenshtein distance, where the distance between strings A and B is the number of operations (additions, deletions or transpositions) necessary to form B from A.

[8] I compare this approach to a previously developed internal crosswalk from Numident place of birth to GNIS places, and find that my approach coincides with the internal crosswalk in almost all cases in the internal crosswalk.

more than 2 filing locations, I assign their location based on the earliest location. Additionally, I assign their family resources in high school by taking the average Adjusted gross income across all tax returns they appear on from ages 14–17.[9].

Using the detailed location information I can then assign pollution exposure at birth for both cohorts, and during High School to each individual in the younger cohort. The older cohort was born between 1965–1976, and was aged ≈30-50 at survey response. The observations of college attendance (or non-attendance) for the younger cohort (16–24 year olds) occur over the period 2005 through 2015. These individuals were in high school from approximately 1997-2014, and were born between 1982-1998. The Numident provides coarse spatial information (county of birth) but fine temporal information (exact date), while the tax records have coarse temporal information (year of tax return) and fine spatial resolution (census block). I leverage two sources of pollution exposure data to do this, taking into account the available data and the relevant spatial and temporal resolutions. To measure exposure in utero and in infancy, I use county-level daily average air quality measured from the EPA's monitoring network. To measure exposure in high school, I use block-level annual average satellite-derived PM2.5 measurements from Voorheis (2016a). It is important to note that there are several limitations and potential sources of error in this linked data set: there is sampling and non-sampling error in the ACS; not every individual can be assigned a PIK; the matching algorithm cannot categorize every individual's county of birth; and pollution exposure at birth is the county average and not necessarily the individual's exposure.

3.2 Satellite Data

The satellite dataset provides annual average exposure to fine particulate matter smaller than 2.5 microns (PM2.5) for each census block in the contiguous United States over the period 1998-2014. This overlaps both with the time span covered by the universe of tax returns, and with the spatial coverage of the ACS (with the exception of Alaska and Hawaii). These data are described in detail in Voorheis (2016a) and van Donkelaar et al. (2016), however I provide a brief summary here.

Several satellites have been launched over the last two decades with a mission to measure aerosol optical depth (i.e. the degree of visual occlusion in satellite images) at fine spatial resolution. Aerosol optical depth (AOD) is related to concentrations of particulate matter, but is not in and of itself sufficient to be used as a measure of air quality (satellite imagery can be occluded by human-caused pollution — smog — as well as by forest fires, dust storms, and sea mist). Thus it is necessary to calibrate AOD retrievals with ground-level

[9]Since individuals may appear on multiple returns–e.g. both as a dependent and as a primary taxpayer if they filed as a dependent taxpayer–I sum across all returns in which an individual appears in a given year when calculating average AGI.

monitoring data to infer particulate matter concentrations from AOD. The dataset used here uses AOD retrievals from three satellites (MODIS, SeaWIFS and MISR) and calibrates these retrievals using ground monitor data from the existing set of worldwide PM2.5 monitors (these are largely concentrated in the US and Europe, although monitoring networks in, e.g. India and China have been expanding). Additional modeling steps attempt to correct for non-anthropogenic particulate species. This results in a gridded dataset at a 0.01 degree resolution, which Voorheis (2016a) interpolates to the census block level and zip+4 level.

I use these interpolated annual values to capture pollution exposure in high school in the following manner. First, to allow comparability with the estimated effects of early life exposure to TSP, I scale up the satellite derived PM2.5 values by a factor of 4.38, which is the average ratio of TSP to PM2.5 observations for all EPA monitor locations that monitored both PM2.5 and TSP between 1980 and 2000. Second, I assign these scaled up PM2.5 observations to individuals based on their reported locations in the universe of form 1040 tax returns when these individuals were aged 14-17. As in Voorheis (2016a), if block level information is available for a form 1040 observation, I use the block level interpolation, otherwise, I use the zip+4 code reported on the form. Finally, I average across all years in which an individual can be located in the universe of tax returns, and use this average as a measure of pollution exposure in adolescence.[10]

3.3 EPA Monitor Data

I capture pollution early in life (in utero and in early childhood) for both cohorts using measurements of particulate matter. Additional analysis using ground-level ozone as a measure of early life pollution exposure is presented in Appendix A. Particulate matter concentration data is not consistently available in AQI form, since unlike criteria gases (e.g. ozone), particulate matter is heterogeneous in composition. Regulation of particulate matter pollution has shifted its focus to smaller and smaller particles over the period since the Clean Air Act of 1970. Through 1990, the EPA mostly regulated and monitored Total Suspended Particles (TSP), defined as the amount of particulate matter less than 100 μm in size, measured in $\mu g/m^3$. From 1990-1998, the EPA shifted its focus to PM10: the amount of particulate matter smaller than 10 μm in size. After 1998, the EPA again shifted focus to smaller particles, this time to PM2.5: particulate matter smaller than 2.5 μm in size. The older cohort was born at a time in which TSP was the only monitor particulate pollutant. However, this shifting focus complicates measuring individual exposure for the younger cohort, since college students in the mid-2000s were born in the era when TSP monitoring was the norm, but college

[10] A small number of individuals appear on multiple form 1040s in a given year. If an individual appears as both a dependent and a primary taxpayer, I use the form in which an individual appears as a dependent. If an individual appears as a dependent across multiple 1040s, I take the average PM2.5 value for each of these locations and assign it to the individual for that year.

students in the 2010s were born when PM10 and PM2.5 monitoring were the norm.

I thus construct a daily particulate matter time series for each county with either TSP or PM10 monitors. To ensure comparability across observations of different sized particles, I calculate an average TSP-PM10 ratio using data from all monitoring sites that monitored both pollutants simultaneously, and then impute TSP from PM10 observations by scaling up PM10 by the TSP-PM10 ratio. This imputation method assumes a constant TSP-PM10 ratio over time. Note that because of the construction of the time series, the particulate matter measurements will be expressed as concentrations (with units $\mu g/m^3$), while the ozone measurements in Appendix Aare expressed in terms of AQI.

There are two main physiological pathways through which we might expect exposure to any of these pollutants to eventually affect later life outcomes. Pollution exposure in utero might affect health in early childhood, which in turn might affect parental investment, effort in school and hence subsequent human capital accumulation in childhood (in the conceptual model above, this corresponds to the term $\frac{\partial f_A}{\partial H_L}\frac{\partial I}{\partial H_E}\frac{\partial H_E}{\partial P_U}$. However, it is also possible that there could be a direct channel from early childhood human capital to later human capital working through direct effects of pollution exposure on cognition. There is evidence in the environmental health and epidemiology literature for health effects for both pollutants of interest. There is also some evidence for cognitive effects for both ozone exposure (Power et al., Ranft et al. (2009)), and particulates (Suglia et al. (2008), Mohai et al. (2011)). For both pollutants, the physiological pathway is hypothesized to work through a combination of inflammation (impacting on cardiopulmonary health) and oxidative stress (impacting neurological function). Additionally, there is some evidence that pollution exposure may be linked to other neuro-behavioral outcomes, such as ADHD (Siddique et al. (2011)) and depression (Fonken et al.).

3.4 Empirical Strategy

The main empirical strategy is to exploit arguably exogenous temporal variation in pollution exposure (in utero and very early childhood) within a child's county of birth. Additionally, for the younger cohort, I am able to control for exposure later in life (specifically during the adolescent years lining up with High school attendance). I describe below the specifications for the younger cohort, for which this intermediate information exists. The regressions estimated using the older cohort's responses are identical, with the exception that they necessarily omit any information during high school. For compactness of notation, assume there are three periods of interest.

The baseline fixed effects ordinary least squares (OLS) specification for the older cohort younger includes

county-of-birth, year-of-birth and survey year fixed effects:

$$Outcome_{i,c,t} = \delta_c + \delta_t + \beta_1 Pollution_{c_t} + X_i\Gamma + X_c\Theta + e_{i,c,t}$$

where c indexes county of birth and t indexes year of birth, X_i is a matrix of individual-specific potential confounders, including age (entering the regression as a categorical variable), month of birth, race, sex, ethnicity, marital status and labor force participation, and X_c is a matrix of county-of-birth level confounders, including population, personal income per capita, annual average temperature, and personal income growth rate. Standard errors are clustered at the state of birth level, allowing for arbitrary correlation across counties within a state, and within counties over time. The baseline OLS specification for the younger cohort includes information about location and environment during adolescence:

$$Outcome_{i,c,t} = \delta_c + \delta_t + \delta_d + \beta_1 Pollution_{c,t} + X_i\Gamma + X_b\Theta + e_{i,c,t}$$

where d indexes county of residence in adolescence, and X_i includes PM2.5 exposure and family income during adolescence. *Outcome* is one of college attendance, high school non-completion or incarceration for the younger cohort, and wages in adulthood for the older cohort.

The model for the younger cohort extends the fixed effects models in Bharadwaj et al. (2014) to include county of residence in adolescence fixed effects (to partially control for unobservables in high school environment) as well as directly observable pollution exposure during high school years. The identifying variation that in this specification comes from temporal variation in exposure within counties of birth, and the temporal and within-county spatial variation in exposure in the county of residence in high school. Assuming that selection into exposure at birth is not occurring due to unobservables, then β_1 captures the causal effect of early life exposure on adult human capital (i.e. $\frac{\partial H_A}{\partial P_E}$), while β_2 captures the causal effect of later childhood exposure (i.e. $\frac{\partial H_A}{\partial P_L}$).

It is possible, however, that endogenous air quality induced migration may violate this assumption. To address this, I exploit variation in air quality caused by changes to nonattainment designations by the Environmental Protection Agency. For the older cohort, I adopt the Isen et al. (2016) identification strategy, and use simulated nonattainment designations of the 1971 NAAQS TSP standards. EPA publication of the actual nonattainment designations under the 1971 TSP NAAQS standards do not appear to exist, so following Isen et al. (2016), I simulate nonattainment based on whether a county violated the standards in 1970. For the younger cohort, I capitalize on a second set of nonattainment designations enacted following the passage

of the Clean Air Act Amendments of 1990 (CAAA 1990).[11] Because of the better record keeping after 1990, the strategy for the younger cohort can utilize actual rather than simulated nonattainment designations. For the younger cohort, I will highlight changes in Nitrogen Dioxide (NO_2) nonattainment designation–NO_2 is a harmful pollutant in its own right, but also acts as important precursor to both ozone and particulate matter formation. Additionally, NO_2 is a direct co-pollutant for particulate matter, as the combustion of fossil fuels such as coal produces both types of emissions. Although the 1970 Clean Air Act included provisions setting standards for NO_2, nonattainment status was never designated until after the CAAA 1990, when several counties were designated as being in nonattainment of these earlier NO_2 standards.

Thus, I estimate 2SLS models using either NO_2 nonattainment designations as an instrument for TSP exposure in utero or in early childhood for the younger cohort or TSP nonattainment status as an instrument for TSP exposure for the older cohort. The first stage of these models for the younger cohort is:

$$Pollution_{c,t} = \delta_c + \delta_d + \delta_t + \beta_1 Nonattainment_{c,t} + X_i\Gamma + X_b\Theta + e_{i,c,t}$$

and the second stage uses the fitted values from the first stage regression in place of actual TSP levels:

$$Outcome_{i,c,t} = \delta_c + \delta_d + \delta_t + \beta_1 \widehat{Pollution}_{c,t} + X_i\Gamma + X_b\Theta + e_{i,c,t}$$

where *Outcome* is one of college attendance, high school non-completion or incarceration for the younger cohort, and wages in adulthood for the older cohort. Likewise, $Nonattainment_{c,t}$ is nonattainment of the TSP standards for the older cohort, nonattainment of the NO_2 standards for the younger cohort.

The reduced form effect of NO_2 (and TSP) nonattainment designations are themselves of interest, in addition to their utility as a source of useful variation. Hence I will supplement the above OLS and IV results with the reduced form effect of nonattainment designation at birth on college attendance and on high school non-completion. These reduced form regressions take the form:

$$Outcome_{i,c,t} = \delta_c + \delta_d + \delta_t + \beta_1 Nonattainment_{c,t} + X_i\Gamma + X_b\Theta + e_{i,c,t}$$

Note that the expected signs of the coefficient on nonattainment status in the reduced form model and the coefficient on, e.g. TSP exposure in the IV models will be reversed. The reduced form model can be

[11] Although in theory it is possible to use a similar IV strategy for adolescent exposure, I eschew this for two reasons. First, the nonattainment designations for the 1997 PM2.5 standards do not line up well with the sample, and second, sorting during adolescence is less of a concern. In particular, results are qualitatively similar for movers–individuals who moved between birth and adolescence–and non-movers.

interpreted as the effect of cleaner air induced by CAAA (hence we expect a positive coefficient), while the IV model can be interpreted as the effect of pollution exposure (hence we expect a negative coefficient). As before, *Outcome* is one of college attendance, high school non-completion or incarceration for the younger cohort, and wages in adulthood for the older cohort.

The IV models rely on variation induced by the nonattainment designations. Since the first stage of these models is essentially a difference-in-difference model of the effect of nonattainment on pollution levels, a visual check of the parallel pre-treatment trends assumption of the first stage bolsters the case for the validity of this IV approach. Figure 1 illustrates the trend in ozone and TSP exposure in utero for individuals in the younger cohort born in nonattainment and "control" counties. The pre-treatment trends for TSP exposure are parallel on visual inspection, and the sharp drop in exposure for non-attainment counties after designation is striking. Pre-treatment trends for ozone exposure, on the other hand, have a less clear pattern, which should be noted in the interpretation of the subsequent ozone results presented in Appendix A.[12]

Figure 2 shows a more formal test of the parallel trends assumption. Consider an event study regression of the effect of nonattainment designations on TSP exposure:

$$Pollution_{i,c,t} = \delta_c + \delta_d + \delta_t + \sum_{j=1987}^{1997} \beta_j \times EverNonattainment_c + X_i\Gamma + X_b\Theta + e_{i,c,t}$$

Where $\beta_{1990} = 0$ for infant exposure and $\beta_{1991} = 0$ for in utero exposure, and $EverNonattainment$ is equal to one if a county of birth was ever in nonattainment of NO_2 standards and zero otherwise. For the older cohort, an equivalent event study would be

$$Pollution_{i,c,t} = \delta_c + \delta_d + \delta_t + \sum_{j=1969}^{1976} \beta_j \times EverNonattainment_c + X_i\Gamma + X_b\Theta + e_{i,c,t}$$

with $\beta_{1971} = 0$, and $EverNonattainment$ defined based on whether a county was ever in nonattainment of the TSP air quality standards. This is an extension of the first stage regression shown above, but allows for separate effects in each year. A test for whether the pre-treatment coefficients are equal to zero then amounts to a test of the parallel trends assumption. Figure 2 graphs the estimated coefficients for these event study regressions for the younger cohort, estimated for TSP exposure, during infancy and in utero. Consistent with the pre-treatment trend graphs above, the pre-treatment coefficients are statistically equal to zero for TSP exposure. Figure 3 repeats this exercise for the older cohort with similar qualitative results.

[12]The mixed evidence of parallel trends for ozone may result in a weak instrument. In this just identified setting, this weak instrument should result in excess noise rather than bias, which is borne out by the relative size of the confidence intervals.

These tests bolster the case that the IV regressions reported below are properly interpreted as the causal effect of pollution exposure at birth on long term outcomes.

4 Long Term Wage Effects

I begin by replicating (and extending) a subset of results from Isen et al. (2016) using the linked ACS-Numident data for the older cohort of ACS respondents born between 1965–1976. The main result from Isen et al. (2016) is that the decline in TSP exposure at birth associated with the Clean Air Act increased wages at ages 29-31 by around $250 per year. This main result is from IV models estimated on a dataset of linked wages during the 1980s and 1990s and location and date of birth that covers 24 states. The linked ACS-Numident dataset provides two key advantages relative to the LEHD data used by Isen et al. (2016): the ACS provides a representative sample of the US population, and the timing of the ACS (2005 through 2015) provides a window into wage effects near the peak of adult earnings, which generally occurs around age 40.

Following Isen et al. (2016), I assign nonattainment status based on the county-level TSP concentrations in 1970: I consider a county in nonattainment if the annual average TSP levels are greater than 75 $\mu g/m^3$, or if the second highest daily TSP level is greater than 260 $\mu g/m^3$. These nonattainment designations went into effect in the middle of 1971, so I consider all ACS respondents born in 1972 or later to be "treated" in infancy, and all ACS respondents born in 1973 or later to be "treated" in utero. I report three sets of results: OLS results regressing adult wages on TSP exposure at birth, reduced form results regressing adult wages on an indicator for nonattainment status being in effect, and IV results where nonattainment status is used as an instrument for TSP levels. I use the same sample restrictions used in Isen et al. (2016): I consider only ACS respondents who are employed, and winsorize wages at $250,000 to avoid the undue influence of outliers.

Table 1 reports results of these three models, describing the effects of TSP exposure, and the CAA 1970, on adult wages. These results are presented as a stacked table, with OLS results in the top panel, IV results in the middle panel, and the reduced form results in the bottom panel. The first column reports results for the effect of TSP exposure in utero, while the second column reports results for TSP exposure during the first year of life. The OLS results in the top panel show a consistent negative effect of TSP exposure on wages, although only the in utero effect is statistically significant–this is smaller in magnitude but opposite in sign (and more precisely estimated) than the relevant OLS results in Isen et al. (2016). This pattern

is borne out in the IV results, which are likewise uniformly negative, and at least marginally statistically significant.[13] These results imply that a 10 $\mu g/m^3$ decrease in TSP (a benchmark used by Isen et al. (2016)) would lead to an increase in wages later in life of $246 per year for the in utero effects, and $405 per year for the infant effects. These effects are larger than the equivalent effects from Isen et al. (2016), which range from $186–$351 per year. The reduced form effects tell a similar story, suggesting that the CAA 1970 nonattainment status designations increased wages by $225 per year for the in utero effects, and $233 per year for the infant effects.

5 College Attendance

These results, replicating the Isen et al. (2016) result that TSP exposure in early life reduces wages in adulthood, strengthen the case for long term economic benefits of pollution reduction. These results, however, are silent as to how this wage effect might occur. The data for the older cohort is missing two key components of the "missing middle": outcomes in adolescence and early adulthood, and intermediate pollution exposure experienced in later childhood and adolescence. These components, however, are readily available for the younger cohort of ACS respondents born around the CAAA 1990, and so I turn now to analysis of how pollution exposure in early life might affect potential determinants of wages in adulthood. I begin with college attendance, an outcome which can be thought of as primarily working through a cognitive skills channel.

