Childhood Exposure to Polluted Neighborhood Environments and Intergenerational Income Mobility, Teenage Birth, and Incarceration in the U.S.

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Abstract

This paper joins a growing body of research linking measures of the physical environment to population well-being, with a focus on neighborhood toxins. Extending a national database on the social mobility of American children growing up in over 70,000 Census tracts, we explore the association between childhood exposure to two forms of pollutants and three socioeconomic outcomes for African Americans, whites, and Latinos. We find that children who grew up in Census tracts with higher levels of traffic-related air pollution and housing-derived lead risk experienced lower adult incomes on average relative to their parents and higher likelihoods of being incarcerated as an adult or having children as teenagers, after controlling for standard socio-demographic characteristics and metropolitan-level effects. The spatial distribution of these two pollutants is surprisingly different, however, with air pollution varying mostly between regions of the country while lead risk varies dramatically between neighborhoods within the same city. Yet each pollutant predicts the three aspects of social mobility similarly, and we show important disparities in exposure by race. Differential exposure to environmental toxins in childhood may be a contributor to racial inequality in socioeconomic outcomes among adults.

In her Presidential address to the Population Association of America, Anne Pebley (1998, p. 379) raised a provocative question: "Why have environmental issues—other than natural resources—*not* been a more central focus of demographic research"? Several possibilities were offered, from theoretical neglect to lack of available data, but Pebley's main conclusion was that "Whatever the reason, environmental issues have been peripheral to the main areas of demographic research" (1998, p. 380).

In a later presidential address, Barbara Entwisle made a similar argument and put more specificity on the lack of environmental research, especially in the study of neighborhood effects. As she stated, "the explosion of research on neighborhoods and dimensions of population well-being has not produced equivalent improvements in the conceptualization and measurement of neighborhoods and their effects" (Entwisle, 2007, p. 691). To document her claim of relative neglect, Entwisle coded the abstracts of over 500 studies in population studies and discovered that exposure to environmental contaminants related to manufacturing and mining, air and water pollution, and noise—what she labeled "environmental toxins"—was, rather remarkably, "the least examined neighborhood attribute" (Entwisle, 2007, p. 693). By far the most commonly studied feature was neighborhood poverty, even though toxins and hazards are concentrated in poor neighborhoods. More recent assessments have confirmed that research on childhood neighborhood effects on life outcomes focuses primarily on the social environment (Sharkey and Faber 2014). Entwisle hypothesized that what appear to be the consequences of poverty may in fact be the consequences of air pollution or some other physical environmental hazard.

Entwisle's hypothesis is important, because while environmental toxins often coincide with neighborhood poverty and racial segregation (Bell & Ebisu, 2012; Sampson & Winter, 2016; Vivier et al., 2011), the channels through which they operate may be distinct. Most studies

of concentrated poverty theorize its effects as operating solely through social pathways, such as social isolation and lack of access to economic and educational resources (Galster, 2012). Toxins, on the other hand, are typically thought to operate in large part by undermining physical health, mental health, and cognitive functioning, which in turn influence social outcomes (Muller et al., 2018). Of course, concentrated poverty and racial segregation can also indirectly work their way into the minds and bodies of children by exposing residents to neighborhood toxins and high levels of stress. Either way, however, the pathway from exposure to toxins to life experiences is distinct from those of poverty and other traditional neighborhood characteristics.

This paper assesses and extends this general hypothesis by examining neighborhood level associations of exposure to environmental hazards among children and outcomes in late adolescence and adulthood. We build on a growing body of work that has prioritized the physical environment and the dangers of exposure to multiple forms of pollutants and toxins. In the social sciences, this work includes studies of the production of toxic sites, many of which are unseen to the public (Elliott & Frickel, 2013; Frickel & Elliott, 2018), the sources and consequences of lead exposure (Aizer & Currie, 2019; Aizer, Currie, Simon, & Vivier, 2018; Muller, Sampson, & Winter, 2018; Sampson & Winter, 2016, 2018), and the measurement and long-term consequences of air pollution for health and cognitive functioning (Alotaibia et al., 2019; Alshire, Karraker, & Clarke, 2017; Isen, Rossin-Slater, & Walker, 2017; N. Kravitz-Wirtz, K. Crowder, A. Hajat, & V. B. Sass, 2016; Sass et al., 2017; Zivin & Neidell, 2018). Our intervention in this paper is to examine the national-level relationship between neighborhood exposure to two types of environmental degradation in childhood—*air pollution* and *lead exposure*—and three outcomes later in life that are central to health and demographic processes—*income mobility*, *teenage childbearing*, and *incarceration*. Unlike previous research,

we provide disaggregated estimates for all three of the largest race/ethnic groups in the U.S.—African Americans, European-Americans or whites, and Latino-Americans—and we assess the predictive power of both environmental toxins independent of factors commonly studied in the neighborhood effects literature, including poverty and race.

To accomplish our goals, we combine newly available data derived from IRS tax records at the Census tract level housed at the Opportunity Atlas (Chetty, Friedman, Hendren, Jones, & Porter, 2018) with estimates of exposure to air pollution from vehicle traffic and the risk of lead exposure due to older housing in poor neighborhoods. Based on a national sample of over 70,000 Census tracts and adjusting for metropolitan area fixed effects, we find that although the risks of exposure to lead and air pollution are not strongly correlated with each other, childhood exposure to each is independently predictive of adverse outcomes in terms of adult income level relative to parents (income mobility), the likelihood of teen motherhood, and the likelihood of incarceration as an adult. Surprisingly, the direction and significance of these effects is broadly similar for whites and African Americans, and to a lesser extent Latinos. Exposure levels differ by race and poverty, as Entwisle (2007) suggested, and by region of the country. But the independent predictive power of these pollutants is largely consistent.

Overall, we show the importance of measuring different environmental toxins simultaneously and exploring their independent long-term associations with well-being for different race/ethnicities. Based on these results, we discuss fruitful lines of future inquiry to extend the scope of measurement and probe the causal mechanisms by which environmental toxins influence developmental processes over the life course.

Research Framework and Strategy

Our theoretical motivation is to measure and assess the life-course disparities associated with growing up in a polluted neighborhood environment, especially the kind of polluted environment that gets into the minds and bodies of children. There are many different toxic environments, of course, including those deriving from social interactions such as violence. Prior research has established plausible causal effects of exposure to violence on the blunting of cognitive development and a diverse set of behavioral problems among children and adolescents (McCoy, Raver, & Sharkey, 2015; Sharkey, 2010; Sharkey, Tirado-Strayer, Papachristos, & Raver, 2012).

We focus in this paper on direct environmental toxins, the first being air pollution. Previous studies have established that exposure to air pollution is associated with compromised physical health, mental health, and cognitive functioning among adults (Alotaibi et al. 2019; Sass et al. 2017; Qiu, Yang, & Lai, 2019) and children (Khreis et al., 2017; Margolis et al., 2016; Newman et al., 2013; Perera et al., 2013; Roberts et al., 2019; Shier et al., 2019; Volk et al., 2013). The diminution of health and cognitive functioning is theoretically a mechanism that undermines later education and labor market success (Zivin & Neidell, 2018). Air pollution has also been directly tied to school absences (Currie, Hanushek, Kahn, Neidell, & Rivkin, 2009; Mohai, Kweon, Lee, & Ard, 2011), which may lead to general detachment from social institutions and therefore behavioral problems. Either direct diminution of cognitive functioning or detachment from social institutions could over time lead to lower adult incomes, greater likelihood of incarceration, and greater likelihood of teen motherhood. Lead exposure has also been linked to later outcomes as diverse as lower test scores, suspension from school, teenage pregnancy, impulsivity, delinquency, and in one study, lower adult socioeconomic status (Aizer et al., 2018; Aizer & Currie, 2019; Reuben et al., 2019; Winter & Sampson, 2017). Again, this

suggests a pathway from toxic exposure in childhood, through the resulting impairments in cognitive functioning and impulse control, to well-known snares in the life course that impede adult development. Previous research has also shown that racial minorities are disproportionately exposed to many environmental toxins (N. Kravitz-Wirtz, K. Crowder, A. Hajat, & V. Sass, 2016; Morello-Frosch, Pastor, & Sadd, 2001; Muller et al., 2018).

Studies seeking to establish explicit causality from toxin exposures in childhood to economic and social outcomes much later in life have used a mix of instrumental variables, such as the distance to long defunct and demolished lead smelting plants (Sampson & Winter, 2018) or cities' use of lead rather than iron water service pipes in the early 20th Century (Feigenbaum & Muller, 2016), and natural quasi-experiments, such as the introduction of lead remediation programs (Aizer et al., 2018) and the passage of the Clean Air Act (Isen et al., 2017).

Manduca and Sampson (2019) built on this literature to assess neighborhood exposure to environmental toxins that pose direct physical and mental harms to children's development and thereby impede social mobility over the long-term. Using data from Chetty et al. (2018) in Chicago, they showed, among other findings, that growing up in neighborhoods with high levels of lead exposure predicted lower intergenerational income mobility and higher adult incarceration for poor black males, after accounting for commonly studied factors such as concentrated poverty and racial composition. Similar patterns emerged for the income mobility of poor white children and teenage birth among both poor black and poor white girls. In Chicago, moreover, toxic environments, which included violence, were racially segregated, with little overlap in exposure rates for blacks and whites: virtually all majority black Census tracts were more exposed to hazards than any majority white tracts. Under the assumptions of their model,

Manduca and Sampson (2019) concluded that this large difference in exposure rates could plausibly account for 20-60% of citywide racial disparities in intergenerational inequality.

There are several limitations to Manduca and Sampson (2019) that we address here. Their study was limited to Chicago, restricted to a comparison of blacks and whites, included only males for the analysis of income mobility, and did not examine exposure to pollutants other than lead. It is thus unknown how generalizable the results are to other cities and neighborhoods around the country, and whether the patterns apply to other environmental toxins or to Latino Americans, who historically have lived in environments that are often distinct from the urban "inner-city" settings typical of the Northeast and Midwest that feature in traditional neighborhood studies. Consequently, we examine neighborhoods across the United States to address these limitations, testing the hypothesis that childhood exposure to neighborhood lead and air pollution is directly associated with adult income mobility, incarceration, and teen birth.

Data

To assess our research questions, we merge Census tract-level estimates of child mobility created from linked income tax and Census records with measures of ambient pollution and the risk of lead exposure. We use data on social outcomes from the Opportunity Atlas (Chetty et al., 2018), constructed from linked IRS, Decennial Census, and American Community Survey data. This dataset covers approximately 96% of the 1978-1983 birth cohorts, with children matched to parents based on who claims them as a dependent in tax returns. The Census tract is our unit of analysis. Estimates are constructed by averaging the adult outcomes for all children who grew up in a given tract. In cases where children moved during childhood, they are assigned to each tract they lived in before age 23, weighted by the number of years they lived in that tract. In total, our

analyses include data for 74,148 Census tracts, although the sample for any particular analysis to follow may be smaller. In some analyses, we account for geographic variation by including fixed effects for Commuting Zones. Commuting Zones are groups of counties linked by commuter flows and are a common way of operationalizing metropolitan areas (Tolbert & Sizer, 1996). Following Manduca and Sampson (2019), we estimate results separately by race but in the present analysis we include Latinos as well as African Americans and whites. Our analysis focuses specifically on the expected outcomes for children whose parents had incomes at the 25th percentile of the national income distribution. The main outcomes we examine are: individual *income rank* in 2014-2015, when children in our sample were ages 31-37; the expected fraction of boys *incarcerated* on April 1, 2010, when they were ages 27-32;[1] and the expected fraction of girls who had *children as teenagers*.