I present the results of the econometric models discussed above in stacked tables for compactness. Tables 2 - 3 each collect OLS (Panel A), IV (Panel B), and reduced form (Panel C) models for TSP exposure in utero and during infancy. I will discuss each set of models together, beginning with the OLS models. The top panel of Table 2 presents the OLS effects of in utero TSP exposure and adolescent exposure on college attendance at age 19–22. All models in Table 2 include the same high school exposure variable, but differ in how in utero exposure is defined. Because the EPA monitor data is measured daily, I am able to calculate average exposure not just within the full term 9-month in utero period, but also for individual trimesters. Column 1 reports the effect of average exposure over the entire pregnancy, while Column 2 considers the effect of average TSP exposure in the first trimester, column 3 considers the average in the second trimester, and column 4 considers the average exposure in the third trimester.

Across all models, pollution exposure during adolescence has a negative and statistically insignificant

[13] For the remainder of the document, results that are statistically significant at the 10% but not 5% levels will be referred to as "marginally" significant.

effect, with a 1 $\mu g/m^3$ increase in PM2.5 exposure (equivalent to 4.38 $\mu g/m^3$ TSP) decreasing the probability an individual will attend college by about 0.25 percent. The OLS effects of in utero TSP exposure are inconsistent in sign and contrary to expectations, however. Exposure to TSP has a negative correlation with college attendance over the full in utero period, but positive correlation in the first trimester, although none of these effects are statistically significant. The top panel of Table 3 reports results from models examining the effect of TSP exposure in infancy and PM2.5 exposure in high school on college attendance at age 19–22. As in the previous table, all models include the same high school exposure variable. Each column includes different measures of "infant exposure", mirroring the time periods in the in utero exposure models. Again, because of the daily temporal resolution of the underlying EPA monitor data, it is possible to measure average exposure not just in the first year of life, but also for 3-month windows. Column 1 defines exposure as the average TSP exposure in the first year of life, while Column 2 defines infant exposure as the average exposure in the first 3 months of life, column 3 months 3-6, column 4 months 6-9. The effect of infant exposure varies across exposure windows, with a mix of small negative and positive coefficients, all imprecisely estimated.

Note however, that these OLS models may not fully account for endogenous exposure due to locational sorting. I thus turn to IV models, where I use plausibly exogenous variation – EPA designations of nonattainment status – to identify the effect of TSP exposure. I instrument for TSP exposure using designations of nonattainment of the 1971 NO_2 national ambient air quality standards which occurred after the passage of the 1990 Clean Air Act Amendments. Previously reported results provide evidence that the identifying assumptions of this IV strategy are likely satisfied, so that these nonattainment designations provide exogenous variation in TSP level that can be used to identify the causal effect of TSP exposure at birth even if there is locational sorting due to air quality.

The middle panel of Table 2 reports results from IV models capturing the effect of TSP exposure in utero on college attendance, with a structure that mirrors Columns 1-4 from the top panel of Table 2. In contrast to the OLS results, the effect of TSP on college attendance is universally negative across exposure definitions, and relatively precisely estimated—the effects of TSP exposure across trimesters are statistically significant at the 1 percent level. The full term in utero effect size is -0.0018, implying that a 10 $\mu g/m^3$ increase in TSP in the county of birth decreases the probability of college attendance by 1.8 percentage points. The middle panel of Table 3 reports analogous results for the effect of TSP exposure during infancy, with slightly larger effect sizes, again relatively precisely estimated. A 10 $\mu g/m^3$ increase in TSP exposure during the first year of life reduces the probability of college attendance at ages 19-22 by 2.5 percentage points.

5.1 Heterogeneity by Race and Family Income

A large literature on environmental justice has gathered robust evidence that disadvantaged groups (poor families, racial and ethnic minority groups) bear a disproportionate burden of exposure to environmental hazards. While this literature often focuses on the siting of fixed point pollution sources (see Banzhaf (2012) for an overview), there is strong evidence for persistent disparities in exposure to ambient air pollution along race and income lines (Clark et al. (2014), Voorheis (2016b)). I draw on this literature to illustrate the consequences of this environmental inequality at birth, by examining how the effects of pollution exposure at birth demonstrated above vary between racial groups (Whites vs. Non-whites) and across the income distribution (by quintile of the AGI distribution).[14]

Given the previous full-sample results, the focus of this exercise will be on TSP exposure over the broadest time frames: in utero, and in the first year of life. To examine the environmental justice dimension of these results, I contrast results of the previously used OLS and IV models estimated on sub-samples corresponding to advantaged and disadvantaged groups. To preserve sample size, I use relatively coarse definitions: contrasting whites and non-whites to capture racial disparities (and then splitting the non-white group into the two largest subgroups, blacks and Hispanics), and the bottom two quintiles versus top three quintiles to captures differences between low and high income individuals.

Tables 4 and 5 summarize the results of OLS and IV models of the effect of TSP exposure in utero and in the first year of life on college attendance, stratified by disadvantage in two ways. The first two columns of each of these tables stratify by race, with the first column reporting results for the sub-sample of white individuals, and the second reporting results for non-white individuals. The middle two columns stratify the non-white sub-sample further, reporting results for the largest non-white groups, blacks and Hispanics. The final two columns stratify by family income, with the fifth column reporting results for the sub-sample of individuals with family incomes in the bottom two quintiles of the county AGI distribution, while the sixth column report results for the top three quintiles. The income data being used for this stratification is taken from IRS tax returns, and is measured when the individuals are in high school.[15]

Looking at heterogeneous effects of TSP exposure, there does not appear to be strong evidence for EJ-type effects when looking at a white vs. non-white stratification. The top and middle panels of Table 4 report IV and OLS results for TSP exposure in utero, while the top and middle panels of Table 5 report these results for TSP exposure during infancy. There is mixed evidence for a disparate impact of TSP exposure in

[14] White here refers to ACS respondents who report no Hispanic ancestry, and identify as White

[15] This may not necessarily accurately reflect the relative status of an individual's family at birth, but due to data limitations, no other information on family resources at birth is available.

utero. The in utero effects on whites and non-whites are both statistically significant and the effect sizes are similar in magnitude, and there are also no substantial differences in the effect sizes for poor and non-poor children. The effects of TSP exposure during infancy are exhibit a similar pattern.

Stratifying the non-white sub-sample further can provide additional nuance as to who the most highly affected subgroups may be, within an overall disadvantaged group. Sample sizes are prohibitively small for some non-white subgroups (Asian-Americans, American Indian/Alaska Natives), but there is sufficient power to identify separate effects for Hispanics and Blacks. The third and fourth columns of Tables 4 and 5 report results of the effect of TSP exposure (in utero and during infancy) on black and Hispanic individuals. These results suggest a disproportionate effect for Hispanic individuals, at least relative to blacks: the effects for Hispanics are substantially larger than for blacks, and statistically significant at conventional levels (the effect for blacks is negative and statistically insignificant, however). Note however that at least in this case, the effects for Hispanics and whites are not substantially different. At least for college attendance, a human capital outcome associated primarily with a cognitive skills channel, there appears to be little evidence of disproportionate effects by race or class.

5.2 Reduced Form Effects of of Nonattainment Designation

As with any instrumental variables strategy, there is some degree of uncertainty as to whether the exclusion restriction is in fact perfectly satisfied. As an additional modeling exercise, I thus also consider the direct, reduced form, effect of nonattainment designations on college attendance. This reduced form effect is also of interest on its own, since the human capital impacts of policies related to the CAAA 1990 (such as the NO_2 nonattainment designations) are an important potential benefit which should be included in retrospective benefit-cost analyses. I consider models with nonattainment indicators for NO_2 for the full sample, as in the OLS and IV models in Table 2, and stratified by race and family income, as in the top two panels of Table 4.

The bottom panels of Tables 2 through 4 report reduced form effects of nonattainment status designations in utero on college attendance. For the full sample, the nonattainment designations increased college attendance by 2.4 percentage points. Similarly, the bottom panels of Tables 3 and 5 reports results of the reduced form effect of nonattainment designation during infancy on college attendance. Nonattainment designation during infancy similarly increased the college attendance rate by 2.4 percentage points for the full sample. There is some heterogeneity by race and class, although not necessarily in a way that supports an environmental justice story. The effects are larger for whites (2.4 percent) and smaller for non-whites (1.8

percentage points). The effect is also smaller for poorer children than for children in the top 3 quintiles.

5.3 Implied Wage Effects

The above results suggest substantial impacts of exposure to ambient air pollution on human capital acquisition. The human capital outcome of interest here–college attendance–is the subject of a large body of literature in labor economics. Thus, it is possible to draw on this expansive literature on the college wage premium to do some rough calculation of the the wage effects implied by the results above. Given that the strongest results rely on policy variation as a result of the Clean Air Act Amendments of 1990, a logical thought experiment is to simulate the wage effects of a reduction in TSP that occurred around the time theses policies went into effect. It is then possible to compare these back of the envelope calculations to the direct wage effects of pollution exposure estimated above for the older cohort.

The "pollution wage premium" can be thought of as being the change in the probability of completing college induced by a change in pollution exposure multiplied by the college wage premium. The estimated effects above provide a number for the effect of pollution exposure on attending college, but not of completing college. I thus scale our effects by the NCES' estimate of graduation rates (60 percent) to obtain the effect of pollution on college completion. I use \$17,500 as the college wage premium (this number is calculated from 2013 March CPS, and is the average wage and salary difference between college educated and high school educated workers).[16] Thus a rough estimate of the wage premium associated with a change in pollution exposure is:

$$\Delta W = \Delta Pollution \times \beta_1 \times 0.6 \times 17500$$

Where β_1 is the estimated coefficient from the OLS or IV models above.

The first stage event study results in Figure 2 suggest that the CAAA-related NO_2 nonattainment designations reduced TSP exposure by 15-20 $\mu g/m^3$ in treated counties. Compared to a no CAAA baseline, what effect on wages would the upper end of the improvement in air quality have? Taking the OLS estimate in Table 2, column 1 as given, this counterfactual improvement in air quality would have increased college attendance by $-20 \times -0.0002 = 0.48$ percent. Thus our estimate of the wage effects using the OLS estimate is

$$\Delta W_{inutero} = -20 \times -0.00001 \times 0.6 \times 17500 = \$2.10$$

On the other hand, we (unsurprisingly) see substantially higher wage premiums for the much larger IV

[16] for more details see: `http://www.pewsocialtrends.org/2014/02/11/the-rising-cost-of-not-going-to-college/`

estimates in Table 2, column 4. The IV estimates imply a wage premium of

$$\Delta W^{IV}_{inutero} = -20 \times -0.00184 \times 0.6 \times 17500 = \$386.40$$

Although the lower range (i.e. the OLS results) of these wage effects seem quite small for an individual, keep in mind two factors. First, these are annual effects. Compounded over a whole working life, these impacts are still somewhat modest – at a 3 percent discount rate, the discounted present value of the stream of infant pollution wage premia is $50 over a 40 year career. Second, these impacts are for a single individual in a single cohort. The aggregate effect of these small changes is much larger. Via CDC vital statistics, around 4 million births occurred in 1996 in the United States. If we sum the expected wage premium over all these individuals, we can see that the in utero OLS effects amount to $8,400,000 in additional aggregate wages. If we sum the flow of these aggregate wage premia, we find that the discounted present value (at a 3 percent discount rate) over an average 40 year career is $193 million for the in utero wage effects. The IV effects, however, imply an substantially larger effect: approximately $1.5 billion per year per cohort in additional aggregate wages (using the infant effect), or a discounted present value of $35 billion over a 40-year career.

Doing these same calculations, assuming instead a 10 $\mu g/m^3$ drop in TSP exposure as in Isen et al. (2016) results in an implied wage premium of about $190/year. To contextualize this number, consider that Isen et al. (2016)'s results for the 1970 CAA suggest that nonattainment designation (or equivalently in their models, a $10\mu g/m^3$ decrease in county average TSP) increased adult earnings by $260 per year.[17] Thus the implied long term wage effects working through college attendance can explain at most 70 percent of the previous estimates of the long term wage effect. This suggests that a cognitive-skills driven channel working through human capital acquisition in the form of college attendance is not the only channel through which pollution exposure at birth affects economic well being in adulthood. To investigate an alternate channel which may also be important, I now turn to the analysis of outcomes which are arguably more related to non-cognitive skills, including high school non-completion and incarceration.

[17] In section 4 above, I replicate these results using the linked ACS-Numident data, and find a similar effect size: a 10 $\mu g/m^3$ decrease in TSP in utero would increase adult wages by about $246 per year.

6 High School Non-completion

It is clear that there is compelling evidence for particulate matter exposure at birth having substantial negative impact on one measure of human capital accumulation—college attendance—common at the top of the income distribution, and which is associated with primarily cognitive skills. In other words, pollution exposure decreases one potential measure of advantage. It is also possible that pollution exposure might increase disadvantage, and thus I examine how pollution exposure affects high-school non-completion, an outcome common at the bottom of the income distribution. The mix of cognitive and non-cognitive skills necessary to succeed in college is very different from the mix of skills necessary to accumulate other types of human capital (such as a high school degree), and so pollution exposure may have disparate effects on the two types of human capital accumulation.

I begin, as in the college attendance case, with simple OLS fixed effects models of pollution exposure in utero on high school non-completion. Since individuals can legally drop out of high school as early as 16, I expand the sample of interest to individuals aged 16–24 at the time of survey response. Results are largely robust when using the same age range (19–22) as in the college attendance analysis. The top panel of Table 6 reports the results of OLS models describing the effect of TSP exposure in utero on high school non-completion later in life. Consistent with the college attendance results, TSP exposure in utero has statistically insignificant, small and positive effects on high school non-completion. Note however, that unlike the college attendance case, there is strong evidence for later life exposure affecting high school non-completion. PM2.5 exposure during ages 14–17 has a positive and statistically significant effect — a 1 $\mu g/m^3$ increase in PM2.5 exposure (equivalent to 4.38 $\mu g/m^3$ of TSP) in late adolescence increases the probability of high school non-completion by 0.05 percentage points.

Effects of exposure during infancy estimated using OLS follow largely similar patterns. The top panel of Table 7 shows the effect of TSP exposure in infancy on high school non-completion, which again implies effects indistinguishable from zero. Here, exposure in each time-frame after birth is positive, although only the effect of TSP exposure at 3-6 months after birth is statistically significant at the 5 percent level. Exposure to particulate matter pollution in adolescence continues to be a marginally significant predictor of high school non-completion, with a 1 $\mu g/m^3$ increase in PM2.5 exposure during adolescence increasing high school dropout rates by 0.05 percent.

As in the college attendance analysis, concerns about potentially endogenous exposure due to non-random sorting suggest the use of quasi-experimental variation in exposure to identify long term effects. I thus turn to IV models which again use designations of nonattainment of NO_2 standards as an instrument for TSP

exposure. The tables again mirror previously used tables used in the college attendance analysis, using a single time frame of exposure as a measure of exposure in each regression.

The middle panel of Table 6 reports results from IV models of the effect of TSP exposure in utero on high school non-completion. In stark contrast to the OLS results, and to the TSP results for college attendance, TSP exposure in utero has large and statistically significant effects on high school non-completion, across all exposure windows. A 10 $\mu g/m^3$ increase in TSP exposure in utero increases the probability of not completing high school by 0.6 percentage points (compared to a mean high school non-completion rate of 6.2 percent in the sample). Exposure to fine particulates in later adolescence continues to have a substantial effect on high school non-completion, with a 1 $\mu g/m^3$ increase in PM2.5 exposure corresponding to a 0.06 percent increase in the high school non-completion rate. The middle panel of Table 7 reports similar IV results for TSP exposure during infancy, and again we find an effect of TSP exposure on high school non-completion which is both economically and statistically significant, of slightly larger magnitude to the in utero results. A 10 $\mu g/m^3$ increase in TSP exposure in the first year of life increases the high school dropout rate by 0.9 percent.

Next, I turn to the consideration of whether exposure to TSP at birth (and PM2.5 in late adolescence) has differential effects on high school non-completion for advantaged versus disadvantaged groups. As in the college attendance analysis, I stratify the sample by race (white vs. non-white, and then splitting the non-white group into Hispanic origin and black) and income group (bottom 2 quintiles vs. top 3 quintiles), and estimate OLS and IV regressions separately for each sub-sample, as in Table 9. Although I estimate OLS, IV and reduced models, I will focus only on the IV results here; OLS and reduced form results are reported in the top and bottom panels of each table.

The middle panel of Table 8 reports results of TSP exposure in utero on high school non-completion, stratified by race and income groups. The effect of in utero TSP exposure is usually positive or statistically indistinguishable from zero across sub-samples (as is the effect of later adolescent exposure), although there is substantial heterogeneity that is consistent with disproportionate effects for disadvantaged groups. This provides the first evidence that the long term human capital effects of pollution exposure are an environmental justice concern, in contrast to the college attendance results. The effect for non-whites is statistically significant and nearly an order of magnitude larger than the effect for whites (a 10-$\mu g/m^3$ increase in TSP exposure corresponds to a 1 percent increase in high school non-completion for non-whites, but only a 0.3 percent increase for whites.) Likewise, the effects for poorer children are statistically significant at the 5 percent level with an effect size slightly larger than for children in the top 3 quintiles. Similar patterns of

heterogeneity across race and class obtain for TSP exposure during infancy (in Table 9): the effects are larger and more precisely estimated for non-whites than for whites, with only small differences across income groups. A 10-$\mu g/m^3$ increase in TSP exposure during the first year of life is associated with a 1 percent increase in high school non-completion for non-whites, but only a 0.3 percent increase for whites.

The third and fourth columns of Tables 8 and 9 collect these results for TSP exposure (in utero and during infancy) stratifying the non-white group further into blacks and Hispanics. It is clear that the source of this non-white vs. white disparity across these results is primarily driven by Hispanic children (as was the case for college attendance): a 10-$\mu g/m^3$ increase in TSP exposure during infancy corresponds to a 1 percent increase in high school non-completion for Hispanics, but a 0.02 percent increase for blacks (albeit statistically insignificant). Note that the effect for blacks is not substantially different from the effect for whites. Similar disparities are clear for TSP exposure in utero, and for the ozone exposure results collected in Appendix A.

7 Incarceration

I consider one final outcome which is an important determinant of wages: incarceration. The rise in the US prison population has become a central focus in the study of the phenomenon of "men without work" (Eberstadt (2016)), and thus incarceration and other exposure to the criminal justice system is a plausible mechanism that may affect future wages. This is likely especially true for individuals in disadvantaged communities, who are more likely to bear the brunt of the costs of incarceration. A small but growing literature has provided some evidence that pollution exposure, either contemporaneous (e.g. Herrnstadt and Muehlegger (2015)) or in early life (e.g. Reyes (2014)) might affect crime rates. This effect is thought to work through a neurological-behavioral channel: exposure to pollutants (specifically airborne lead particles and other particulate matter) can affect the functioning of the parts of the nervous system associated with impulse control. Thus incarceration, like high school non-completion, can be thought of as a proxy for non-cognitive skills, in the same way that college attendance can be thought of as a proxy for cognitive skills. This increased impulsiveness in turn is thought to, at least on the margin, increase the probability that an individual will commit a criminal act. Although most of this literature has focused on the special case of lead exposure, there is ample evidence that particulates may also affect crime.