We combine the Opportunity Atlas data with control variables at the Census tract level from the Census and Opportunity Atlas. We measure the share of the population that is African American and the share that is foreign-born; the fraction of adults who have a bachelor's degree; the tract poverty rate; the percentage of working residents who are in manufacturing industries; the share of housing units built before 1940; and the fraction of workers who commute less than 15 minutes. We also measure the tract population density. These variables span the essential variation of a wider range of covariates, including the mean household income, the share of the population that is Latino or Asian, and the share of children living with single parents. In a robustness check reported later, we also include these additional variables using a principal

[1] Although the average age at first arrest is in the teens, the average age at first admission to prison is in the mid-twenties and has been getting older. National data from around the time of our measurement, in 2013, also show that 78% or more of prison admissions in 2013 were among those age 25 or older (Carson & Sabol, 2016). There may be some small proportion of boys in in our sample who were previously incarcerated *and* released before April 1, 2010, who would not be measured as "incarcerated" in our data.

components procedure and repeat the analysis.

We use Census variables interpolated to 1995, when the birth cohorts in our sample were 12-17, for the main measures of neighborhood social composition, such as racial demographics and education levels. This timing aligns neighborhood measurements with the period when the cohorts in our sample were in adolescence. For example, the youngest cohort in our data grew from age 12 to 17 from 1995 to 2000, while the oldest grew from age 17 to 22. Recent research on these social mobility data shows that the effects of neighborhood context are somewhat stronger in adolescence than in early childhood (Chetty et al., 2018). The more environmental measures like population density, home age, manufacturing, and commuting times are all measured in 2000, as are our two toxins. Like Chetty and Hendren (2018), we assume that relative differences between neighborhoods in our measured characteristics are reasonably stable over the period of measurement.

In addition to the control variables, our two explanatory variables of greatest interest are measures of exposure to traffic-related air pollution and housing lead. Neither of these variables is directly measured at the scale and resolution necessary for our analysis, so we use estimates constructed by other researchers. For air pollution, we use Census block-level estimates of particulate matter—both PM 10 and PM 2.5—NO2, and CO in the year 2000, constructed by Alotaibi et al. (2019), aggregated to 2010 Census tracts. Estimates of particulate matter are constructed using a universal kriging framework to interpolate between regulatory monitoring points, while estimates of NO2 and CO are constructed using land use regression, as described in Alotaibi et al. (2019). Although these four types of pollution are all emitted from vehicles and are often grouped in studies of "traffic-related air pollution," (e.g. Health Effects Institute, 2010; Khreis et al., 2017; Volk et al., 2013), it is important to note that they can originate from

different sources—particulate matter especially is produced from a variety of sources including power plants, industries, and automobiles (Mukherjee & Agrawal, 2017)—and can have different spatial distributions, especially at small scales. Nonetheless, they are strongly correlated in our tract-level data (all pairwise correlations are above 0.65 except for those of PM 2.5 with NO2 and CO), and they load onto one factor in a principal components analysis. We use this single factor of "traffic-related air pollution" in our main analysis, but also report results for a separate analysis where we divide the four pollutants into two factors. One factor is created combining PM 2.5 and PM 10, which are produced from multiple sources and tend to vary at larger spatial scales, while a second factor is created for CO and NO2, which are predominantly associated with traffic pollution and tend to vary at small spatial scales.

Direct measurement of lead exposure is likewise not available at the national scale, so we use a proxy measure of lead exposure *risk* developed by the Washington State Department of Health (2015), which is based on a combination of Census tract poverty rate and housing age. A main source of lead exposure in many cities is paint from housing units built in the first half of the 20th Century that have not been remediated (Jacobs et al., 2002). The Washington researchers determined that a weighted average of these two Census variables best tracks the spatial distribution of lead exposure in Washington State. We apply the coefficients from their analysis to the housing age and poverty rate breakdown in the 2000 Census.[2] While this is an

[2] The Washington State measure estimates the fraction of houses with lead risk due to age as 0% of the houses built after 1980, 8% of the houses built from 1960-1979, 43% of the houses built from 1940-1959, and 68% of the houses built before 1940, based on the findings of Jacobs et al. (2002). Applying those percentages to the housing age breakdown in the 2000 Census gives an estimate of the fraction of houses in each tract at risk of lead exposure due to housing age. To create the final score of lead risk from both housing age and poverty, the Washington State measure normalizes both the poverty rate and housing age risk rates so that each variable has mean 0 and standard deviation 1, then averages the two normalized measures, weighting the poverty measure at 42% and the age risk measure at 58%. These weights capture the relative size of the difference in mean Blood Lead Levels from each category (see Washington State Department of Health 2015, p 30-31).

imperfect measure, neglecting risk of lead exposure from water in particular, we believe that it is a reasonable approximation given the limited data at the national scale. To validate our measure, we compared these estimates to a direct measure of lead exposure obtained from blood-lead levels in children at the tract level from the city of Chicago in the mid-1990s (Sampson & Winter, 2016). The Washington State-derived procedure for measuring lead risk correlated with actual blood lead concentrations in Chicago at 0.70, while actual lead concentration was correlated with the poverty rate alone at 0.65 and with the housing age risk score alone at 0.25. These patterns support the construct validity of the lead risk measure.

Both air pollution and lead exposure risk are measured in 2000, when children in our sample were 17-22 years old. They are thus measures of exposure in late childhood and adolescence rather than early childhood. We believe that they are nonetheless informative, both as a proxy for exposure levels earlier in childhood and because exposure effects are likely to be cumulative. To the extent that effects continue into adulthood, as previous literature suggests they might, the measures capture this as well.

Perhaps surprisingly, traffic-related air pollution and lead risk have relatively limited spatial overlap. The overall tract-level correlation between the two is just 0.21, suggesting that these are two distinct forms of environmental exposure. To explore this divergence further, Figures 1 and 2 map the spatial profiles of each pollutant at the national and metropolitan scales. Figure 1A maps levels of traffic-related air pollution by county for the country as a whole. Average traffic-related air pollution levels vary dramatically across regions of the country, and are highest in the Southwest, most notably in Southern California and Arizona. The median child in the Los Angeles Commuting Zone lived in a tract with a traffic pollution score of 2.57, at the 96th percentile of children nationwide. Lead risk, in contrast, is more evenly distributed across

the country: no Commuting Zone has an overall median exposure above 1.28, the 89th percentile for the nation as a whole (both pollution measures are in units of national standard deviations). Further, while traffic pollution is concentrated most heavily in large cities, many rural areas—most notably in the great plains and the northeast—have elevated risk of housing lead exposure, at least by this measurement, a pattern likely a function of the older housing there compared to Southwest.

The spatial distributions of the two pollutants within metropolitan areas are again quite different. Figure 2 maps traffic pollution and housing lead risk by Census tract within the Houston Commuting Zone, one of the nation's largest and most diverse metropolitan areas. At the tract level, there is much more spatial variation in lead risk than in air pollution. While air pollution is generally higher in the city than in the outlying rural areas, levels are fairly similar within the dense parts of the urban area. In contrast, Houston contains neighborhoods with both very high and very low levels of lead risk, even in close physical proximity. While the median child in the Houston Commuting Zone lived in a tract with a lead risk score of -0.72, almost one standard deviation below the national average, more than 40,000 children were in tracts with scores above 1.5. The median child's exposure to traffic pollution was much higher, at 0.27, but no children lived in tracts with traffic pollution levels above 1.11. On the whole, then, traffic pollution varies a great deal regionally but comparatively little at the tract scale, while lead risk has substantial tract-level variation despite having less regional variation. These different spatial distributions have important but largely unstudied implications, especially for the demographics of who is exposed to which pollutants.

Methods

Our primary analysis consists of descriptive regressions at the Census tract level in which we estimate the association between adolescent air pollution and lead risk levels and adult income rank, likelihood of incarceration, and likelihood of teen motherhood, net of standard sociodemographic control measures. We follow Manduca and Sampson (2019) in including percent black, percent foreign born, poverty rate, percent with a college degree, and percent of employed residents commuting less than 15 minutes to work as our main predictors. Based on theoretical considerations with respect to pollution exposure, we also include population density, percent of employed residents working in manufacturing, and percent of housing units built before 1940. The tract poverty rate and the percentage of housing units built before 1940 are the two main components of our lead risk score, so we include them separately to determine if the weighted combination used for the lead risk score contributes additional explanatory power. In a robustness check, we omit these two control variables and just include the lead risk score.

In our primary, national-level analysis, we include Commuting Zone-level fixed effects to control for city-level variation in upward mobility and other outcomes, which is known to be substantial (Chetty and Hendren 2016; Chetty et al. 2014) but is unlikely to be strongly related to the mechanisms that we are interested in here. Although we do not attempt to establish a causal relationship between our explanatory and outcome variables, we are motivated by previous research that does suggest a causal link from exposure to lead and particulate pollution to behaviors and outcomes associated with lower mobility (Aizer & Currie, 2019; Isen et al., 2017; Sampson & Winter, 2018). Our measures could thus reasonably be interpreted as upper bounds on the magnitude of this relationship, although with measurement error they might be underestimated. Further, even though we do not assert that our estimates are causal, measures of

airborne pollution are arguably subject to less selection bias, conditional on covariates, than housing-based lead exposure or traditional neighborhood variables (such as neighborhood poverty or racial composition), in large part because particulate air pollution is often invisible to the naked eye and thus may be less noticeable to potential residents than degraded housing structures, in turn making it less likely to operate as a selection factor in deciding where to live. Even lead exposure in older homes may be hidden due to the common practice of landlords painting over lead-based paint rather than removing it first (Muller et al., 2018).

All this said, we are sensitive to the possibility that the relationships we observe might result from mechanisms other than those we are most interested in. The most relevant alternative mechanism, in our opinion, is the possibility of explicit residential sorting or avoidance behavior by those who are able to choose to live in neighborhoods with lower pollution exposure or environmental degradation (Zivin & Neidell, 2018). This possibility is potentially problematic for our estimates if the relationships we observe are induced by selection in terms of who lives in more versus less polluted neighborhoods. Proximity to highways may also be used to infer traffic-related air pollution, which may lead home buyers or real estate agents to reflect pollution in housing costs. To determine whether this may be driving our results, we conduct a supporting analysis of environmental toxins and neighborhood selection the Los Angeles.

In addition to examining the relationship between exposure to environment toxins and social outcomes, we also explore variation in levels of exposure by race, and the geographic scale at which this variation occurs. We conduct counterfactual exercises in which we adjust population weights to simulate what exposure to pollutants would be like if Latinos were distributed across Commuting Zones, or African Americans were distributed across Census tracts within Commuting Zones, like whites were.

Findings

Table 1, Panel A presents standardized coefficients from our primary regressions estimating the relationship between outcomes of interest and exposure to pollution for black, white, and Latino children. For black children, all three outcomes have strong and statistically significant associations with both traffic-related air pollution and lead exposure risk. Among black children, a one standard deviation increase in lead exposure was associated with a 0.19 standard deviation decrease in income rank—about two-thirds of a percentile rank nationally, compared to a mean expected income rank among black children in our sample at the 34th percentile of the country. A one standard deviation increase in traffic pollution was associated with a 0.11 standard deviation decline in income rank, about half a percentile rank. For incarceration and teen motherhood, the coefficient signs are flipped, as expected, indicating that more pollution exposure was associated with significantly higher likelihoods of the life-course challenges of being incarcerated as an adult or having children while a teenager. A one standard deviation increase in air pollution was associated with a 0.78 percentage point increase in the likelihood of incarceration and a 1.43 percentage point increase in the likelihood of teen motherhood, while a one standard deviation increase in lead risk was associated with 0.60 percentage point increase in incarceration and a 0.74 percentage point increase in likelihood of teen motherhood. In our overall sample, black boys with parents at the 25th percentile of income on average had a 11% chance of being incarcerated and black girls had a 42% chance of becoming teen mothers.