I do not observe individual criminal acts, however, I do observe whether an individual resided in correctional facilities group quarters (and which type of group quarters in which they reside) at ACS survey

response. I can thus categorize an individual as incarcerated if they reside in a federal detention facility, a federal or state prison, a local jail, a residential corrections facility, or a juvenile corrections facility. Note that this means that I cannot distinguish between the effects of pollution exposure on different types of crime–the previous literature suggests that pollution exposure may disproportionately affect violent crimes–only on one end result of crime, incarceration.[18] As in the previous analysis, the population of interest is individuals aged 16–24 at the time of survey response, who were born around the time enactment of the CAAA 1990, the source of quasi-experimental variation used in the IV models.

Tables 10 and 11 report results of models examining the effect of TSP exposure in utero and during infancy on incarceration at ages 16–24. These tables report the overall results, as well as results stratified by race (white vs. non-white) and class (bottom 2 quintiles vs. top 3 quintiles). Consistent with the previous set of results, suggesting generally economically and statistically significant effects of early life exposure on several human capital outcomes, across pollutants and subgroups I find generally strong evidence of a statistically significant effect of pollution exposure in early life on incarceration. Exposure to TSP in utero increases the probability of incarceration for children of poor parents, and have a marginally significant and small positive effect on incarceration for whites. Curiously, the effect of exposure at birth across time frames on incarceration is statistically insignificant for non-whites. However, there is consistent evidence for a positive effect of PM2.5 exposure during adolescence on incarceration, across all subgroups. There is substantial heterogeneity in this adolescent exposure effect consistent with environmental justice concerns - a $1\mu g/m^3$ increase in PM2.5 exposure (equivalent to 4.38 $\mu g/m^3$ of TSP) during adolescence is associated with a 0.09 percent increase in incarceration for non-whites, but only a 0.04 percent increase for whites.

As in previous human capital analyses, it may be instructive to further disaggregate the non-white subgroup to disentangle the somewhat unexpected null result. The middle two columns of Tables 10 and 11 report results of the effect of TSP exposure in utero and during infancy for blacks and Hispanics separately. These results suggest substantial heterogeneity within the non-white subgroup: TSP exposure in utero and during infancy has a statistically significant and positive effect on the incarceration of blacks, but has a statistically insignificant effect on Hispanics (and in fact, the effect is negative for TSP exposure during infancy, resulting in an overall small and statistically insignificant effect for non-whites). A 10-$\mu g/m^3$ increase in TSP exposure in utero increases the incarceration rate of blacks by 0.3 percent, but has no statistically significant effect on the Hispanic incarceration rate (the effect for whites is positive and statistically insignificant at 0.01 percent). This disparity is also apparent in the later life exposure: a 1

[18]Not all inmates in the ACS sample are necessarily convicted of a crime, since inmates can be held in jails, prisons and detention facilities after arrest but before conviction.

$\mu g/m^3$ increase in PM2.5 exposure during adolescence increases the black incarceration rate by 0.1 percent, compared to 0.01 percent for whites.

The results from this stratification present something of a puzzle: why would there be statistically significant effects (with heterogeneity) for later life exposure across all subgroups, but consistent statistically significant effects for only one subgroup (blacks) when examining the long term effects of early life exposure? One possibility is simply that the early life effects are measured with more error than are the later life effects, and hence are attenuated. A second possibility, not necessarily mutually exclusive with the first, however, is that the long term effects on incarceration may work through a series of social and institutional channels which end up disproportionately affecting specific disadvantaged groups. In light of the increased salience of incidents of police violence against black men, and the discussion of racial disparities in the criminal justice system broadly, these results seem consistent with a mechanism working through compounding disadvantage, where initial exposure to pollution's effect on non-cognitive skills compounds over time in the context of racially biased institutions. Speculatively, one specific channel could work as follows through a combination of pollution exposure increasing the propensity to commit "quality of life" crimes for all exposed individuals, and "broken windows" police response leading to a disproportionately high arrest rate for black offenders, resulting in an effect of early life pollution exposure on incarceration for blacks, but only small effects for other groups.

8 Conclusion

These results deepen our understanding of the economic effects of pollution exposure by improving our understanding of the "missing middle"–previous literature has identified effects of pollution exposure at birth on wages at prime working ages (around 30), and the effects of contemporaneous pollution exposure and exposure at birth on early life health and human capital outcomes, but the intermediate period has remained a mystery. I examine several human capital outcomes in this missing middle, which allow me to identify potential channels from early life exposure to later life outcomes. I find that pollution exposure at birth affects not just human capital outcomes that are associated with cognitive skills (college attendance), but also outcomes that may be driven by non-cognitive skills — pollution exposure at birth increases high school dropout rates and incarceration, and increases labor force participation but lowers wages among employed non-college students. These effects operate separately from short term effects of pollution exposure later in life, which increases primarily non-cognitive skill related outcomes (incarceration, high school non-

completion) but has no detectable effect on college attendance, a cognitive-skill related outcome.

Additionally, I am able to leverage detailed demographic information to examine heterogeneous effects across subgroups. Doing so suggests a stark fact: not only are disadvantaged groups more exposed to pollution exposure, as has been established by the environmental justice literature (e.g. Banzhaf (2012)), but they are also more highly affected by this pollution exposure at birth and during adolescence. These two mutually reinforcing trends suggest an important environmental pathway for the intergenerational transmission of inequality, and may in turn inform the growing literature on the determinants of income mobility. Additionally, neighborhood variation in the exposure of disadvantaged children to pollution can inform analyses of the long term effects of neighborhood quality on income. In "moving to opportunity", disadvantaged families are not just ensuring access to potentially better schools and higher quality peers, but also better air quality (e.g. by moving from polluted central city neighborhoods to the less polluted suburbs).

These effects underline how important correctly characterizing the long term effects of pollution exposure are for accurately accounting for the benefits and costs of air quality regulations. Further, they underline the degree to which these benefits are distributed across disadvantaged groups. A more optimistic counterpart to these long term effects driving intergenerational transmission of inequality is that these effects imply that disadvantaged groups disproportionately benefit from air quality improvements. Conversely, the burden of any future worsening of air quality will likely accrue to these disadvantaged groups. These results suggest that two results of future increases in ambient air pollution could be an increase in black men in prison, and a decrease in the high school graduation rate, especially among Hispanics.

There are still several areas where data limitations bind, and future studies which can loosen these constraints will continue to move this literature forward. The current study uses administrative records to locate individuals at birth and in adolescence, but still leaves exposure in the period after birth and before secondary school unmeasured. The outcome measures used are all survey based, which allows for the use of multiple outcomes, however there may be administrative records measures of some outcomes (i.e. college attendance) which can be leveraged. Additionally, in light of the heterogeneity in the estimated effects of pollution across disadvantaged groups, an important extension of this work will be to further examine the effect of pollution exposure on outcomes that are endemic to these disadvantaged communities, such as the "school-to-prison pipeline", recidivism and risky behaviors such as crime and drug use. All of these extensions will be more feasible than would have been the case otherwise, due to the linkage infrastructure provided by the Census Bureau.

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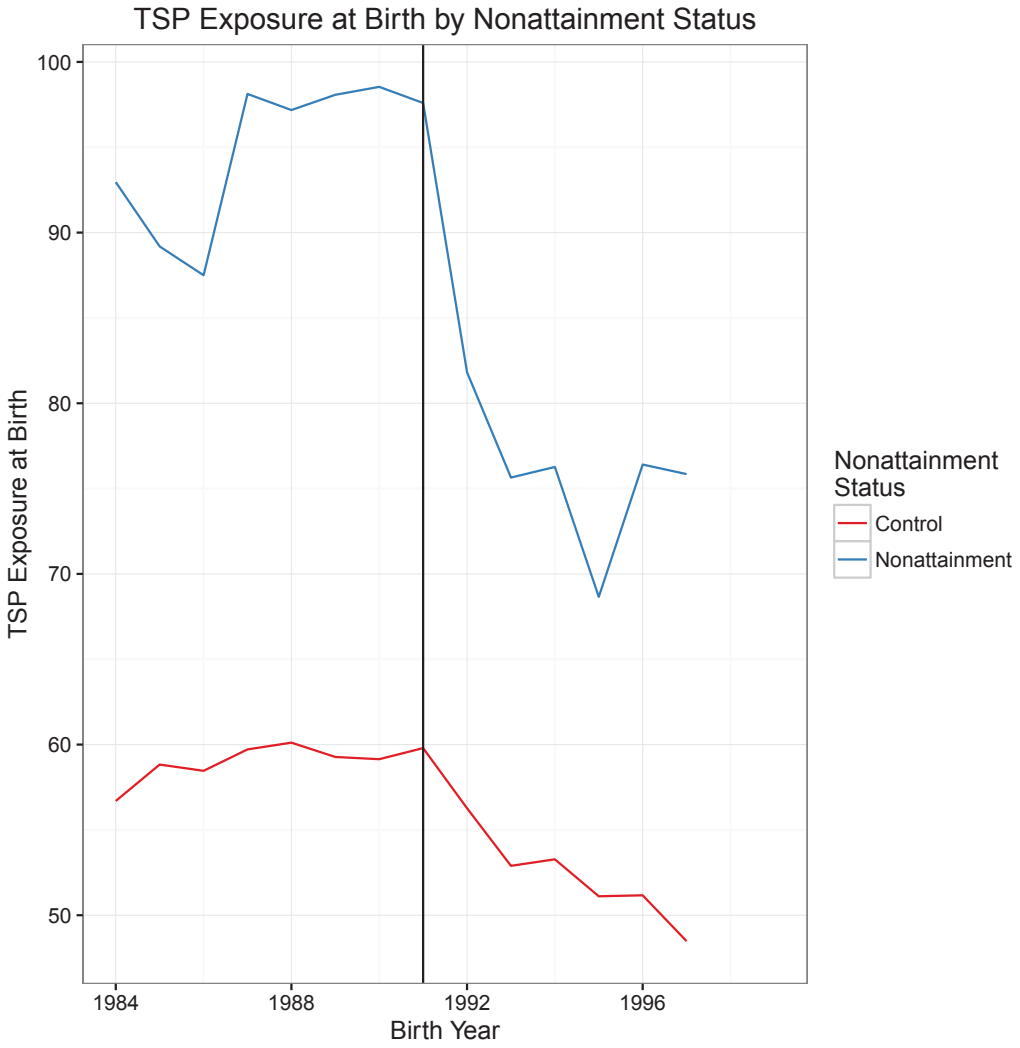
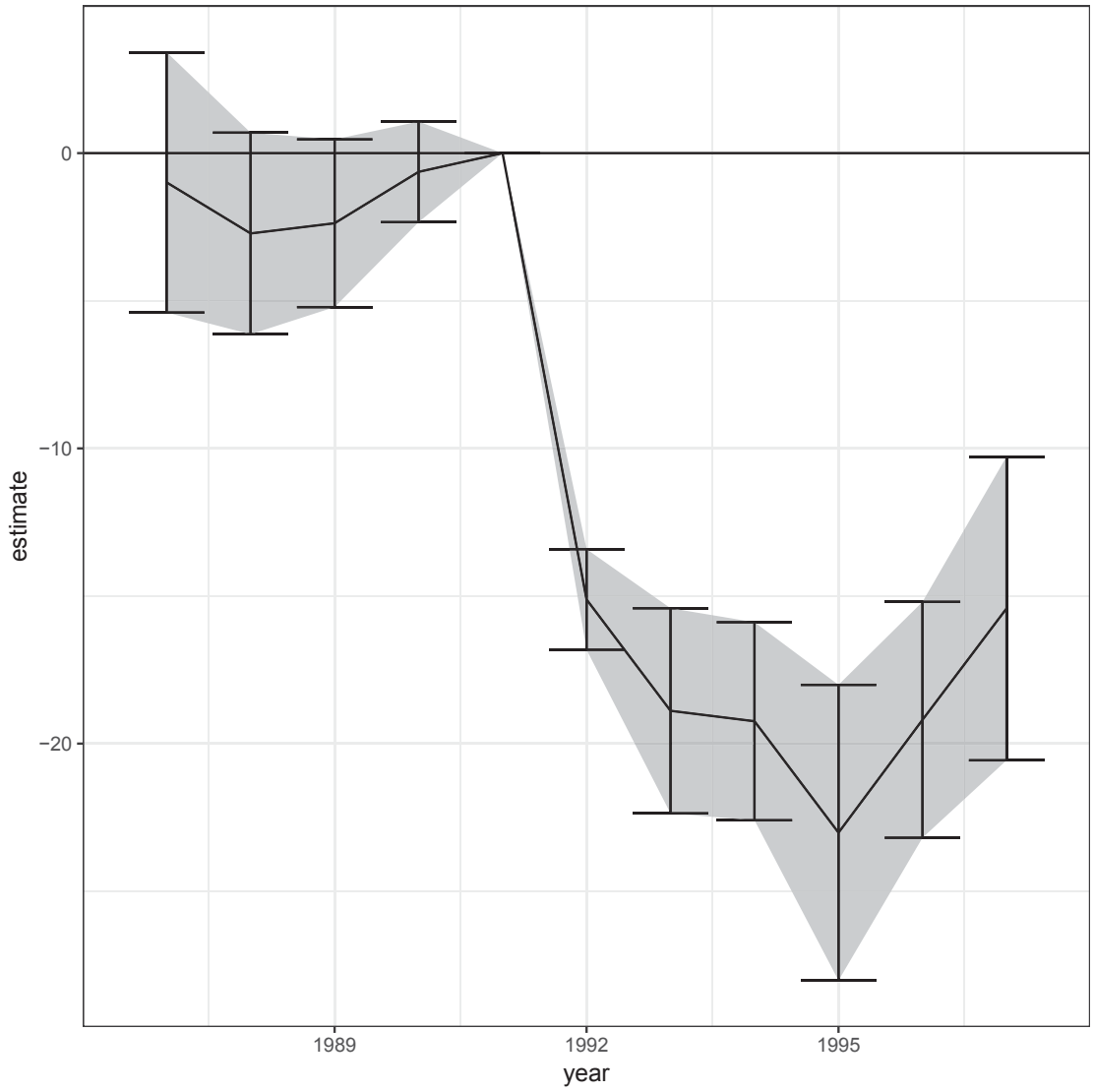
9 Figures

Figure 1: Comparing Pre-treatment Trends, TSP Exposure



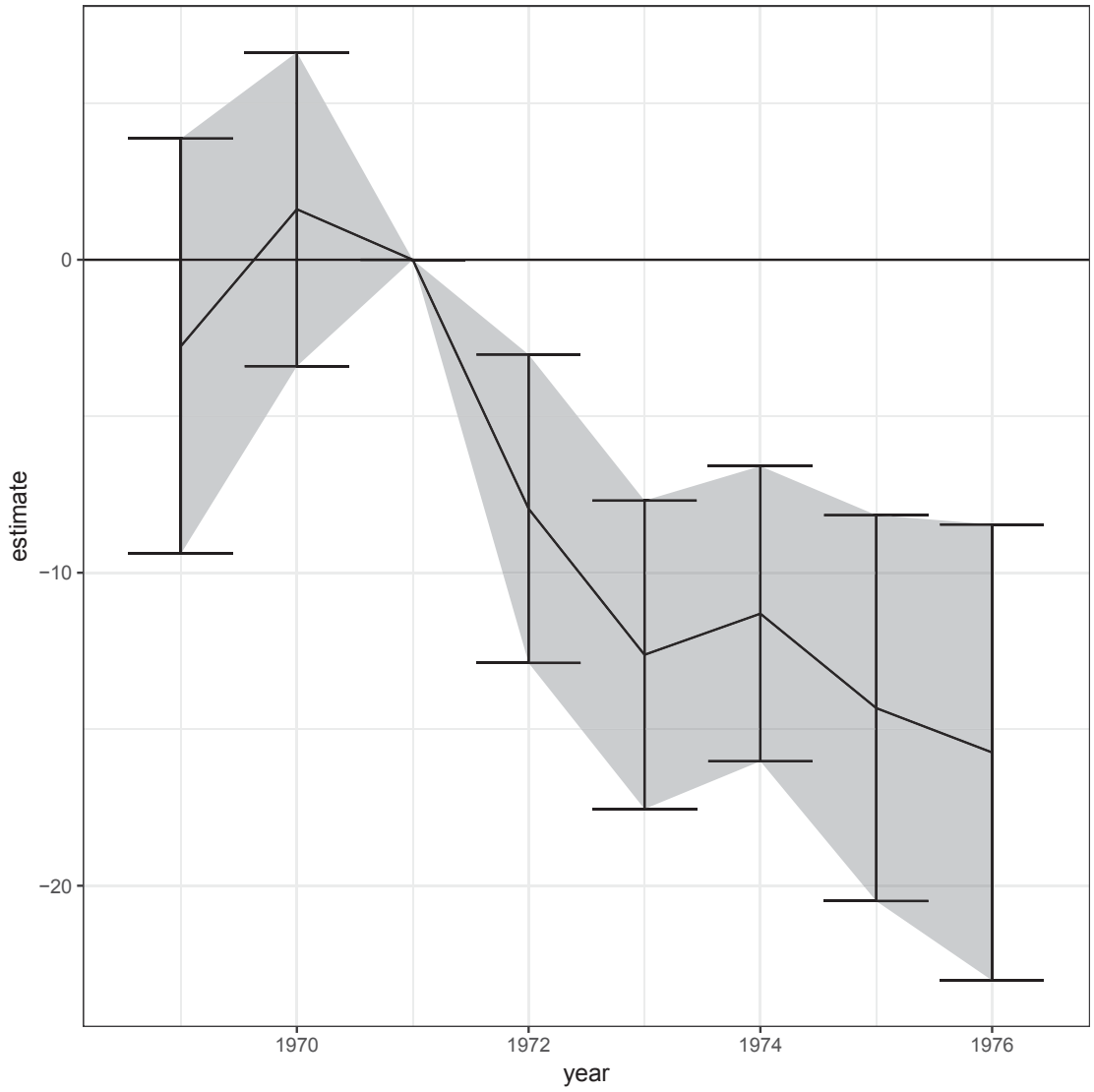
Source: 2005 through 2015 ACS and EPA monitor data
Note: This figure shows the average Ozone and TSP exposure in utero for individuals born before and after the 1991 NO_2 Nonattainment designations.