For white children both pollution variables are significantly associated with lower incomes and higher probabilities of incarceration and teen motherhood, but the coefficients are smaller in magnitude, between 0.03 and 0.07 in most cases, although the coefficient of lead risk on income is -0.14, almost as large as the coefficient for black children. This indicates that an

additional standard deviation of air pollution was associated with a 0.4 percentile decline in adult income rank, a 0.14 percentage point increase in the likelihood of incarceration, and a 0.86 percentage point increase in the likelihood of teen motherhood for white children in our sample. An additional standard deviation of lead risk was associated with a 0.99 percentile decrease in income rank, a 0.27 percentage point increase in incarceration likelihood, and a 0.45 percentage point increase in likelihood of teen motherhood. The mean predicted income rank for white children in our sample was at the 46th percentile nationally, while the mean likelihood of incarceration was 2.9% and that of teen motherhood was 20%. Thus, even though white children are on average exposed to much lower levels of pollution than black children (see below), the consequences of such exposure appear to be similar in direction, if smaller in magnitude, to those for African Americans.

The findings hold to a lesser extent for Latinos. The coefficient of lead exposure risk on income for Latinos is negative and significant, though smaller in magnitude than that for whites or blacks. The coefficient of lead risk on incarceration is likewise positive and significant, though smaller in magnitude than that for whites. Interestingly, the coefficient of lead on teen motherhood is negative, possibly indicating a protective effect or an unobserved confounding factor. In concrete terms, one additional standard deviation of lead risk was associated with a 0.3 percentile decrease in expected income rank, a 0.14 percentage point increase in likelihood of incarceration, and a 0.43 percentage point decrease in likelihood of teenage pregnancy, compared to sample-wide predicted means of the 44th percentile of family income, a 3.1% chance of incarceration, and a 31.6% chance of teen motherhood.

The association between traffic pollution and our outcomes of interest for Latinos is less expected. For income, the coefficient is small in magnitude and only marginally significant,

while for incarceration and teen motherhood it is negative—indicating a protective effect—and significant. A one standard deviation increase in exposure to traffic pollution is associated with a 0.16 percentage point decrease in incarceration and a 0.90 percentage point decrease in teen motherhood. We believe that these results may be due in part to the spatial clustering of Latinos in regions of the United States that have high average levels of traffic pollution. Supporting this interpretation, if we run our analysis omitting the Commuting Zone fixed effects, the coefficient of traffic pollution on income mobility for Latinos is -0.09, comparable to those for African Americans and whites, and highly statistically significant. This implies that the concentration of Latinos in Commuting Zones with high average levels of air pollution may be hiding the relationship between air pollution and income mobility among Latinos. The coefficients for incarceration and teen motherhood become smaller in magnitude, though they are still negative and statistically significant. The coefficients on traffic pollution for whites and African Americans are not sensitive to the inclusion or exclusion of Commuting Zone fixed effects. Regional variation by race/ethnicity thus plausibly explains some, although not all, of the divergent results for Latinos.

Robustness Checks and Supporting Analysis

We conducted three sets of analysis to address (1) the sensitivity of results to specification of the pollution and lead risk measures, (2) the specification of control variables, and (3) concerns about residential sorting or selection based on our environmental toxin measures.

Table 1, Panel B reports results from our sensitivity analysis of pollution and lead risk. Specification B1, presented in the first row, shows the coefficients on lead risk in an analysis that omits the tract poverty rate and the fraction of housing units built before 1940, the two

components of the lead risk score. This specification is aimed at addressing concerns about collinearity among these three variables. Consistent with the presence of collinearity, when the poverty rate and the fraction of houses built prior to 1940 are excluded, the coefficients on lead risk are much larger in magnitude.

The next two rows of Table 1B present coefficients from Specification B2, in which we split our primary air pollution variable into two factors: one combining PM2.5 and PM10, and a second combining CO and NO2. These groupings conform to the strength of correlations among the four variables (though overall correlations are high, as discussed above). As Table 1B shows, when two factors are used it is the CO/NO2 factor that more strongly tracks the overall result. The coefficients on the PM factor are statistically significant and generally of the opposite sign as those on the CO/NO2 factor, as might be expected given the high collinearity between the two factors.

Our second robustness check concerned the use of control variables. To limit collinearity, in our primary specification we include eight control variables that are not strongly correlated with one another (maximum pairwise correlation = 0.49). As an alternative, we ran a principal components analysis on a larger set of Census variables, including the share of tract population that is Asian, Latino, and under 18, the tract unemployment rate, the percentage of tract residents who are homeowners; the median length of home tenure; the mean household income; the percentage who are married; the percentage of tract children who have single parents; and the racial diversity of the tract population as measured using the Theil index; in addition to the variables in our primary specification. This resulted in five factors. Results (not shown) are similar to our main specification.

Another potential threat to our analysis is selection. We conducted a sub-analysis to

directly estimate the extent to which particulate air pollution factors into residential mobility decisions in Los Angeles County—one of nation's largest with a population of approximately ten million. Air pollution is highly salient in Los Angeles (see Figure 1) and often the subject of local attention. Further, while the average level of traffic pollution in LA is extremely high, at 2.57, there is still wide variation across the city. For instance, roughly 22% of children in our LA sample grew up in tracts with traffic pollution scores below 1.5, while 20% grew up in neighborhoods with scores above 3.5. If selection by neighborhood on air pollution occurs, then, we should see it in Los Angeles.

Drawing on the data and analysis of Schachner and Sampson (2020), we estimated discrete choice models of neighborhood selection using data from three waves of the Los Angeles Family and Neighborhood Survey from 2001-2012 merged with the pollution measures we study. In models broadly aligned with the specification in Schachner and Sampson (2020: Table 5, Model 4), the coefficient on air pollution is not statistically significant, nor is its interaction with individual race and household income.[3] Residential sorting appears driven instead by neighborhoods' housing market conditions, socio-demographics, and public schools' test scores. This result gives us more confidence that our estimates are not purely a result of residential selection on traffic pollution. There is some evidence of sorting based on our measure of lead risk, which is expected given that it is constructed from neighborhood poverty and housing age, both of which are highly salient to potential movers. Further research should probe the relationship between residential section and actual lead exposure rather than our proxy.