Figure 2: Test of Parallel Trends Assumption, TSP Exposure, Younger Cohort



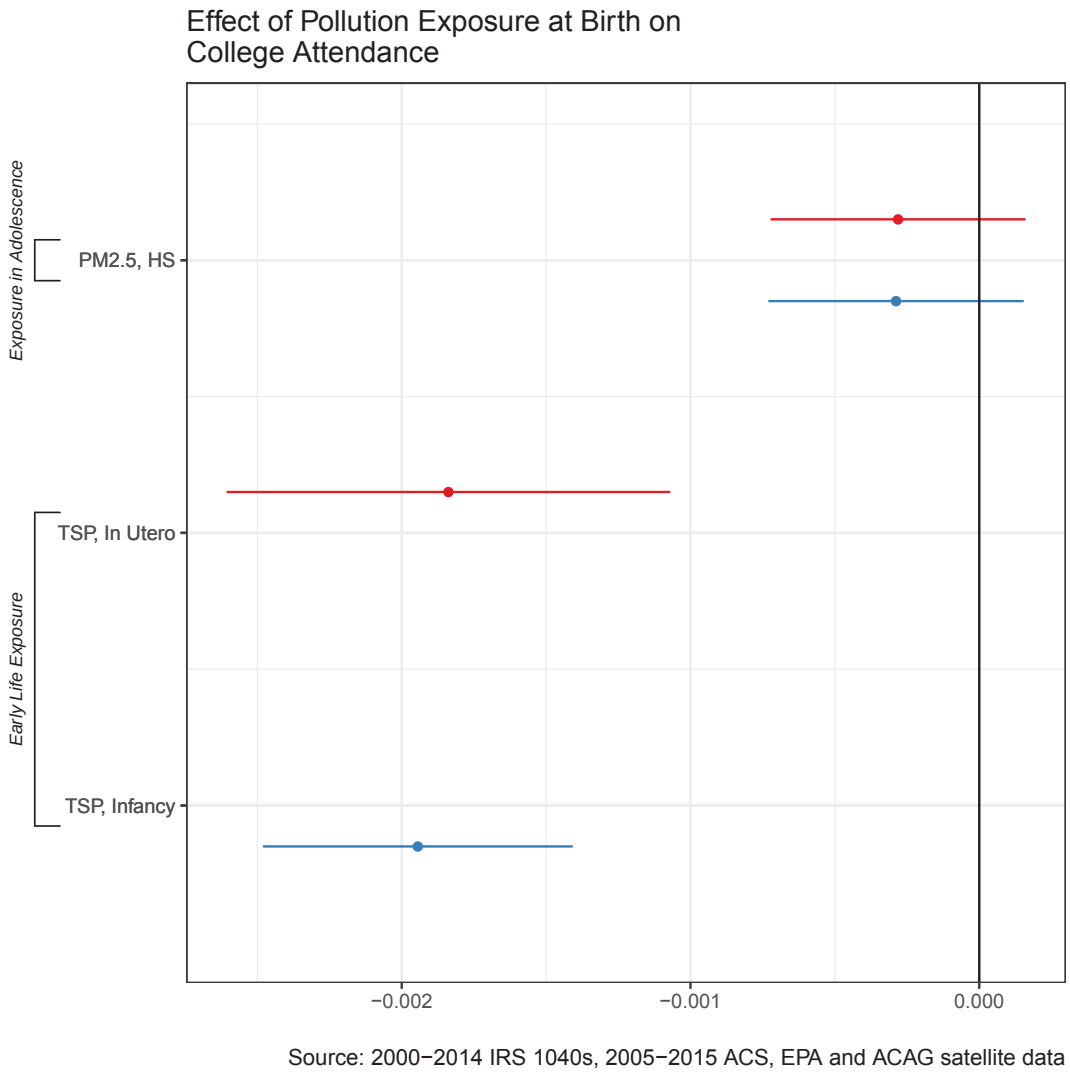
Source: 2000–2014 IRS 1040, 2005 through 2015 ACS and EPA monitor data
Note: This figure shows the year-specific effects of NO_2 Nonattainment designations on TSP and Ozone exposure in utero and during infancy (each panel represents a different model). Confidence intervals that include zero before nonattainment designations took effect indicate that the parallel trends assumption holds.

Figure 3: Test of Parallel Trends Assumption, TSP Exposure, Older Cohort



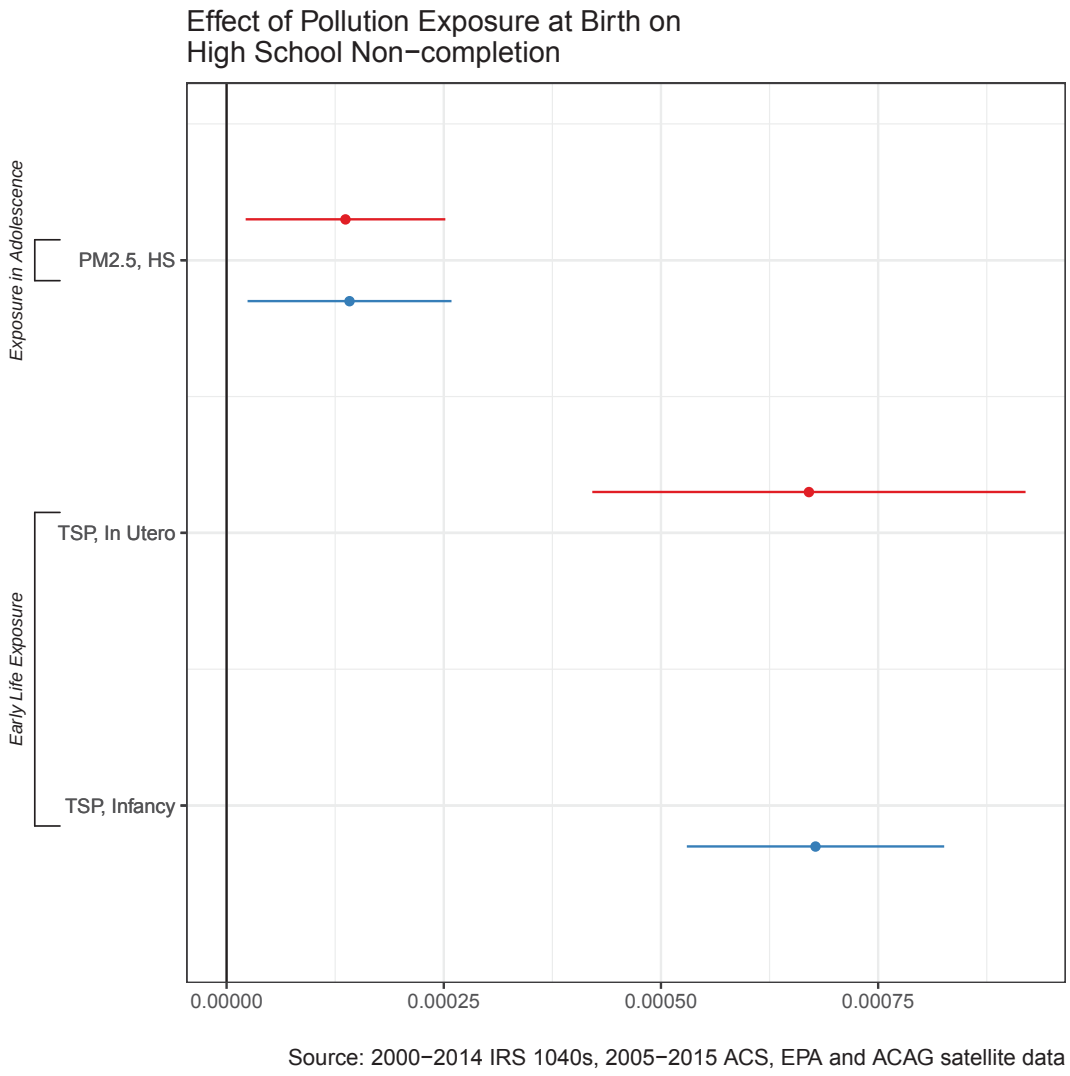
Source: 2000–2014 IRS 1040, 2005 through 2015 ACS and EPA monitor data
Note: This figure shows the year-specific effects of NO_2 Nonattainment designations on TSP and Ozone exposure in utero and during infancy (each panel represents a different model). Confidence intervals that include zero before nonattainment designations took effect indicate that the parallel trends assumption holds.

Figure 4: Graphical Summary of IV Results: College Attendance



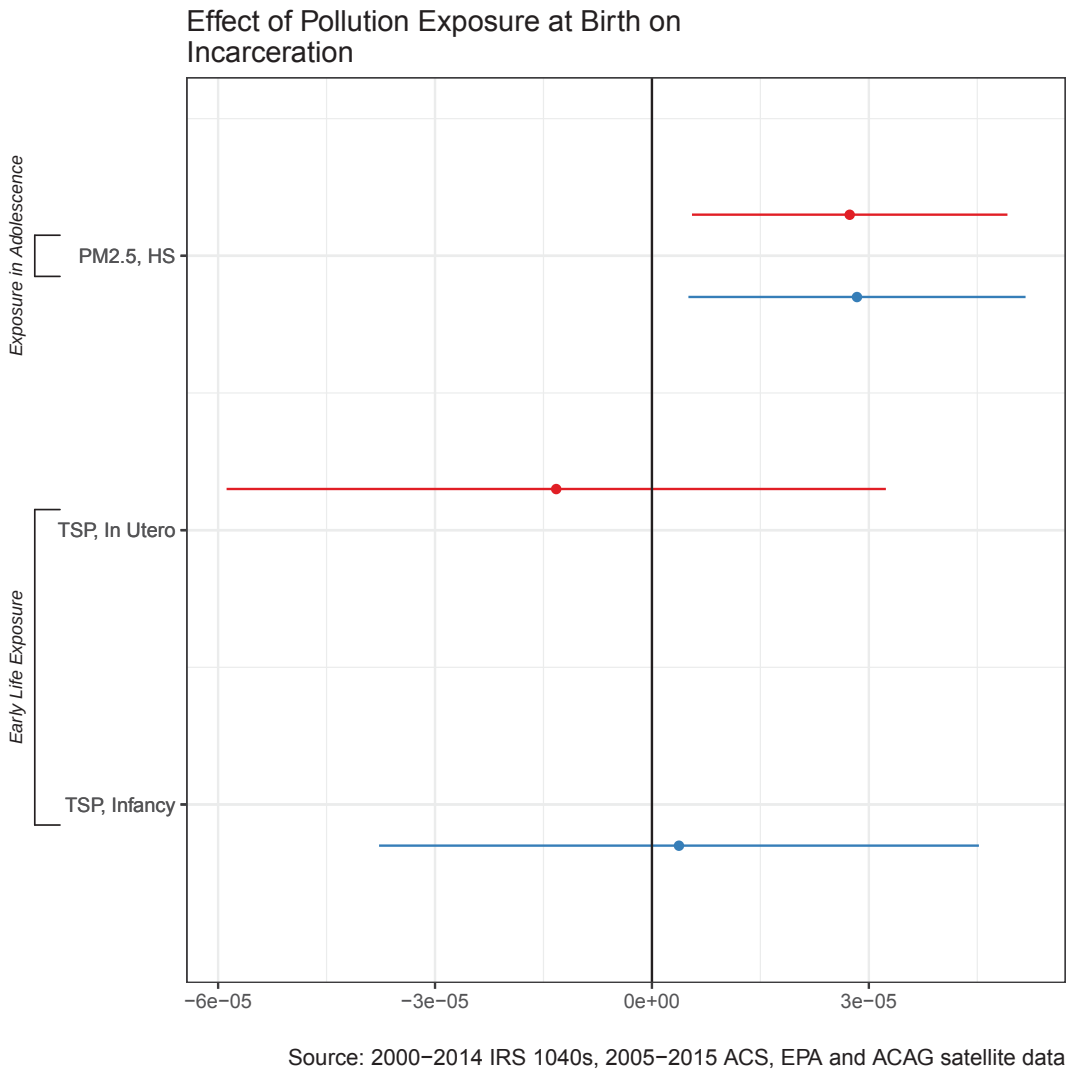
Note: This figure shows the effect of pollution exposure at adolescence on college attendance (top row) and the IV estimates of the effect of pollution exposure at birth on college attendance (bottom four rows) from Tables 2–13. The point estimates can be interpreted as the effect of a 1 unit ($\mu g/m^3$ for TSP and PM2.5 or AQI for ozone) increase in pollution exposure at birth (or in adolescence) on the probability of attending college at ages 19–22.

Figure 5: Graphical Summary of IV Results: High School Non-completion



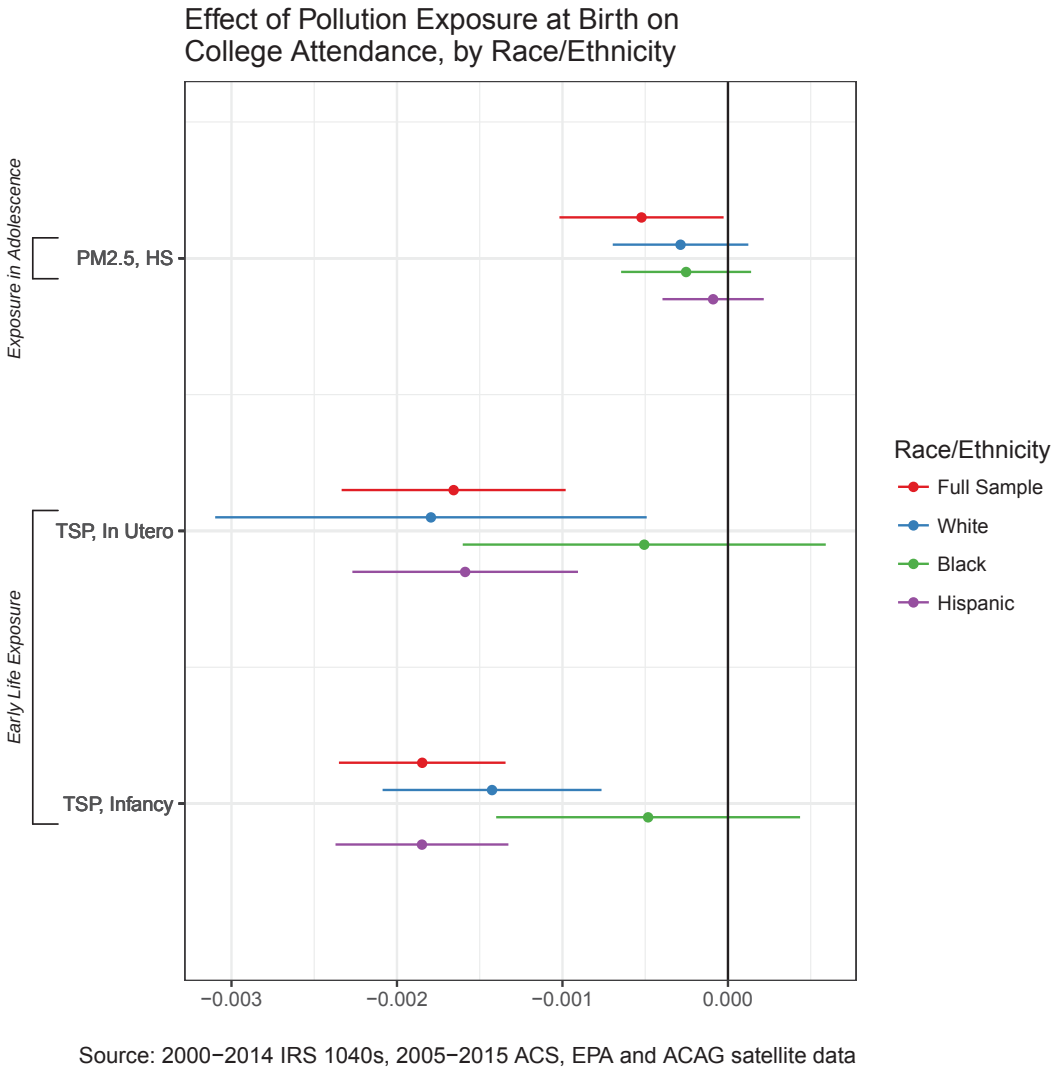
Note: This figure shows the effect of pollution exposure at adolescence on High School Non-completion (top row) and the IV estimates of the effect of pollution exposure at birth on High School Non-completion (bottom four rows) from Tables 6–15. The point estimates can be interpreted as the effect of a 1 unit ($\mu g/m^3$ for TSP and PM2.5 or AQI for ozone) increase in pollution exposure at birth (or in adolescence) on the probability of being a high school dropout at ages 16–24.

Figure 6: Graphical Summary of IV Results: Incarceration



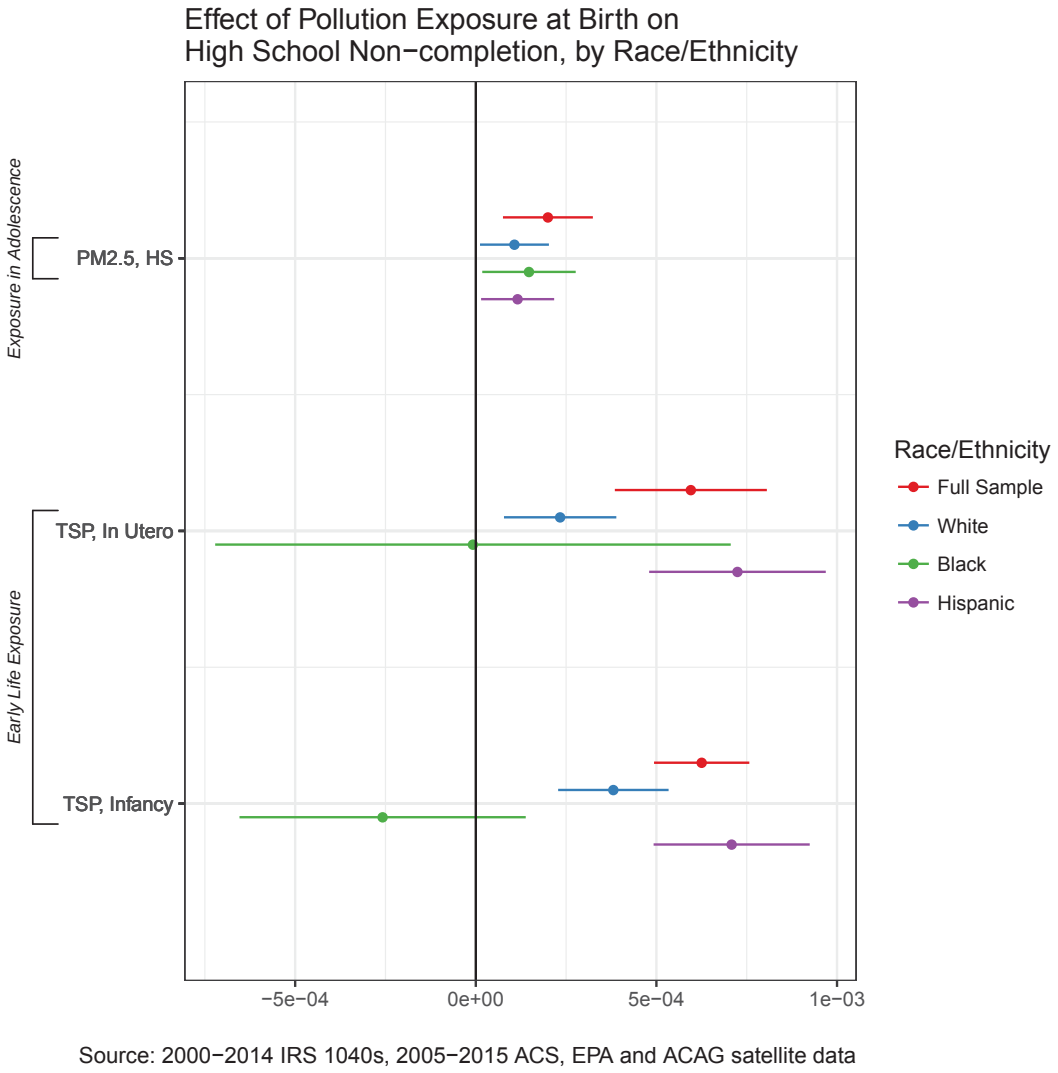
Note: This figure shows the effect of pollution exposure at adolescence on Incarceration (top row) and the IV estimates of the effect of pollution exposure at birth on Incarceration (bottom four rows) from Tables 20–11. The point estimates can be interpreted as the effect of a 1 unit ($\mu g/m^3$ for TSP and PM2.5 or AQI for ozone) increase in pollution exposure at birth (or in adolescence) on the probability of being a incarcerated at ages 16–24.

Figure 7: Graphical Summary of IV Results, Stratified by Race: College Attendance



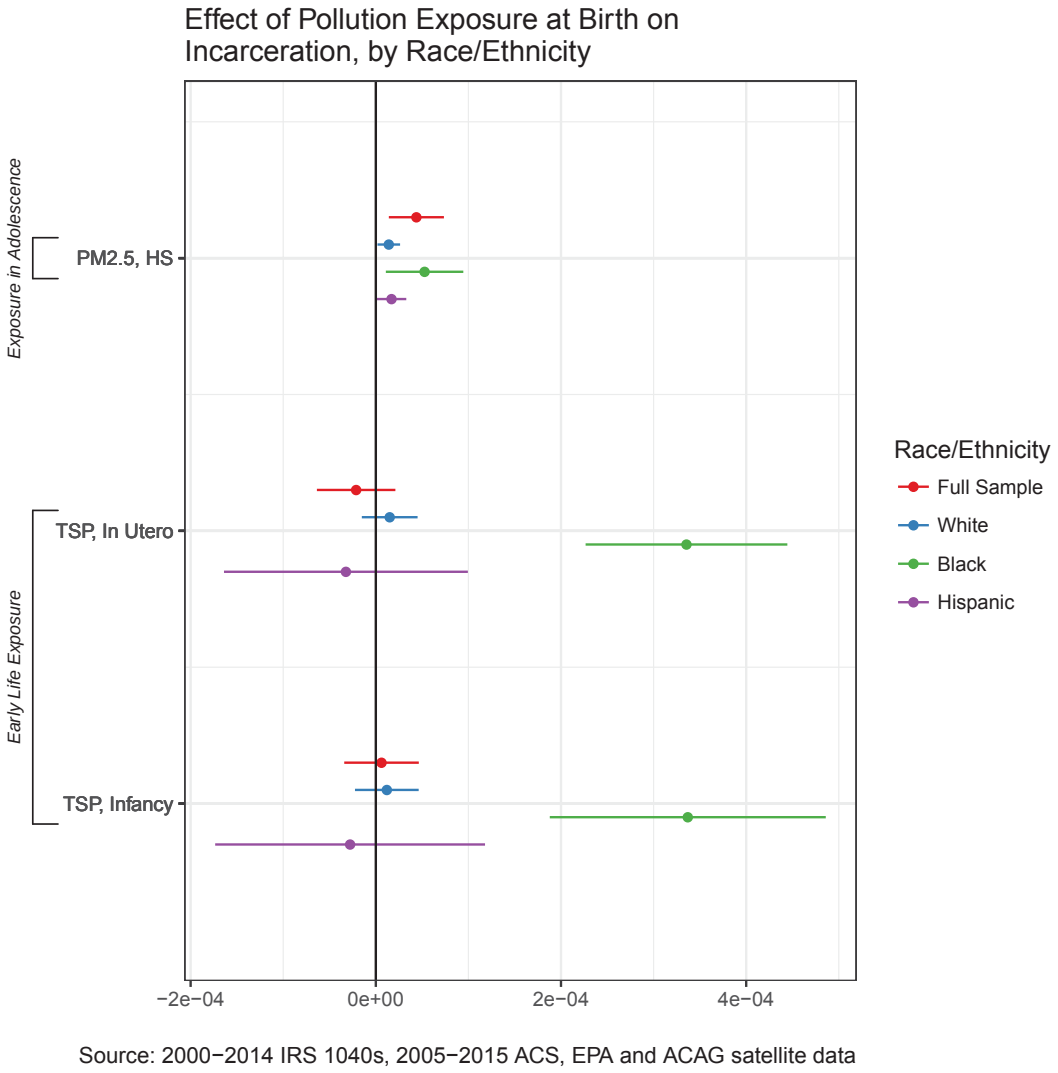
Note: This figure shows the effect of pollution exposure at adolescence on college attendance (top row) and the IV estimates of the effect of pollution exposure at birth on college attendance (bottom four rows) from Tables 16–5. Each row reports 5 results, stratified by race (full sample, all Whites, all non-Whites, all Hispanics, and all non-Hispanic Blacks). The point estimates can be interpreted as the effect of a 1 unit ($\mu g/m^3$ for TSP and PM2.5 or AQI for ozone) increase in pollution exposure at birth (or in adolescence) on the probability of attending college at ages 19–22.

Figure 8: Graphical Summary of IV Results, Stratified by Race: High School Non-completion



Note: This figure shows the effect of pollution exposure at adolescence on High School Non-completion (top row) and the IV estimates of the effect of pollution exposure at birth on High School Non-completion (bottom four rows) from Tables 18–9. Each row reports 5 results, stratified by race (full sample, all Whites, all non-Whites, all Hispanics, and all non-Hispanic Blacks). The point estimates can be interpreted as the effect of a 1 unit ($\mu g/m^3$ for TSP and PM2.5 or AQI for ozone) increase in pollution exposure at birth (or in adolescence) on the probability of being a high school dropout at ages 16–24.

Figure 9: Graphical Summary of IV Results, Stratified by Race: Incarceration



Note: This figure shows the effect of pollution exposure at adolescence on Incarceration (top row) and the IV estimates of the effect of pollution exposure at birth on Incarceration (bottom four rows) from Tables 20–11. Each row reports 5 results, stratified by race (full sample, all Whites, all non-Whites, all Hispanics, and all non-Hispanic Blacks). The point estimates can be interpreted as the effect of a 1 unit ($\mu g/m^3$ for TSP and PM2.5 or AQI for ozone) increase in pollution exposure at birth (or in adolescence) on the probability of being a incarcerated at ages 16–24.

10 Tables

Table 1: Replicating Isen et al. (2016): The Effect of CAA 1970 on Wages at Ages 30-50

	In Utero	Infancy
	(1)	(2)
	Panel A: OLS Results	
TSP Exposure	-4.243^{***}	-2.147
	(1.444)	(1.530)
	Panel B: IV Results	
TSP Exposure	-24.641^{*}	-40.530^{**}
	(12.597)	(19.154)
	Panel C: Reduced Form Results	
Nonattainment	225.257^{*}	233.671
	(135.434)	(149.423)
Observations	1,431,831	1,448,745
First Stage F	21.56	16.71

*p<0.1; **p<0.05; ***p<0.01

All models include birth year, birth month and birth county FE

Standard errors are clustered at the birth state level

Source: 2005 through 2015 ACS and EPA monitor data

Note: This table reports the results of 6 separate regressions (2 in each panel). The outcome variable for each regression is the real wages earned by individuals at ACS response, expressed in 2016 dollars. The regressions in Panel A estimate the effect of TSP exposure in utero or during infancy on wages using OLS. The regressions in Panel B estimate the effect of TSP exposure on wages using nonattainment of TSP NAAQS standards as an instrument for TSP. The regressions in Panel C estimate the reduced form effect of TSP nonattainment status designation on wages. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text. The estimating sample includes all ACS respondents born between 1965–1976 in a county with at least one active TSP monitor in their year of birth.

Table 2: The Effect of TSP Exposure in utero on College Attendance

	Full Term	First Trimester	Second Trimester	Third Trimester
	(1)	(2)	(3)	(4)
		Panel A: OLS Results		
PM2.5, In HS	−0.00023	−0.00023	−0.00023	−0.00024
	(0.00021)	(0.00021)	(0.00021)	(0.00022)
TSP, In Utero	−0.00001	0.00001	−0.000001	−0.00002
	(0.00008)	(0.00004)	(0.00005)	(0.00006)
		Panel B: IV Results		
PM2.5, In HS	−0.00028	−0.00028	−0.00029	−0.00029
	(0.00023)	(0.00023)	(0.00023)	(0.00023)
TSP, In Utero	−0.00184***	−0.00191***	−0.00202***	−0.00212***
	(0.00039)	(0.00044)	(0.00045)	(0.00049)
		Panel C: Reduced Form Results		
PM2.5, In HS	−0.00023	−0.00023	−0.00023	−0.00023
	(0.00021)	(0.00021)	(0.00021)	(0.00021)
Nonattainment	0.02385***	0.02374***	0.02395***	0.02406***
	(0.00405)	(0.00413)	(0.00417)	(0.00414)
Observations	1,115,263	1,105,321	1,102,685	1,100,157
First Stage F	19.5	16.84	18.04	13.97

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 12 separate regressions (4 in each panel). The outcome variable for each regression is an indicator for whether an individual was attending college at ACS survey response. The regressions in Panel A estimate the effect of TSP exposure in utero on college attendance using OLS. The regressions in Panel B estimate the effect of Total Suspended Particulates (TSP) exposure on college attendance using nonattainment of NO_2 NAAQS standards as an instrument for TSP. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on wages. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. The estimating sample includes all ACS respondents aged 19–22, who born between 1985–1996 in a county with at least one active TSP monitor in the year before they were born.

Table 3: The Effect of TSP Exposure during infancy on College Attendance

	0-12 Mos.	0-3 Mos.	3-6 Mos.	6-9 Mos.
	(1)	(2)	(3)	(4)
Panel A: OLS Results				
PM2.5, In HS	−0.00023	−0.00023	−0.00023	−0.00023
	(0.00021)	(0.00021)	(0.00021)	(0.00022)
TSP, Infancy	−0.00001	−0.00004	0.00003	−0.00008
	(0.00009)	(0.00006)	(0.00007)	(0.00007)
Panel B: IV Results				
PM2.5, In HS	−0.00030	−0.00031	−0.00032	−0.00032
	(0.00022)	(0.00022)	(0.00022)	(0.00022)
TSP, Infancy	−0.00250***	−0.00304**	−0.00295***	−0.00307***
	(0.00071)	(0.00121)	(0.00099)	(0.00100)
Panel C: Reduced Form Results				
PM2.5, In HS	−0.00023	−0.00023	−0.00023	−0.00023
	(0.00021)	(0.00021)	(0.00021)	(0.00021)
Nonattainment	0.02402***	0.02401***	0.02434***	0.02405***
	(0.00383)	(0.00412)	(0.00382)	(0.00369)
Observations	1,110,030	1,097,436	1,094,795	1,091,798
First Stage F	9.36	4.59	6.44	7.43

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 12 separate regressions (4 in each panel). The outcome variable for each regression is an indicator for whether an individual was attending college at ACS survey response. The regressions in Panel A estimate the effect of Total Suspended Particulates (TSP) exposure during infancy on college attendance using OLS. The regressions in Panel B estimate the effect of TSP exposure on college attendance using nonattainment of NO_2 NAAQS standards as an instrument for TSP. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on wages. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. The estimating sample includes all ACS respondents aged 19–22, who born between 1985–1996 in a county with at least one active TSP monitor in their year of birth.

Table 4: The Effect of TSP Exposure in utero on College Attendance, by Race and Family Income

	Whites	Non-whites	Black	Hispanics	Bottom 2 quintiles	Top 3 quintiles
	(1)	(2)	(3)	(4)	(5)	(6)
			Panel A: OLS Results			
PM2.5, In HS	−0.0002	−0.0001	−0.0002	−0.0001	−0.0001	−0.0002
	(0.0002)	(0.0002)	(0.0002)	(0.0001)	(0.0001)	(0.0001)
TSP, In Utero	0.00003	−0.00005	0.00001	0.00002	−0.0001	0.00002
	(0.0001)	(0.0001)	(0.0003)	(0.0001)	(0.0001)	(0.0001)
			Panel B: IV Results			
PM2.5, In HS	−0.0003	−0.0002	−0.0003	−0.0001	−0.0001	−0.0003*
	(0.0002)	(0.0002)	(0.0002)	(0.0002)	(0.0002)	(0.0002)
TSP, In Utero	−0.002***	−0.001***	−0.001	−0.002***	−0.001***	−0.002***
	(0.001)	(0.0002)	(0.001)	(0.0003)	(0.0004)	(0.0004)
			Panel C: Reduced Form Results			
PM2.5, In HS	−0.0002	−0.0001	−0.0002	−0.0001	−0.0001	−0.0002
	(0.0002)	(0.0002)	(0.0002)	(0.0001)	(0.0001)	(0.0001)
Nonattainment	0.024***	0.018***	0.009	0.018***	0.015***	0.025***
	(0.005)	(0.005)	(0.009)	(0.005)	(0.004)	(0.005)
Observations	785,441	329,822	148,628	167,110	428,433	686,830
First Stage F	21.43	17.56	54.35	9.93	16.7	22.95

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 18 separate regressions (6 in each panel). The outcome variable for each regression is an indicator for whether an individual was attending college at ACS survey response. The regressions in Panel A estimate the effect of TSP exposure in utero on college attendance using OLS. The regressions in Panel B estimate the effect of Total Suspended Particulates (TSP) exposure on college attendance using nonattainment of NO_2 NAAQS standards as an instrument for TSP. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on wages. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. Each column represents a different subsample based on race (columns 1-4) or family income (columns 5-6).

Table 5: The Effect of TSP Exposure during infancy on College Attendance, by Race and Family Income

	Whites	Non-whites	Black	Hispanics	Bottom 2 quintiles	Top 3 quintiles
	(1)	(2)	(3)	(4)	(5)	(6)
			Panel A: OLS Results			
PM2.5, In HS	−0.0002	−0.0001	−0.0002	−0.0001	−0.0001	−0.0002
	(0.0002)	(0.0002)	(0.0002)	(0.0001)	(0.0001)	(0.0001)
TSP, Infancy	0.0001	−0.0001	0.0003	−0.0002*	0.0001	−0.0001
	(0.0001)	(0.0001)	(0.0003)	(0.0001)	(0.0001)	(0.0001)
			Panel B: IV Results			
PM2.5, In HS	−0.0003	−0.0002	−0.0002	−0.0001	−0.0002	−0.0003**
	(0.0002)	(0.0002)	(0.0002)	(0.0002)	(0.0001)	(0.0001)
TSP, Infancy	−0.002**	−0.002***	−0.001	−0.002***	−0.002**	−0.003***
	(0.001)	(0.0004)	(0.001)	(0.001)	(0.001)	(0.001)
			Panel C: Reduced Form Results			
PM2.5, In HS	−0.0002	−0.0001	−0.0002	−0.0001	−0.0001	−0.0002
	(0.0002)	(0.0002)	(0.0002)	(0.0001)	(0.0001)	(0.0001)
Nonattainment	0.024***	0.019***	0.013	0.017***	0.015***	0.026***
	(0.005)	(0.005)	(0.011)	(0.004)	(0.003)	(0.005)
Observations	780,849	329,181	148,406	166,811	426,903	683,127
First Stage F	10.43	10.05	21.6	8.09	9.85	9.16

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 18 separate regressions (6 in each panel). The outcome variable for each regression is an indicator for whether an individual was attending college at ACS survey response. The regressions in Panel A estimate the effect of TSP exposure during infancy on college attendance using OLS. The regressions in Panel B estimate the effect of Total Suspended Particulates (TSP) exposure on college attendance using nonattainment of NO_2 NAAQS standards as an instrument for TSP. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on wages. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. Each column represents a different subsample based on race (columns 1-4) or family income (columns 5-6).

10.1 High School Non-completion

Table 6: The Effect of TSP Exposure in utero on High School Non-completion

	Full Term	First Trimester	Second Trimester	Third Trimester
	(1)	(2)	(3)	(4)
		Panel A: OLS Results		
PM2.5, In HS	0.00012**	0.00012**	0.00012**	0.00012**
	(0.00005)	(0.00005)	(0.00005)	(0.00006)
TSP, In Utero	0.00001	0.00001	0.00001	−0.00001
	(0.00003)	(0.00002)	(0.00002)	(0.00002)
		Panel B: IV Results		
PM2.5, In HS	0.00014**	0.00014**	0.00014**	0.00014**
	(0.00006)	(0.00006)	(0.00006)	(0.00006)
TSP, In Utero	0.00067***	0.00068***	0.00072***	0.00077***
	(0.00013)	(0.00014)	(0.00014)	(0.00017)
		Panel C: Reduced Form Results		
PM2.5, In HS	0.00012**	0.00012**	0.00012**	0.00012**
	(0.00005)	(0.00005)	(0.00005)	(0.00006)
Nonattainment	−0.00955***	−0.00947***	−0.00957***	−0.00950***
	(0.00114)	(0.00114)	(0.00113)	(0.00114)
Observations	2,671,235	2,647,219	2,639,993	2,633,069
First Stage F	46.95	33.24	39.76	36.76

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 12 separate regressions (4 in each panel). The outcome variable for each regression is an indicator for whether an individual had not completed High School at ACS survey response. The regressions in Panel A estimate the effect of Total Suspended Particulates (TSP) exposure in utero on High School non-completion using OLS. The regressions in Panel B estimate the effect of TSP exposure on High School non-completion using nonattainment of NO_2 NAAQS standards as an instrument for TSP. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on High School non-completion. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. The estimating sample includes all ACS respondents aged 16–24, who born between 1985–1996 in a county with at least one active TSP monitor in the year before they were born.