[3] Based on the present article, our models slightly diverge from those of Schachner and Sampson (2020) beyond adding the tract-level toxin measures. We use tracts' family poverty rate and % bachelor's degree to proxy neighborhood socioeconomics rather than their tract status index, which averages the z-scores of median family income (logged) and % bachelor's degree. We also added a proxy for housing stock age and excluded the parental cognitive skills measure employed in that article's models.

Geographic Scale and Racial/Ethnic Disparities in Exposure to Pollution

Having established a clear pattern of results that are reasonably robust, we now examine whether exposure to pollutants varies by race and ethnicity. Here too the pattern is clear. The median Latino child in our sample lived in a tract with a traffic-related air pollution score of 0.46, at the 77th percentile of all children, while the median white child lived in a tract with a traffic pollution score of -0.39, at the 39th percentile. This translates to a gap of 0.85 standard deviations between the median white and Latino children, with the median black child falling in the middle, in a tract with a traffic pollution score of -0.03, at the 59th percentile. The gap between African American and white children in lead exposure is similar to that between Latinos and whites for traffic pollution: the median black child in our sample lived in a tract with a lead risk score of 0.48, at the 73rd percentile of all children, while the median white child lived in a tract with a score of 0.40, near the 42nd percentile—0.88 standard deviations lower. The median Latino child was at the 65th percentile, in a tract with a lead risk score of 0.21.

Although the difference in lead risk exposure between black and white children is similar in magnitude to that in traffic-related air pollution between whites and Latinos, the structure of this difference is quite different. Latinos are disproportionately exposed to traffic pollution because of the broad regions of the country where they are concentrated. African Americans, on the other hand, disproportionately bear lead risk because of the neighborhoods within cities where they live.

The geographic distribution of the Latino population in the United States, particularly for the cohorts in our sample who grew up in the 1990s, overlapped substantially with regional patterns of traffic pollution, most notably in Southern California. Within any given metro area, Latino children were still more exposed to traffic pollution than whites, but not by nearly as

much. This is shown in Figure 3A. The top panel shows a histogram of observed Latino exposure to pollution in our sample. In addition to the overall median of 0.46, there is a very long right tail, with substantial numbers of children living in tracts with pollution levels 2 standard deviations above the national average.

The second panel of Figure 3A shows what exposure to pollution among Latino children would have looked like if they were distributed across Commuting Zones in the same way as white children, holding their distribution within metropolitan areas constant. This counterfactual simulation is constructed by multiplying each tract's population of Latino children by the ratio of the fraction of all white children who lived in that Commuting Zone divided by the fraction of all Latino children who lived there. This will preserve any difference in exposure that is due to the within-metropolitan differences in where whites and Latinos live, while eliminating the difference that is due to the region of the country.

Accounting for Commuting Zone-level variation closes more than 70% of the gap in traffic pollution exposure between whites and Latinos. If Latino children had lived in the same Commuting Zones as white children did, the median Latino would have lived in a tract with a traffic pollution score of -0.16. Comparing this simulation in the second panel of Figure 3A to the observed distributions for Latinos and whites in the top and bottom panels respectively, it is especially striking how the long right tail largely disappears once the cross-regional distribution is held constant. The third panel of Figure 3A shows the opposite simulation, in which the distribution of Latinos within metro areas is altered to match that of whites, but the distribution across Commuting Zones is kept as in reality. This has much less of an effect: the overall median would drop only to 0.14, closing just 37% of the gap with whites, and the long right tail would stay largely present.

Figure 3B shows the results of a similar exercise comparing lead exposure for black and white children in our sample. Here, regional differences explain hardly any of the gap: in the simulation where the African American population is scaled to match that of whites at the Commuting Zone level, shown in the second panel, the median exposure is still 0.44, closing a mere 4% of the gap with whites. The histograms appear almost identical. In contrast, in the simulation where the within-Commuting Zone distribution is altered to match that of whites while the cross-Commuting Zone distribution is held constant, shown in the third panel, the overall exposure levels would be even lower than those observed for whites in reality: the median child would have lived in a tract with a lead risk score of -0.55, 0.16 standard deviations lower than the median white child in reality.

Together, these simulations highlight the very different spatial structures that underly the similarly sized gaps in pollution exposure between Latinos and whites in traffic pollution and African Americans and whites in lead risk. These different scales suggest different approaches may be required to reduce the two disparities: traffic pollution will likely require region-wide changes to vehicle standards or commuting methods, while lead risk could be equalized by targeted rehabilitations of dilapidated buildings and stricter code enforcement by city government of rental properties.

Conclusion

Though too often overlooked by social scientists, as Pebley (1998) and Entwisle (2007) highlighted, the physical environment is a key contributor to many demographic processes. Since their writing, the study of environmental toxins has grown considerably, although long-term estimates for social mobility and behavioral outcomes related to health and well-being remain relatively

rare. In this paper, we addressed this challenge, and showed that exposure to two different types of pollution in adolescence is predictive of three different major life outcomes in adulthood. The directions of relationships, if not always their magnitudes, are broadly similar for African Americans and whites, and for the most part Latinos as well. Yet exposure to the pollutants varies substantially by race. Differential exposure to pollution in childhood may thus be a contributor to racial inequality among adults.

We make no claims to have established causality. Our motivation was generative in intent, seeking to advance the measurement and analysis of environmental toxins at the national level for neighborhoods, including a rigorous descriptive analysis of linkages to income mobility and other outcomes of relevance to demographic processes and social inequality. The results indicate that the physical environmental should be further considered and tested as an important mechanism through which more commonly studied indicators of neighborhood disadvantage, such as the poverty rate, operate (Manduca & Sampson, 2019). We have also shown that traffic-related air pollution is a more equal opportunity toxin as it varies more independently with respect to concentrated poverty and racial composition.