Table 7: The Effect of TSP Exposure during Infancy on High School Non-completion

	0-12 Mos.	0-3 Mos.	3-6 Mos.	6-9 Mos.
	(1)	(2)	(3)	(4)
Panel A: OLS Results				
PM2.5, In HS	0.00012**	0.00012**	0.00012**	0.00012**
	(0.00006)	(0.00006)	(0.00006)	(0.00006)
TSP, Infancy	0.00006	0.00001	0.00004**	0.00003
	(0.00005)	(0.00002)	(0.00002)	(0.00002)
Panel B: IV Results				
PM2.5, In HS	0.00015***	0.00015**	0.00015***	0.00015***
	(0.00006)	(0.00006)	(0.00006)	(0.00006)
TSP, Infancy	0.00091***	0.00098***	0.00098***	0.00105***
	(0.00022)	(0.00029)	(0.00024)	(0.00025)
Panel C: Reduced Form Results				
PM2.5, In HS	0.00012**	0.00012**	0.00012**	0.00012**
	(0.00006)	(0.00006)	(0.00006)	(0.00006)
Nonattainment	−0.00961***	−0.00947***	−0.00963***	−0.00951***
	(0.00116)	(0.00112)	(0.00109)	(0.00108)
Observations	2,604,511	2,622,625	2,605,830	2,581,561
First Stage F	23.55	17.08	22.24	23.96

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 12 separate regressions (4 in each panel). The outcome variable for each regression is an indicator for whether an individual had not completed High School at ACS survey response. The regressions in Panel A estimate the effect of Total Suspended Particulates (TSP) exposure during infancy on High School non-completion using OLS. The regressions in Panel B estimate the effect of TSP exposure on High School non-completion using nonattainment of NO_2 NAAQS standards as an instrument for TSP. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on High School non-completion. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. The estimating sample includes all ACS respondents aged 16–24, who born between 1985–1996 in a county with at least one active TSP monitor in the year they were born.

Table 8: The Effect of TSP Exposure in utero on High School Non-completion, by Race and Family Income

	Whites	Non-whites	Black	Hispanics	Bottom 2 quintiles	Top 3 quintiles
	(1)	(2)	(3)	(4)	(5)	(6)
			Panel A: OLS Results			
PM2.5, In HS	0.0001**	0.0001**	0.0001**	0.0001**	0.0001***	0.00005**
	(0.00005)	(0.0001)	(0.0001)	(0.00004)	(0.00005)	(0.00002)
TSP, In Utero	0.00001	0.00000	−0.0001	−0.00000	0.00003	0.00001
	(0.00002)	(0.00003)	(0.0001)	(0.00003)	(0.00003)	(0.00002)
			Panel B: IV Results			
PM2.5, In HS	0.0001**	0.0001**	0.0001**	0.0001**	0.0002***	0.0001***
	(0.00005)	(0.0001)	(0.0001)	(0.0001)	(0.0001)	(0.00002)
TSP, In Utero	0.0003***	0.001***	−0.00001	0.001***	0.001***	0.001***
	(0.0001)	(0.0001)	(0.0004)	(0.0002)	(0.0001)	(0.0001)
			Panel C: Reduced Form Results			
PM2.5, In HS	0.0001**	0.0001**	0.0001**	0.0001**	0.0001***	0.00005**
	(0.00005)	(0.0001)	(0.0001)	(0.00005)	(0.00005)	(0.00002)
Nonattainment	−0.003***	−0.010***	0.0001	−0.009***	−0.010***	−0.008***
	(0.001)	(0.001)	(0.007)	(0.002)	(0.001)	(0.001)
Observations	1,859,657	780,336	349,019	396,839	1,020,737	1,619,256
First Stage F	41.04	30.8	159.92	13.52	40.6	39.36

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 18 separate regressions (6 in each panel). The outcome variable for each regression is an indicator for whether an individual had not completed High School at ACS survey response. The regressions in Panel A estimate the effect of Total Suspended Particulates (TSP) exposure in utero on High School non-completion using OLS. The regressions in Panel B estimate the effect of TSP exposure on High School non-completion using nonattainment of NO_2 NAAQS standards as an instrument for TSP. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on High School non-completion. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. Each column represents a different subsample based on race (columns 1-4) or family income (columns 5-6).

Table 9: The Effect of TSP Exposure infant on High School Non-completion, by Race and Family Income

	Whites	Non-whites	Black	Hispanics	Bottom 2 quintiles	Top 3 quintiles
	(1)	(2)	(3)	(4)	(5)	(6)
			Panel A: OLS Results			
PM2.5, In HS	0.0001**	0.0001**	0.0002**	0.0001**	0.0001***	0.00005***
	(0.00005)	(0.0001)	(0.0001)	(0.00004)	(0.00005)	(0.00002)
TSP, Infancy	0.00005	0.0001	0.0001	0.0001	0.0001	0.0001
	(0.00004)	(0.0001)	(0.0001)	(0.0001)	(0.0001)	(0.00004)
			Panel B: IV Results			
PM2.5, In HS	0.0001**	0.0001**	0.0002**	0.0001**	0.0002***	0.0001***
	(0.00005)	(0.0001)	(0.0001)	(0.0001)	(0.0001)	(0.00002)
TSP, Infancy	0.0003**	0.001***	0.0002	0.001***	0.001***	0.001***
	(0.0001)	(0.0002)	(0.0004)	(0.0002)	(0.0002)	(0.0001)
			Panel C: Reduced Form Results			
PM2.5, In HS	0.0001**	0.0001**	0.0002**	0.0001**	0.0001***	0.00005**
	(0.00005)	(0.0001)	(0.0001)	(0.00004)	(0.00005)	(0.00002)
Nonattainment	−0.003**	−0.011***	−0.003	−0.009***	−0.010***	−0.008***
	(0.001)	(0.001)	(0.006)	(0.002)	(0.002)	(0.001)
Observations	1,834,761	769,750	346,300	389,603	1,008,361	1,596,150
First Stage F	22.7	24.42	56.57	14.82	27.54	20.71

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 12 separate regressions (4 in each panel). The outcome variable for each regression is an indicator for whether an individual had not completed High School at ACS survey response. The regressions in Panel A estimate the effect of Total Suspended Particulates (TSP) exposure during infancy on High School non-completion using OLS. The regressions in Panel B estimate the effect of TSP exposure on High School non-completion using nonattainment of NO_2 NAAQS standards as an instrument for TSP. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on High School non-completion. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. Each column represents a different subsample based on race (columns 1-4) or family income (columns 5-6).

Table 10: The Effect of TSP Exposure in utero on Incarceration, by Race and Family Income

	Whites	Non-whites	Black	Hispanics	Bottom 2 quintiles	Top 3 quintiles
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: OLS Results						
PM2.5, In HS	0.00001**	0.00004**	0.00005**	0.00002**	0.00005***	0.00001***
	(0.00001)	(0.00002)	(0.00002)	(0.00001)	(0.00002)	(0.00000)
TSP, In Utero	0.00000	−0.00003	0.00004	−0.00001	−0.00001	0.00000
	(0.00001)	(0.00002)	(0.00004)	(0.00002)	(0.00002)	(0.00001)
Panel B: IV Results						
PM2.5, In HS	0.00001**	0.00004**	0.0001**	0.00002**	0.00004***	0.00001***
	(0.00001)	(0.00002)	(0.00002)	(0.00001)	(0.00002)	(0.00000)
TSP, In Utero	0.00002	−0.0001	0.0003***	−0.00003	−0.0001***	0.00003**
	(0.00002)	(0.0001)	(0.0001)	(0.0001)	(0.00003)	(0.00001)
Panel C: Reduced Form Results						
PM2.5, In HS	0.00001**	0.00004**	0.00005**	0.00002**	0.00005***	0.00001***
	(0.00001)	(0.00002)	(0.00002)	(0.00001)	(0.00002)	(0.00000)
Nonattainment	−0.0002	0.001	−0.006***	0.0004	0.001**	−0.0004**
	(0.0002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.0002)
Observations	1,883,137	788,098	353,611	399,560	1,033,789	1,637,446
First Stage F	46.39	38.01	140.47	16.3	45.52	48.57

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 18 separate regressions (6 in each panel). The outcome variable for each regression is an indicator for whether an individual was incarcerated at ACS survey response. The regressions in Panel A estimate the effect of TSP exposure in utero on incarceration using OLS. The regressions in Panel B estimate the effect of TSP exposure on incarceration using nonattainment of NO_2 NAAQS standards as an instrument for TSP. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on incarceration. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. Each column represents a different subsample based on race (columns 1-4) or family income (columns 5-6).

Table 11: The Effect of TSP Exposure infant on Incarceration, by Race and Family Income

	Whites	Non-whites	Black	Hispanics	Bottom 2 quintiles	Top 3 quintiles
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: OLS Results						
PM2.5, In HS	0.00001**	0.00004**	0.00005**	0.00002**	0.00005***	0.00001***
	(0.00001)	(0.00002)	(0.00002)	(0.00001)	(0.00002)	(0.00000)
TSP, Infancy	0.00001	−0.00002	0.0001	0.00001	−0.00000	0.00001
	(0.00001)	(0.00003)	(0.00004)	(0.00003)	(0.00002)	(0.00001)
Panel B: IV Results						
PM2.5, In HS	0.00001**	0.00004**	0.0001**	0.00002*	0.00004***	0.00002***
	(0.00001)	(0.00002)	(0.00002)	(0.00001)	(0.00002)	(0.00000)
TSP, Infancy	0.00001	−0.0001	0.0004***	−0.00004	−0.0001***	0.00004***
	(0.00002)	(0.0001)	(0.0001)	(0.0001)	(0.00004)	(0.00002)
Panel C: Reduced Form Results						
PM2.5, In HS	0.00001**	0.00004**	0.00005**	0.00002**	0.00005***	0.00001***
	(0.00001)	(0.00002)	(0.00002)	(0.00001)	(0.00002)	(0.00000)
Nonattainment	−0.0001	0.001	−0.006***	0.0004	0.001**	−0.0004**
	(0.0002)	(0.001)	(0.001)	(0.001)	(0.0005)	(0.0002)
Observations	1,834,761	769,750	346,300	389,603	1,008,361	1,596,150
First Stage F	22.7	24.42	56.57	14.82	27.54	20.71

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 8 separate regressions (6 in each panel). The outcome variable for each regression is an indicator for whether an individual was incarcerated at ACS survey response. The regressions in Panel A estimate the effect of TSP exposure during infancy on incarceration using OLS. The regressions in Panel B estimate the effect of TSP exposure on incarceration using nonattainment of NO_2 NAAQS standards as an instrument for TSP. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on incarceration. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. Each column represents a different subsample based on race (columns 1-4) or family income (columns 5-6).

A The Long Term Effects of Ozone Exposure

The key results for the younger cohort of individuals are hinge on the effect of EPA nonattainment designations on the emissions of NOx, which is a precursor to the formation of some species of particulate matter, and NOx co-pollutants, which include both particulate matter itself as well as other PM precursors (such as SO_2). The key results for TSP suggest that this strategy is sound: the first stage effect of nonattainment status on TSP exposure is relevant and passes a series of placebo tests, which bolster the case that the identifying assumptions hold. However, note that the process by which nonattainment designations reduce TSP exposure – by affecting the amount of fossil fuel combustion, either in coal-fired power plants or diesel-fueled mobile sources – might also affect other co-pollutants to TSP such as ozone. To examine this, I produce a set of results paralleling the above TSP analysis using instead ozone exposure (measure in Air Quality Index units) as a pollutant of interest.

Tables 12 - 13 report OLS, IV and reduced form results characterizing the relationship between ozone exposure and college attendance, while Tables 14 - 15 repeat this exercise for high school non-completion, and Tables 20 -21 repeat this for incarceration. In general, these results are in line with the TSP results above - ozone exposure decreases college attendance, increases high school non-completion and increases incarceration. There are some differences, however. Note that the ozone exposure OLS results are more precisely estimated than the equivalent TSP results, with a majority of the ozone OLS effects being statistically significant at the 5 percent level.

The top panel of Table 12 reports results from models exploring how in utero ozone exposure affects college attendance, again with a structure mirroring previous in utero tables. In contrast to the TSP models, there is statistically significant evidence of a negative effect of pollution exposure on future college attendance. The effect of average exposure over a full-term 9-month pregnancy (column 1) is both statistically and economically significant: a 1 AQI point increase in ozone exposure decreases the probability of college attendance at ages 19–22 by 0.02 percent. To contextualize this, the nationwide average AQI for ozone has declined by about 30 AQI points between 1980-1990. An AQI drop of this magnitude implies an increase in the probability of college attendance of 0.6 percent. Within individual trimesters, the most statistically significant evidence points to effects in the third trimester being important. There is a similar pattern of effects for infant exposure, as shown in Table 13. The effect of ozone exposure in the first year of life is statistically significant, implying that a 1 AQI increase in ozone decreases the probability of college attendance by 0.03 percent. Again exposure near birth: in the first 3 months of life, seems to be most important.

While there has been somewhat mixed evidence of a statistically significant effects of TSP on college attendance, the IV models describing the effect of ozone on college attendance provide somewhat clearer evidence of a clear negative link between air quality and human capital accumulation than did the OLS results in the top panel of 12. The middle panel of Table 12 reports results from IV models describing the effect of ozone exposure in utero on college attendance at ages 19–22, where NO_2 nonattainment designation is used as an instrument for ozone exposure. The first column reports overall in utero exposure, while columns 2-4 summarise models using average exposure in individual trimesters as the independent variable of interest. Exposure in the first trimester (column 2) has a larger effect than exposure in the second and third trimesters, although this effect is imprecisely estimated (it is only marginally statistically significant). The effect of in utero average ozone exposure (column 1) is negative and statistically significant and implies a economically substantial effect size. A 10 AQI increase in ozone exposure in utero decreases college attendance by 1 percent (compared to an average attendance rate of 53 percent in the ACS sample).

Similarly, the middle panel of Table 13 reports results for infant exposure to ozone, instrumenting for ozone exposure with NO_2 nonattainment designation. Columns 2-4, which report results using individual trimesters after birth as the exposure window, report large but imprecise estimate for the first 6 months after birth, but larger and more precise estimates for exposure windows after 6 months (column 3) and for the average exposure in the first year of life (column 1). This effect size is comparable to the effect size for the overall in utero exposure model in the middle panel of Table 12, implying a large 2 percent decrease in college attendance in response to a 10 AQI increase in ozone exposure.

In 1987, the national average ozone AQI was 174. Ten years later, in 1996, the national average ozone AQI stood at 150, a decline of 24 AQI points. As a first thought experiment, assume that absent the CAAA, national average ozone AQI would have remained at 174. Compared to this no CAAA baseline, what effect on wages did the actual improvement in air quality have? Consider first children who were born in 1997 (who would have been exposed to an ozone AQI of 150 in utero in reality, versus 174 in the counterfactual). Taking the OLS estimate in Table 12, column 4 as given, this counterfactual improvement in air quality would have increased college attendance by $-24 \times -0.0002 = 0.48$ percent. Thus our estimate of the wage effects using the OLS estimate is

$$\Delta W_{inutero} = -24 \times -0.0002 \times 0.6 \times 17500 = \$50.40$$

On the other hand, we (unsurprisingly) see substantially higher wage premiums for the much larger IV

estimates in Table 12, column 4. The IV estimates imply a wage premium of

$$\Delta W^{IV}_{inutero} = -24 \times -0.001 \times 0.6 \times 17500 = \$252$$

There is a similar range of potential wage effects for the cohort of children born in 1996 (who would have been exposed to an ozone AQI of 150 in the first year of life, compared to a counterfactual 174). Again taking the relevant OLS estimate as given (from Table 13, column 4), the implied pollution wage premium is

$$\Delta W_{infant} = -24 \times -0.0003 \times 0.6 \times 17500 = \$75.60$$

Again, the larger IV estimates (Table 13, column 4) imply a sharper effect

$$\Delta W^{IV}_{infant} = -24 \times -0.002 \times 0.6 \times 17500 = \$504$$

Although the lower range (i.e. the OLS results) of these wage effects seem relatively small for an individual, keep in mind two factors. First, these are annual effects. Compounded over a whole working life, these impacts are much larger – at a 3 percent discount rate, the discounted present value of the stream of infant pollution wage premia is $1799.90 over a 40 year career. Second, these impacts are for a single individual in a single cohort. The aggregate effect of these small changes is much larger. Via CDC vital statistics, around 4 million births occurred in 1997 in the United States. (A similar number occurred in 1996). If we sum the expected wage premium over all these individuals, we can see that the in utero OLS effects amount to $201,600,000 in additional aggregate wages, while the early life effects amount to $302,400,000 in aggregate additional wages. Again these are numbers for an individual cohort. If we sum the flow of these aggregate wage premia, we find that the discounted present value (at a 3 percent discount rate) over an average 40 year career is $4.8 billion for the in utero wage effects, and $7.2 billion for the early life effects. The IV effects imply an even larger effect: approximately $2 billion per year per cohort in additional aggregate wages (using the infant effect), or a discounted present value of $48 billion over a 40-year career.

Likewise, the top panel of Table 14 shows results from OLS models analyzing the effect of ozone exposure in utero on high school non-completion. The results here are decidedly mixed: ozone exposure increases high school non completion in the first trimester, but has a negative effect on non-completion in the third trimester; the overall in utero effect is positive but statistically insignificant. As in the models of TSP exposure in utero, the effect of exposure to particulates during late adolescence increases the probability of

high school non-completion separately from the infant TSP exposure (although these parameters are less precisely estimated than in Table 6).

The middle panel of Table 14 reports results from IV models of the effect of ozone exposure in utero on high school non-completion. The effects are larger than the OLS results, uniformly positive and statistically significant. A 1 AQI unit increase in ozone exposure corresponds to a 0.3 percent increase in the high school non-completion rate. Similarly, ozone exposure after birth uniformly leads to increases in the high school dropout rate, with a 1 AQI increase in ozone exposure corresponding to a 0.2 percent increase in the probability of high school non-completion. The effect sizes appear to support the first trimester in utero, and the period corresponding to 6-9 months after birth as particularly crucial windows of exposure.