The results further suggest that scholars should take note of the different spatial scales at which pollutants can be distributed, which have important implications for both the distribution of harms and the potential prospects for ameliorating them. In our study, levels of traffic pollution varied a great deal across regions, but less so within each metropolitan area. This pattern points to a larger scale problem, but one that might be able to attract a broad base of public support. In contrast, there was sharp variation within cities in the level of lead risk, which meant that African American children were disproportionately exposed to lead hazards even compared to their white peers growing up nearby. While the smaller scale of lead exposure

suggests that it could be addressed via targeted interventions, securing these may present political challenges given the enduring legacies of racial segregation (Massey & Denton, 1993) and contemporary opposition to policies seeking to undo them (Trounstine, 2018).

In the case of Latinos, there may even be countervailing forces that suppress the negative long-term consequences of exposure to environmental toxins. Further work is clearly needed to uncover how ambient pollution in neighborhoods and regions interacts with race and ethnicity.

References

Aizer, A., & Currie, J. (2019). Lead and Juvenile Delinquency: New Evidence from Linked Birth, School, and Crime Records. *The Review of Economics and Statistics, 102*(4), 575–587.

Aizer, A., Currie, J., Simon, P., & Vivier, P. (2018). Do Low Levels of Blood Lead Reduce Children's Future Test Scores? *American Economic Journal: Applied Economics, 10*, 307-341.

Alotaibia, R., Bechled, M., Marshall, J. D., Ramania, T., Zietsmana, J., Nieuwenhuijsene, M. J., & Khreisa, H. (2019). Traffic Related Air Pollution and the Burden of Childhood Asthma in the Contiguous United States in 2000 and 2010. *Environment International, 127*, 858-867.

Alshire, J., Karraker, A., & Clarke, P. (2017). Neighborhood social stressors, fine particulate matter air pollution, and cognitive function among older U.S. adults. *Social Science and Medicine, 172*, 56-63.

Bell, M. L., & Ebisu, K. (2012). Environmental Inequality in Exposures to Airborne Particulate Matter Components in the United States. *Environmental Health Perspectives*, *120*(12), 1699–1704.

Carson, E. A., & Sabol, W. J. (2016). *Aging of the State Prison Population, 1993–2013* (p. 38). US Bureau of Justice Statistics. https://www.bjs.gov/content/pub/pdf/aspp9313.pdf

Chetty, R., Friedman, J. N., Hendren, N., Jones, M. R., & Porter, S. R. (2018). *The Opportunity Atlas: Mapping the Childhood Roots of Social Mobility*. NBER Working Paper No. 25147. DOI: 10.3386/w25147.

Chetty, R., & Hendren, N. (2018). The Impacts of Neighborhoods on Intergenerational Mobility I: Childhood Exposure Effects. *The Quarterly Journal of Economics, 133*(3), 1107–1162.

Currie, J., Hanushek, E. A., Kahn, M., Neidell, M., & Rivkin, S. G. (2009). Does Pollution Increase School Absences? *The Review of Economics and Statistics, 91*(4), 682–694.

Elliott, J. R., & Frickel, S. (2013). The Historical Nature of Cities: A Study of Urbanization and Hazardous Waste Site Accumulation. *American Sociological Review, 78*(4), 521-543.

Entwisle, B. (2007). Putting People into Place. *Demography, 44*, 687-703.

Feigenbaum, J. J., & Muller, C. (2016). Lead exposure and violent crime in the early twentieth century. *Explorations in Economic History*, *62*, 51–86.

Frickel, S., & Elliott, J. R. (2018). *Sites Unseen: Uncovering Hidden Hazards in American Cities*. New York: Russell Sage Foundation.

Galster, G. C. (2012). The mechanism (s) of neighbourhood effects: Theory, evidence, and policy implications. In *Neighbourhood effects research: New perspectives* (pp. 23–56). Springer.

Health Effects Institute. (2010). *Traffic-related air pollution: A critical review of the literature on emissions, exposure, and health effects*. Health Effects Institute.

Isen, A., Rossin-Slater, M., & Walker, W. R. (2017). Every Breath You Take—Every Dollar You'll Make: The Long-Term Consequences of the Clean Air Act of 1970. *Journal of Political Economy, 125*, 848-902.

Jacobs, D. E., Clickner, R. P., Zhou, J. Y., Viet, S. M., Marker, D. A., Rogers, J. W., . . . Friedman, W. (2002). The Prevalence of Lead-Based Paint Hazards in U.S. Housing. *Environmental Health Perspectives, 110*(10), A599-A606.

Khreis, H., Kelly, C., Tate, J., Parslow, R., Lucas, K., & Nieuwenhuijsen, M. (2017). Exposure to traffic-related air pollution and risk of development of childhood asthma: A systematic review and meta-analysis. *Environment International*, *100*, 1–31.

Kravitz-Wirtz, N., Crowder, K., Hajat, A., & Sass, V. (2016). The Long-Term Dynamics of Racial/Ethnic Inequality in Neighborhood Air Pollution Exposure, 1990-2009. *Du Bois Review: Social Science Research on Race, 13*(2), 237-259.

Kravitz-Wirtz, N., Crowder, K., Hajat, A., & Sass, V. B. (2016). The Long-Term Dynamics of Racial/Ethnic Inequality in Neighborhood Air Pollution Exposure, 1990-2009. *Du Bois Review: Social Science Research on Race, 13*(2), 237-259.

Manduca, R., & Sampson, R. J. (2019). Punishing and toxic neighborhood environments independently predict the intergenerational social mobility of black and white children. *Proceedings of the National Academy of Sciences, 116*, 7772-7777.