Table 12: The Effect of Ozone Exposure in utero on College Attendance

	Full Term	First Trimester	Second Trimester	Third Trimester
	(1)	(2)	(3)	(4)
		Panel A: OLS Results		
PM2.5, In HS	−0.0002	−0.0002	−0.0002	−0.0002
	(0.0002)	(0.0002)	(0.0002)	(0.0002)
Ozone, In Utero	−0.0002**	−0.0001**	−0.0001	−0.00003
	(0.0001)	(0.00005)	(0.00004)	(0.00003)
		Panel B: IV Results		
PM2.5, In HS	−0.0002	−0.0002	−0.0002	−0.0002
	(0.0002)	(0.0002)	(0.0002)	(0.0002)
Ozone, In Utero	−0.005**	−0.003***	−0.004***	−0.029
	(0.002)	(0.001)	(0.001)	(0.085)
		Panel C: Reduced Form Results		
PM2.5, In HS	−0.0002	−0.0002	−0.0002	−0.0002
	(0.0002)	(0.0002)	(0.0002)	(0.0002)
Nonattainment	0.026***	0.026***	0.026***	0.025***
	(0.004)	(0.004)	(0.004)	(0.004)
Observations	964,391	910,171	908,114	905,846
First Stage F	4.23	11.21	5.23	0.11

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 12 separate regressions (4 in each panel). The outcome variable for each regression is an indicator for whether an individual was attending college at ACS survey response. The regressions in Panel A estimate the effect of ozone exposure in utero on college attendance using OLS. The regressions in Panel B estimate the effect of ozone exposure on college attendance using nonattainment of NO_2 NAAQS standards as an instrument for ozone. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on wages. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. The estimating sample includes all ACS respondents aged 19–22, who born between 1987–1997 in a county with at least one active ozone monitor in the year before they were born.

Table 13: The Effect of Ozone Exposure infant on College Attendance

	0-12 Mos.	0-3 Mos.	3-6 Mos.	6-9 Mos.
	(1)	(2)	(3)	(4)
Panel A: OLS Results				
PM2.5, In HS	−0.00022	−0.00020	−0.00019	−0.00020
	(0.00023)	(0.00022)	(0.00022)	(0.00022)
Ozone, Infancy	−0.00036***	−0.000002	−0.00003	−0.00011**
	(0.00014)	(0.00004)	(0.00003)	(0.00005)
Panel B: IV Results				
PM2.5, In HS	−0.00021	−0.00018	−0.00017	−0.00019
	(0.00022)	(0.00022)	(0.00021)	(0.00022)
Ozone, Infancy	−0.00323***	−0.00485***	−0.00401***	−0.00238***
	(0.00052)	(0.00141)	(0.00078)	(0.00029)
Panel C: Reduced Form Results				
PM2.5, In HS	−0.00022	−0.00020	−0.00019	−0.00020
	(0.00023)	(0.00022)	(0.00022)	(0.00022)
Nonattainment	0.02612***	0.02571***	0.02592***	0.02585***
	(0.00428)	(0.00442)	(0.00396)	(0.00402)
Observations	974,500	912,845	908,891	911,292
First Stage F	21.81	7.24	15.84	26.54

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 12 separate regressions (4 in each panel). The outcome variable for each regression is an indicator for whether an individual was attending college at ACS survey response. The regressions in Panel A estimate the effect of ozone exposure during infancy on college attendance using OLS. The regressions in Panel B estimate the effect of ozone exposure on college attendance using nonattainment of NO_2 NAAQS standards as an instrument for ozone. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on wages. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. The estimating sample includes all ACS respondents aged 19–22, who born between 1987–1997 in a county with at least one active ozone monitor in their year of birth.

Table 14: The Effect of Ozone Exposure in utero on High School Non-completion

	Full Term	First Trimester	Second Trimester	Third Trimester
	(1)	(2)	(3)	(4)
		Panel A: OLS Results		
PM2.5, In HS	0.0001**	0.0001*	0.0001*	0.0001*
	(0.0001)	(0.0001)	(0.0001)	(0.0001)
Ozone, In Utero	0.00003	−0.00003***	0.00001	0.00004***
	(0.00003)	(0.00001)	(0.00001)	(0.00001)
		Panel B: IV Results		
PM2.5, In HS	0.0001**	0.0001*	0.0001**	0.0001*
	(0.0001)	(0.0001)	(0.0001)	(0.0001)
Ozone, In Utero	0.002**	0.001***	0.001**	0.002
	(0.001)	(0.0004)	(0.001)	(0.001)
		Panel C: Reduced Form Results		
PM2.5, In HS	0.0001**	0.0001*	0.0001*	0.0001*
	(0.0001)	(0.0001)	(0.0001)	(0.0001)
Nonattainment	−0.010***	−0.010***	−0.010***	−0.010***
	(0.001)	(0.001)	(0.001)	(0.001)
Observations	2,322,823	2,187,117	2,179,973	2,176,255
First Stage F	4.3	6.91	4.01	2.27

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 12 separate regressions (4 in each panel). The outcome variable for each regression is an indicator for whether an individual had not completed High School at ACS survey response. The regressions in Panel A estimate the effect of ozone exposure in utero on High School non-completion using OLS. The regressions in Panel B estimate the effect of ozone exposure on High School non-completion using nonattainment of NO_2 NAAQS standards as an instrument for ozone. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on High School non-completion. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. The estimating sample includes all ACS respondents aged 16–24, who were born between 1987–1997 in a county with at least one active ozone monitor in the year before they were born.

Table 15: The Effect of Ozone Exposure during Infancy on High School Non-completion

	0-12 Mos.	0-3 Mos.	3-6 Mos.	6-9 Mos.
	(1)	(2)	(3)	(4)
Panel A: OLS Results				
PM2.5, In HS	0.00011**	0.00010*	0.00010*	0.00010*
	(0.00006)	(0.00005)	(0.00005)	(0.00005)
Ozone, Infancy	0.00009	0.00002	0.00004***	0.00001
	(0.00006)	(0.00001)	(0.00001)	(0.00001)
Panel B: IV Results				
PM2.5, In HS	0.00011*	0.00010*	0.00010*	0.00010*
	(0.00006)	(0.00006)	(0.00005)	(0.00005)
Ozone, Infancy	0.00116***	0.00134***	0.00121***	0.00096***
	(0.00027)	(0.00045)	(0.00032)	(0.00025)
Panel C: Reduced Form Results				
PM2.5, In HS	0.00011**	0.00010*	0.00010*	0.00010*
	(0.00006)	(0.00005)	(0.00005)	(0.00005)
Nonattainment	−0.01010***	−0.00970***	−0.00974***	−0.00998***
	(0.00131)	(0.00121)	(0.00115)	(0.00114)
Observations	2,298,465	2,192,206	2,175,397	2,165,494
First Stage F	16.73	8.63	14.33	14.12

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 12 separate regressions (4 in each panel). The outcome variable for each regression is an indicator for whether an individual had not completed High School at ACS survey response. The regressions in Panel A estimate the effect of ozone exposure during infancy on High School non-completion using OLS. The regressions in Panel B estimate the effect of ozone exposure on High School non-completion using nonattainment of NO_2 NAAQS standards as an instrument for ozone. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on High School non-completion. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. The estimating sample includes all ACS respondents aged 16–24, who born between 1985–1996 in a county with at least one active ozone monitor in the year they were born.

Table 16: The Effect of Ozone Exposure in utero on College Attendance, by Race and Family Income

	Whites	Non-whites	Black	Hispanics	Bottom 2 quintiles	Top 3 quintiles
	(1)	(2)	(3)	(4)	(5)	(6)
			Panel A: OLS Results			
PM2.5, In HS	−0.0003	−0.0001	−0.0002	−0.0001	−0.0001	−0.0002
	(0.0002)	(0.0002)	(0.0002)	(0.0001)	(0.0001)	(0.0001)
Ozone, In Utero	−0.0003**	−0.00004	0.00002	−0.0002	−0.0001	−0.0002**
	(0.0001)	(0.0001)	(0.0002)	(0.0001)	(0.0001)	(0.0001)
			Panel B: IV Results			
PM2.5, In HS	−0.0003	−0.0001	−0.0002	−0.0001	−0.0001	−0.0002
	(0.0002)	(0.0002)	(0.0002)	(0.0001)	(0.0001)	(0.0002)
Ozone, In Utero	−0.004**	−0.005**	−0.001	−0.005**	−0.003*	−0.005**
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
			Panel C: Reduced Form Results			
PM2.5, In HS	−0.0002	−0.0001	−0.0002	−0.0001	−0.0001	−0.0002
	(0.0002)	(0.0002)	(0.0002)	(0.0001)	(0.0001)	(0.0001)
Nonattainment	0.027***	0.017***	0.007	0.015***	0.015***	0.028***
	(0.005)	(0.005)	(0.010)	(0.004)	(0.003)	(0.006)
Observations	667,271	297,120	131,282	155,283	363,636	600,755
First Stage F	7.01	2.69	6.17	2.66	3.96	4.45

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 18 separate regressions (6 in each panel). The outcome variable for each regression is an indicator for whether an individual was attending college at ACS survey response. The regressions in Panel A estimate the effect of ozone exposure in utero on college attendance using OLS. The regressions in Panel B estimate the effect of ozone exposure on college attendance using nonattainment of NO_2 NAAQS standards as an instrument for ozone. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on wages. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. Each column represents a different subsample based on race (columns 1-4) or family income (columns 5-6).

Table 17: The Effect of Ozone Exposure during infancy on College Attendance, by Race and Family Income

	Whites	Non-whites	Black	Hispanics	Bottom 2 quintiles	Top 3 quintiles
	(1)	(2)	(3)	(4)	(5)	(6)
			Panel A: OLS Results			
PM2.5, In HS	−0.0002	−0.0001	−0.0002	−0.0001	−0.0001	−0.0002
	(0.0002)	(0.0002)	(0.0002)	(0.0001)	(0.0001)	(0.0001)
Ozone, Infancy	−0.0004***	−0.0002	−0.0002	−0.00004	−0.0004**	−0.0002*
	(0.0001)	(0.0002)	(0.0003)	(0.0003)	(0.0002)	(0.0001)
			Panel B: IV Results			
PM2.5, In HS	−0.0002	−0.0001	−0.0002	−0.00005	−0.0001	−0.0002
	(0.0002)	(0.0002)	(0.0002)	(0.0001)	(0.0001)	(0.0001)
Ozone, Infancy	−0.003***	−0.003***	−0.001	−0.002***	−0.002***	−0.003***
	(0.001)	(0.0004)	(0.001)	(0.0005)	(0.001)	(0.001)
			Panel C: Reduced Form Results			
PM2.5, In HS	−0.0002	−0.0001	−0.0002	−0.0001	−0.0001	−0.0002
	(0.0002)	(0.0002)	(0.0002)	(0.0001)	(0.0001)	(0.0001)
Nonattainment	0.027***	0.018***	0.011	0.014***	0.016***	0.028***
	(0.005)	(0.005)	(0.011)	(0.004)	(0.004)	(0.006)
Observations	674,428	300,072	132,800	156,591	368,435	606,065
First Stage F	26.32	18.83	25.58	15.52	24.27	20.36

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 18 separate regressions (6 in each panel). The outcome variable for each regression is an indicator for whether an individual was attending college at ACS survey response. The regressions in Panel A estimate the effect of ozone exposure during infancy on college attendance using OLS. The regressions in Panel B estimate the effect of ozone exposure on college attendance using nonattainment of NO_2 NAAQS standards as an instrument for ozone. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on wages. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. Each column represents a different subsample based on race (columns 1-4) or family income (columns 5-6).

Table 18: The Effect of Ozone Exposure in utero on High School Non-completion, by Race and Family Income

	Whites	Non-whites	Black	Hispanics	Bottom 2 quintiles	Top 3 quintiles
	(1)	(2)	(3)	(4)	(5)	(6)
			Panel A: OLS Results			
PM2.5, In HS	0.0001*	0.0001**	0.0001**	0.0001**	0.0001***	0.00004**
	(0.0001)	(0.0001)	(0.0001)	(0.00004)	(0.00005)	(0.00002)
Ozone, In Utero	0.00001	−0.00004	0.00005	−0.0001	0.00002	−0.00000
	(0.00003)	(0.0001)	(0.0001)	(0.0001)	(0.00004)	(0.00004)
			Panel B: IV Results			
PM2.5, In HS	0.0001*	0.0001**	0.0001**	0.0001**	0.0001***	0.00004**
	(0.0001)	(0.0001)	(0.0001)	(0.00004)	(0.00005)	(0.00002)
Ozone, In Utero	0.0004**	0.002*	0.0001	0.002**	0.002**	0.001***
	(0.0002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.0005)
			Panel C: Reduced Form Results			
PM2.5, In HS	0.0001*	0.0001**	0.0001**	0.0001**	0.0001***	0.00004**
	(0.0001)	(0.0001)	(0.0001)	(0.00004)	(0.00005)	(0.00002)
Nonattainment	−0.003**	−0.010***	−0.0005	−0.009***	−0.010***	−0.008***
	(0.001)	(0.001)	(0.007)	(0.002)	(0.002)	(0.001)
Observations	1,610,426	712,397	313,857	372,307	883,127	1,439,696
First Stage F	6.85	3.1	5.17	3.9	3.94	4.7

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 12 separate regressions (4 in each panel). The outcome variable for each regression is an indicator for whether an individual had not completed High School at ACS survey response. The regressions in Panel A estimate the effect of ozone exposure in utero on High School non-completion using OLS. The regressions in Panel B estimate the effect of ozone exposure on High School non-completion using nonattainment of NO_2 NAAQS standards as an instrument for ozone. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on High School non-completion. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. Each column represents a different subsample based on race (columns 1-4) or family income (columns 5-6).

Table 19: The Effect of Ozone Exposure infant on High School Non-completion, by Race and Family Income

	Whites	Non-whites	Black	Hispanics	Bottom 2 quintiles	Top 3 quintiles
	(1)	(2)	(3)	(4)	(5)	(6)
			Panel A: OLS Results			
PM2.5, In HS	0.0001*	0.0001**	0.0001**	0.0001**	0.0001***	0.00004**
	(0.0001)	(0.0001)	(0.0001)	(0.00004)	(0.00005)	(0.00002)
Ozone, Infancy	0.00004	0.00002	0.00005	−0.0001	0.0001	0.00004
	(0.0001)	(0.0001)	(0.0001)	(0.0001)	(0.0001)	(0.00004)
			Panel B: IV Results			
PM2.5, In HS	0.0001*	0.0001**	0.0001**	0.0001**	0.0001***	0.00004**
	(0.0001)	(0.0001)	(0.0001)	(0.00004)	(0.00005)	(0.00002)
Ozone, Infancy	0.0003***	0.001***	0.0004	0.001***	0.001***	0.001***
	(0.0001)	(0.0003)	(0.001)	(0.0002)	(0.0002)	(0.0002)
			Panel C: Reduced Form Results			
PM2.5, In HS	0.0001*	0.0001**	0.0001**	0.0001**	0.0001***	0.00004**
	(0.0001)	(0.0001)	(0.0001)	(0.00004)	(0.00005)	(0.00002)
Nonattainment	−0.003**	−0.011***	−0.004	−0.008***	−0.010***	−0.008***
	(0.001)	(0.001)	(0.006)	(0.002)	(0.002)	(0.001)
Observations	1,594,751	703,714	311,083	366,648	875,272	1,423,193
First Stage F	21.04	14.76	18.47	14.61	17.69	16.55

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 12 separate regressions (4 in each panel). The outcome variable for each regression is an indicator for whether an individual had not completed High School at ACS survey response. The regressions in Panel A estimate the effect of ozone exposure during infancy on High School non-completion using OLS. The regressions in Panel B estimate the effect of ozone exposure on High School non-completion using nonattainment of NO_2 NAAQS standards as an instrument for ozone. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on High School non-completion. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. Each column represents a different subsample based on race (columns 1-4) or family income (columns 5-6).

Table 20: The Effect of Ozone Exposure in utero on Incarceration, by Race and Family Income

	Whites	Non-whites	Black	Hispanics	Bottom 2 quintiles	Top 3 quintiles
	(1)	(2)	(3)	(4)	(5)	(6)
			Panel A: OLS Results			
PM2.5, In HS	0.00001**	0.00004**	0.00005**	0.00002**	0.00004**	0.00001***
	(0.00001)	(0.00002)	(0.00002)	(0.00001)	(0.00002)	(0.00000)
Ozone, In Utero	0.00001	−0.00000	0.00003	−0.00002	0.00001	0.00001
	(0.00001)	(0.00002)	(0.00003)	(0.00002)	(0.00002)	(0.00001)
			Panel B: IV Results			
PM2.5, In HS	0.00001**	0.00004**	0.0001**	0.00002**	0.00004**	0.00002***
	(0.00001)	(0.00002)	(0.00002)	(0.00001)	(0.00002)	(0.00000)
Ozone, In Utero	0.00001	−0.0002	0.001**	−0.0001	−0.0002**	0.00005**
	(0.00003)	(0.0002)	(0.0003)	(0.0003)	(0.0001)	(0.00002)
			Panel C: Reduced Form Results			
PM2.5, In HS	0.00001**	0.00004**	0.00005**	0.00002**	0.00004**	0.00001***
	(0.00001)	(0.00002)	(0.00002)	(0.00001)	(0.00002)	(0.00000)
Nonattainment	−0.0001	0.001	−0.006***	0.001	0.001**	−0.0003
	(0.0002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.0002)
Observations	1,610,426	712,397	313,857	372,307	883,127	1,439,696
First Stage F	6.85	3.1	5.17	3.9	3.94	4.7

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 18 separate regressions (6 in each panel). The outcome variable for each regression is an indicator for whether an individual was incarcerated at ACS survey response. The regressions in Panel A estimate the effect of ozone exposure in utero on incarceration using OLS. The regressions in Panel B estimate the effect of ozone exposure on incarceration using nonattainment of NO_2 NAAQS standards as an instrument for ozone. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on incarceration. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. Each column represents a different subsample based on race (columns 1-4) or family income (columns 5-6).

Table 21: The Effect of Ozone Exposure infant on Incarceration, by Race and Family Income

	Whites	Non-whites	Black	Hispanics	Bottom 2 quintiles	Top 3 quintiles
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: OLS Results						
PM2.5, In HS	0.00001**	0.00004**	0.00005**	0.00002**	0.00004**	0.00002***
	(0.00001)	(0.00002)	(0.00002)	(0.00001)	(0.00002)	(0.00000)
Ozone, Infancy	0.00000	−0.00004*	0.00000	−0.0001**	−0.00002	0.00000
	(0.00001)	(0.00003)	(0.00005)	(0.00002)	(0.00002)	(0.00001)
Panel B: IV Results						
PM2.5, In HS	0.00001**	0.00004**	0.00005**	0.00002**	0.00004**	0.00002***
	(0.00001)	(0.00002)	(0.00002)	(0.00001)	(0.00002)	(0.00000)
Ozone, Infancy	0.00001	−0.0001	0.001***	−0.0001	−0.0001	0.00004**
	(0.00002)	(0.0001)	(0.0001)	(0.0001)	(0.0001)	(0.00002)
Panel C: Reduced Form Results						
PM2.5, In HS	0.00001**	0.00004**	0.00005**	0.00002**	0.00004**	0.00002***
	(0.00001)	(0.00002)	(0.00002)	(0.00001)	(0.00002)	(0.00000)
Nonattainment	−0.0001	0.001	−0.007***	0.001	0.001	−0.0003*
	(0.0002)	(0.001)	(0.001)	(0.001)	(0.0005)	(0.0002)
Observations	1,594,751	703,714	311,083	366,648	875,272	1,423,193
First Stage F	21.04	14.76	18.47	14.61	17.69	16.55

*p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level

Source: 2000–2014 IRS Form 1040, 2005 through 2015 ACS, EPA monitor data and ACAG satellite data
Note: This table reports the results of 18 separate regressions (6 in each panel). The outcome variable for each regression is an indicator for whether an individual was incarcerated at ACS survey response. The regressions in Panel A estimate the effect of ozone exposure during infancy on incarceration using OLS. The regressions in Panel B estimate the effect of ozone exposure on incarceration using nonattainment of NO_2 NAAQS standards as an instrument for ozone. The regressions in Panel C estimate the reduced form effect of NO_2 nonattainment status designation on incarceration. Kleinbergen-Paap F statistics are presented as a test of the first stage strength of the instrument for the IV regressions in Panel B. All regressions include particulate matter smaller than 2.5 microns (PM2.5) exposure during adolescence as an additional independent variable of interest, which is included in the table. All regressions additionally include county-of-birth, year-of-birth, month-of-birth and year of survey response fixed effects, as well as individual demographic characteristics, and county-of-birth characteristics, as described in the text, which are omitted from the table. Each column represents a different subsample based on race (columns 1-4) or family income (columns 5-6).

B Re-weighted Results

There may be some concern that non-random PIK assignment may lead to bias. This is of particular concern as the results above point towards substantial environmental justice concerns, with disproportionate effects observed for disadvantaged populations (black, Hispanics, and poorer households). As these populations are also less likely to receive a PIK, I present an alternate set of regression results, re-weighted by the inverse probability of receiving a PIK.

To generate the inverse probability weights, I estimate a probit regression of the form:

$$P(PIK|X) = \alpha + \beta X + e$$

Where X is a vector of demographic information, including race, ethnicity, sex, age, state of residence and marital status. I then calculate predicted probabilities from this probit model $\hat{p}$, and assign inverse probability weights $ipw = \frac{1}{\hat{p}}$ to each individual in the estimating sample. Since the previous baseline regressions are estimated using ACS 1-year final sampling weights w_a, I calculate final weights as $w_f = w_a \times ipw$, and estimate a subset of the IV regressions of interest using these final weights.

Table 22 reproduces a subset of the IV college attendance results using these new weights. Columns 1 and 2 present the effect of ozone exposure and TSP exposure respectively, on college attendance, while columns 3 and 4 present the effect of ozone and TSP exposure in the first year of life on college attendance. These re-weighted results are still statistically significant and negative for ozone (although not for TSP), and the point estimates are slightly smaller: a 1 AQI increase in ozone exposure in utero decreases college attendance by 0.22 percent in the re-weighted results, compared to a 0.3 percent decrease in the baseline results in table 12.

Table 23 reports a selection of re-weighted results of the effect of ozone and TSP exposure on High School non-completion, with identical structure to Table 22. Again, the effects continue to be broadly consistent with previous baseline results — ozone and TSP exposure have statistically significant and positive effects on High School non-completion, although the point estimates are slightly smaller. This pattern is broadly true when examining the effect of pollution exposure on incarceration, as shown in Table 24. This table replicates the results for only black individuals (this was the substantively affected sub-population). Again, the re-weighted results are consistent with the baseline results above, suggesting that pollution exposure at birth increases incarceration rates for blacks. The results of these re-weighted models are strong evidence that the baseline results are not driven by bias due to non-random PIK assignment.

Table 22: Effect of Pollution Exposure on College Attendance, IV Models (Reweighted)

	Dependent Variable: College Attendance			
	(1)	(2)	(3)	(4)
PM2.5, in HS	−0.004	−0.004	−0.004	−0.005
	(0.004)	(0.005)	(0.004)	(0.004)
Ozone, in utero	−0.002**			
	(0.001)			
TSP, in utero		−0.001		
		(0.0003)		
Ozone, 0-12 Mo. Old			−0.002**	
			(0.001)	
TSP, 0-12 Mo. Old				−0.001
				(0.0004)
Observations	677,740	751,208	819,331	934,507
Adjusted R^2	0.145	0.148	0.143	0.145

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth birth region by birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

Table 23: Effect of Pollution Exposure on High School Non-completion, IV Models (Reweighted)

	Dependent Variable: High School Non-completion			
	(1)	(2)	(3)	(4)
PM2.5, in HS	0.002*	0.002*	0.002*	0.003**
	(0.001)	(0.001)	(0.001)	(0.001)
Ozone, in utero	0.002**			
	(0.001)			
TSP, in utero		0.001***		
		(0.0003)		
Ozone, 0-12 Mo. Old			0.002**	
			(0.001)	
TSP, 0-12 Mo. Old				0.001***
				(0.0003)
Observations	1,286,272	1,418,139	1,544,039	1,751,397
Adjusted R^2	0.041	0.044	0.042	0.043

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth birth region by birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

Table 24: Effect of Pollution Exposure on Incarceration, IV Models (Reweighted)

	Dependent Variable: Incarceration			
	(1)	(2)	(3)	(4)
PM2.5, in HS	0.001	0.001*	0.001	0.001**
	(0.001)	(0.0005)	(0.001)	(0.0004)
Ozone, in utero	0.002***			
	(0.001)			
TSP, in utero		0.0005***		
		(0.0001)		
Ozone, 0-12 Mo. Old			0.001**	
			(0.001)	
TSP, 0-12 Mo. Old				0.0004***
				(0.0001)
Observations	168,236	187,875	206,517	234,637
Adjusted R^2	0.074	0.078	0.072	0.075

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth birth region by birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

C Labor Market Effects of NO_2 Nonattainment Designations

It is clear that pollution exposure at birth and during later adolescence decreases college attendance rates, increases high school dropout rates, and increases incarceration rates. If pollution exposure has such a profound impact on human capital acquisition, it is important to understand what happens to these individuals left behind: who do not end up enrolling in college. Do they enter the labor force? What wages do they earn? To answer these questions, I consider how pollution exposure at birth and in later adolescence affects several labor market outcomes: labor force participation, employment and wages. For brevity, only the IV models, using NO_2 nonattainment status designations as an instrument are presented in the following tables.[19] These results should be viewed as something of a residual: for the age group of interest, to a first approximation, everyone is in the labor force, in school or in prison. We have seen that pollution exposure reduces college attendance and increases incarceration. The balance of these two effects determines the effect on labor market outcomes.

Table 25 reports results from models of the effect of ozone exposure in utero on labor force participation, stratified by race and class (Table 27 reports largely similar results for ozone exposure during infancy, and Tables 26 and 27 report results for TSP exposure). These results use the full sample of individuals aged 16–24 at time of survey response. Across subgroups, ozone exposure in utero appears to increase labor force participation, although there is some degree of heterogeneity in effect sizes (and precision), especially between income groups. The effect of ozone exposure in utero and during infancy is substantially larger for individuals from poorer families (and the effect for richer individuals is marginally significant or statistically insignificant). For example, a 1 $\mu g/m^3$ increase in TSP exposure during infancy increases the labor force participation rate by 0.3 percent for poorer individuals, compared to a statistically insignificant effect of 0.1 percent for richer individuals. Exposure to PM2.5 during high has no statistically significant effect, for either pollutant and for any subgroup. Our previous results suggest that ozone exposure decreases college attendance, the other major activity individuals would engage in at these ages, and hence a positive effect on labor force participation might be expected on purely compositional grounds.

Labor force participation is defined in the conventional sense: an individual is in the labor market if they are employed or if they are unemployed but actively searching for work. Table 29 goes one step further, examining the effect of ozone exposure in utero on employment, conditional on labor force participation (Table 31 repeats this for ozone exposure during infancy, while Tables 30 and 31 report results for TSP exposure). In general, early life exposure to ozone and TSP appears to increase employment. Again, there

[19] OLS results will be made available in an online appendix.

is heterogeneity by income groups, with larger effects for individuals from poorer families— the effect of a 1 $\mu g/m^3$ increase in TSP exposure during infancy for poorer children (a 0.3 percent increase in employment) is nearly 3 times as large as the effect for richer children. Although the effect is not always statistically significant across models and sub-samples, it appears that exposure to PM2.5 in adolescence has the opposite effect, decreasing employment. This later life effect is largest for non-white children, and implies that a 1 $\mu g/m^3$ increase in PM2.5 exposure reduces employment by 0.1-0.5 percent.

I take this analysis an additional step further, and examine how pollution exposure affects the wages of these individuals, conditional on employment. Tables 33 and 35 report results of the effect of ozone exposure on wages, while Tables 34 and 36 report these results for TSP exposure. The full sample effects are negative, but insignificant for all pollutants and exposure windows. There is an interesting disparity in wage effects across family income groups, however. Pollution exposure (for both pollutants), has a negative and statistically significant effect on wages of richer individuals, but a positive (and insignificant) effect on the wages of poorer individuals. Across subgroups and models, however, pollution exposure during adolescence has a large and statistically significant negative effect on wages, consistent with studies showing a short term effect of pollution exposure on productivity (e.g. Chang et al. (2014)). A 1 $\mu g/m^3$ increase in PM2.5 exposure during adolescence is associated with a decrease in wages ranging from \$92 per year (for whites) to \$105 per year (for non-whites).

Table 25: Effect of Ozone Exposure in utero on Labor Force Participation, IV models, by Race and Income

	Dependent Variable: Labor Force Participation				
	Full Sample	Non-Whites	Whites	Bottom 40%	Top 60%
	(1)	(2)	(3)	(4)	(5)
PM2.5, in HS	0.0003	−0.0002	0.001	−0.001	0.0004
	(0.001)	(0.001)	(0.003)	(0.001)	(0.002)
Ozone, In Utero	0.007***	0.007***	0.008***	0.007***	0.003**
	(0.003)	(0.002)	(0.003)	(0.002)	(0.002)
Observations	1,672,781	536,154	1,136,627	629,491	1,043,290
Adjusted R^2	0.113	0.127	0.111	0.121	0.116

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

Table 26: Effect of TSP Exposure in utero on Labor Force Participation, IV models, by Race and Income

	Dependent Variable: Labor Force Participation				
	Full Sample	Non-Whites	Whites	Bottom 40%	Top 60%
	(1)	(2)	(3)	(4)	(5)
PM2.5, in HS	0.001	0.001	0.002	0.0004	0.001
	(0.002)	(0.001)	(0.003)	(0.002)	(0.002)
TSP, In Utero	0.003***	0.002***	0.004**	0.003***	0.001*
	(0.001)	(0.001)	(0.001)	(0.001)	(0.0004)
Observations	1,794,948	557,099	1,237,849	669,127	1,125,821
Adjusted R^2	0.120	0.131	0.116	0.126	0.118

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

Table 27: Effect of Ozone Exposure during infancy on Labor Force Participation, IV models, by Race and Income

	Dependent Variable: Labor Force Participation				
	Full Sample	Non-Whites	Whites	Bottom 40%	Top 60%
	(1)	(2)	(3)	(4)	(5)
PM2.5, in HS	−0.0004	−0.001	0.0003	−0.002	0.0002
	(0.001)	(0.001)	(0.003)	(0.001)	(0.002)
Ozone, In Utero	0.006***	0.006***	0.006**	0.005***	0.003*
	(0.002)	(0.001)	(0.002)	(0.001)	(0.002)
Observations	2,014,216	644,796	1,369,420	766,395	1,247,821
Adjusted R^2	0.121	0.134	0.118	0.129	0.118

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

Table 28: Effect of TSP Exposure during infancy on Labor Force Participation, IV models, by Race and Income

	Dependent Variable: Labor Force Participation				
	Full Sample	Non-Whites	Whites	Bottom 40%	Top 60%
	(1)	(2)	(3)	(4)	(5)
PM2.5, in HS	0.001	0.001	0.002	0.001	0.001
	(0.002)	(0.001)	(0.003)	(0.001)	(0.002)
TSP, during infancy	0.003***	0.002***	0.003**	0.003***	0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.0005)
Observations	2,192,329	677,263	1,515,066	838,704	1,353,625
Adjusted R^2	0.122	0.133	0.118	0.128	0.118

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

Table 29: Effect of Ozone Exposure in utero on Employment, IV models, by Race and Income

	Dependent Variable: Employment				
	Full Sample	Non-Whites	Whites	Bottom 40%	Top 60%
	(1)	(2)	(3)	(4)	(5)
PM2.5, in HS	-0.002^{*}	-0.003^{*}	-0.002^{*}	-0.002^{*}	-0.001^{*}
	(0.001)	(0.002)	(0.001)	(0.001)	(0.001)
Ozone, In Utero	0.006^{***}	0.005^{**}	0.004^{***}	0.009^{***}	0.001
	(0.002)	(0.002)	(0.001)	(0.003)	(0.001)
Observations	1,003,454	303,477	699,977	348,381	655,073
Adjusted R^2	0.038	0.048	0.022	0.038	0.029

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

Table 30: Effect of TSP Exposure in utero on Employment, IV models, by Race and Income

	Dependent Variable: Employment				
	Full Sample	Non-Whites	Whites	Bottom 40%	Top 60%
	(1)	(2)	(3)	(4)	(5)
PM2.5, in HS	0.001	-0.003^{**}	-0.001	-0.001	-0.001
	(0.002)	(0.002)	(0.001)	(0.001)	(0.001)
TSP, In Utero	0.003^{***}	0.001^{***}	0.002^{*}	0.004^{***}	0.0003
	(0.001)	(0.0004)	(0.001)	(0.001)	(0.0003)
Observations	1,794,948	316,607	771,746	375,821	712,532
Adjusted R^2	0.120	0.052	0.024	0.049	0.029

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

Table 31: Effect of Ozone Exposure during infancy on Employment, IV models, by Race and Income

	Dependent Variable: Employment				
	Full Sample	Non-Whites	Whites	Bottom 40%	Top 60%
	(1)	(2)	(3)	(4)	(5)
PM2.5, in HS	-0.004^{***}	-0.005^{**}	-0.003^{***}	-0.004^{***}	-0.002^{***}
	(0.001)	(0.002)	(0.001)	(0.001)	(0.001)
Ozone, during infancy	0.005^{***}	0.005^{***}	0.004^{**}	0.007^{***}	0.003
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Observations	1,194,274	359,599	834,675	418,571	775,703
Adjusted R^2	0.044	0.053	0.024	0.051	0.029

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

Table 32: Effect of TSP Exposure during infancy on Employment, IV models, by Race and Income

	Dependent Variable: Employment				
	Full Sample	Non-Whites	Whites	Bottom 40%	Top 60%
	(1)	(2)	(3)	(4)	(5)
PM2.5, in HS	0.001	-0.004^{**}	-0.001	-0.002	-0.001
	(0.002)	(0.002)	(0.001)	(0.001)	(0.001)
TSP, during infancy	0.003^{***}	0.001^{***}	0.002^{*}	0.004^{***}	0.001
	(0.001)	(0.0004)	(0.001)	(0.001)	(0.0004)
Observations	2,192,329	379,442	937,154	466,056	850,540
Adjusted R^2	0.122	0.054	0.025	0.052	0.030

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

Table 33: Effect of Ozone Exposure in utero on Wages, IV models, by Race and Income

	Dependent Variable: Wages, 2015 Dollars				
	Full Sample	Non-Whites	Whites	Bottom 40%	Top 60%
	(1)	(2)	(3)	(4)	(5)
PM2.5, in HS	−92.454***	−87.985***	−84.101***	−116.907***	−92.496***
	(15.209)	(29.975)	(21.031)	(20.524)	(23.873)
Ozone, In Utero	−26.231	−10.192	−2.993	43.317	−179.289
	(32.014)	(32.231)	(29.699)	(34.592)	(117.384)
Observations	1,003,454	303,477	699,977	348,381	655,073
Adjusted R^2	0.144	0.123	0.168	0.137	0.138

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

Table 34: Effect of TSP Exposure in utero on Wages, IV models, by Race and Income

	Dependent Variable: Wages, 2015 Dollars				
	Full Sample	Non-Whites	Whites	Bottom 40%	Top 60%
	(1)	(2)	(3)	(4)	(5)
PM2.5, in HS	−96.599***	−93.701***	−86.862***	−117.519***	−95.358***
	(15.330)	(26.916)	(22.062)	(18.556)	(20.485)
TSP, In Utero	−8.559	−0.143	−2.569	18.565	−39.245***
	(8.735)	(8.053)	(12.982)	(11.461)	(9.949)
Observations	1,088,353	316,607	771,746	375,821	712,532
Adjusted R^2	0.142	0.123	0.163	0.134	0.145

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

Table 35: Effect of Ozone Exposure during infancy on Wages, IV models, by Race and Income

	Dependent Variable: Wages, 2015 Dollars				
	Full Sample	Non-Whites	Whites	Bottom 40%	Top 60%
	(1)	(2)	(3)	(4)	(5)
PM2.5, in HS	−102.422***	−105.881***	−92.950***	−127.994***	−81.438***
	(14.242)	(34.744)	(21.029)	(22.277)	(17.678)
Ozone, during infancy	−9.893	23.533	−0.239	41.133	−134.399
	(33.206)	(35.472)	(30.082)	(27.484)	(158.800)
Observations	1,194,274	359,599	834,675	418,571	775,703
Adjusted R^2	0.138	0.111	0.162	0.131	0.137

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data

Table 36: Effect of TSP Exposure during infancy on Wages, IV models, by Race and Income

	Dependent Variable: Wages, 2015 Dollars				
	Full Sample	Non-Whites	Whites	Bottom 40%	Top 60%
	(1)	(2)	(3)	(4)	(5)
PM2.5, in HS	−107.599***	−100.024***	−101.037***	−117.919***	−107.555***
	(16.442)	(32.431)	(20.492)	(21.955)	(19.296)
TSP, during infancy	−6.195	5.985	−3.708	16.438	−25.958**
	(10.452)	(9.633)	(14.644)	(10.056)	(12.093)
Observations	1,316,596	379,442	937,154	466,056	850,540
Adjusted R^2	0.140	0.112	0.160	0.133	0.142

Note: *p<0.1; **p<0.05; ***p<0.01
All models include birth year, birth month and birth county FE
Standard errors are clustered at the birth state level
See Table 1 for more details

Source: 2000–2014 IRS Form 1040, 2005–2014 ACS, EPA monitor data and ACAG satellite data