Margolis, A. E., Herbstman, J. B., Davis, K. S., Thomas, V. K., Tang, D., Wang, Y., Wang, S., Perera, F. P., Peterson, B. S., & Rauh, V. A. (2016). Longitudinal effects of prenatal exposure to air pollutants on self-regulatory capacities and social competence. *Journal of Child Psychology and Psychiatry*, *57*(7), 851–860.

Massey, D. S., & Denton, N. (1993). *American Apartheid: Segregation and the Making of the Underclass*. Cambridge, MA: Harvard University Press.

McCoy, D., Raver, C. C., & Sharkey, P. (2015). Children's Cognitive Performance and Selective Attention Following Recent Community Violence. *Journal of Health and Social Behavior, 56*, 19-36.

Mohai, P., Kweon, B.-S., Lee, S., & Ard, K. (2011). Air Pollution around Schools Is Linked to Poorer Student Health and Academic Performance. *Health Affairs, 30*(5), 852–862.

Morello-Frosch, R., Pastor, M., & Sadd, J. (2001). Environmental Justice and Southern California's 'Riskscape' the Distribution of Air Toxics Exposures and Health Risks among Diverse Communities. *Urban Affairs Review, 36*(4), 551–578.

Mukherjee, A., & Agrawal, M. (2017). World air particulate matter: Sources, distribution and health effects. *Environmental Chemistry Letters*, *15*(2), 283–309.

Muller, C., Sampson, R. J., & Winter, A. (2018). Environmental Inequality: The Social Causes and Consequences of Lead Exposure. *Annual Review of Sociology, 44*, 263–282.

Newman, N. C., Ryan, P., LeMasters, G., Levin, L., Bernstein, D., Hershey, G. K. K., Lockey, J. E., Villareal, M., Reponen, T., & Grinshpun, S. (2013). Traffic-related air pollution exposure in the first year of life and behavioral scores at 7 years of age. *Environmental Health Perspectives*, *121*(6), 731–736.

Pebley, A. R. (1998). Demography and the Environment. *Demography, 35*, 377-389.

Perera, F. P., Wang, S., Rauh, V., Zhou, H., Stigter, L., Camann, D., Jedrychowski, W., Mroz, E., & Majewska, R. (2013). Prenatal exposure to air pollution, maternal psychological distress, and child behavior. *Pediatrics*, *132*(5), e1284–e1294.

Qiu, Y., Yang, F.-A., & Lai, W. (2019). The Impact of Indoor Air Pollution on Health Outcomes and Cognitive Abilities: Empirical Evidence from China. *Population and Environment, 40*(4), 388–410.

Reuben, A., Caspi, A., Belsky, D. W., Broadbent, J., Harrington, H., Sugden, K., . . . Moffitt, T. E. (2017). Association of Childhood Blood Lead Levels With Cognitive Function and Socioeconomic Status at Age 38 Years and With IQ Change and Socioeconomic Mobility Between Childhood and Adulthood. *JAMA, 317*(12), 1244–1251.

Reuben, A., Schaefer, J. D., Moffitt, T. E., Broadbent, J., Harrington, H., Houts, R. M., Ramrakha, S., Poulton, R., & Caspi, A. (2019). Association of childhood lead exposure with adult personality traits and lifelong mental health. *JAMA Psychiatry*, *76*(4), 418–425.

Roberts, S., Arseneault, L., Barratt, B., Beevers, S., Danese, A., Odgers, C. L., Moffitt, T. E., Reuben, A., Kelly, F. J., & Fisher, H. L. (2019). Exploration of NO2 and PM2. 5 air pollution and mental health problems using high-resolution data in London-based children from a UK longitudinal cohort study. *Psychiatry Research*, *272*, 8–17.

Sampson, R. J., & Winter, A. (2016). The Racial Ecology of Lead Poisoning: Toxic Inequality in Chicago Neighborhoods, 1995-2013. *Du Bois Review: Social Science Research on Race, 13*, 261-283.

Sampson, R. J., & Winter, A. (2018). Poisoned Development: Assessing Lead Exposure as a Cause of Crime in a Birth Cohort Followed through Adolesence. *Criminology, 56*, 269-301.

Sass, V., Kravitz-Wirtz, N., Karceskia, S. M., Hajatc, A., Crowder, K., & Takeuchi, D. (2017). The effects of air pollution on individual psychological distress. *Health and Place, 48*, 72–79.

Schachner, J. N., & Sampson, R. J. (2020). Skill-Based Contextual Sorting: How Parental Cognition and Residential Mobility Produce Unequal Environments for Children. *Demography 57*, 675–703.

Sharkey, P. T. (2010). The Acute Effect of Local Homicides on Children's Cognitive Performance. *Proceedings of the National Academy of Sciences, 107*, 11733-11738.

Sharkey, P. T., Tirado-Strayer, N., Papachristos, A. V., & Raver, C. C. (2012). The Effect of Local Violence on Children's Attention and Impulse Control. *American Journal of Public Health, 102*(12), 2287-2293. doi:10.2105/AJPH.2012.300789

Shier, V., Nicosia, N., Shih, R., & Datar, A. (2019). Ambient Air Pollution and Children's Cognitive Outcomes. *Population and Environment, 40*(3), 347–367.

Tolbert, C. M., & Sizer, M. (1996). *US commuting zones and labor market areas: A 1990 update*. https://trid.trb.org/view.aspx?id=471923

Trounstine, J. (2018). *Segregation by Design: Local Politics and Inequality in American Cities*. New York: Cambridge University Press.

Vivier, P. M., Hauptman, M., Weitzen, S. H., Bell, S., Quilliam, D. N., & Logan, J. R. (2011). The Important Health Impact of Where a Child Lives: Neighborhood Characteristics and the Burden of Lead Poisoning. *Maternal and Child Health Journal*, *15*(8), 1195–1202.

Volk, H. E., Lurmann, F., Penfold, B., Hertz-Picciotto, I., & McConnell, R. (2013). Traffic-Related Air Pollution, Particulate Matter, and Autism. *JAMA Psychiatry*, *70*(1), 71.

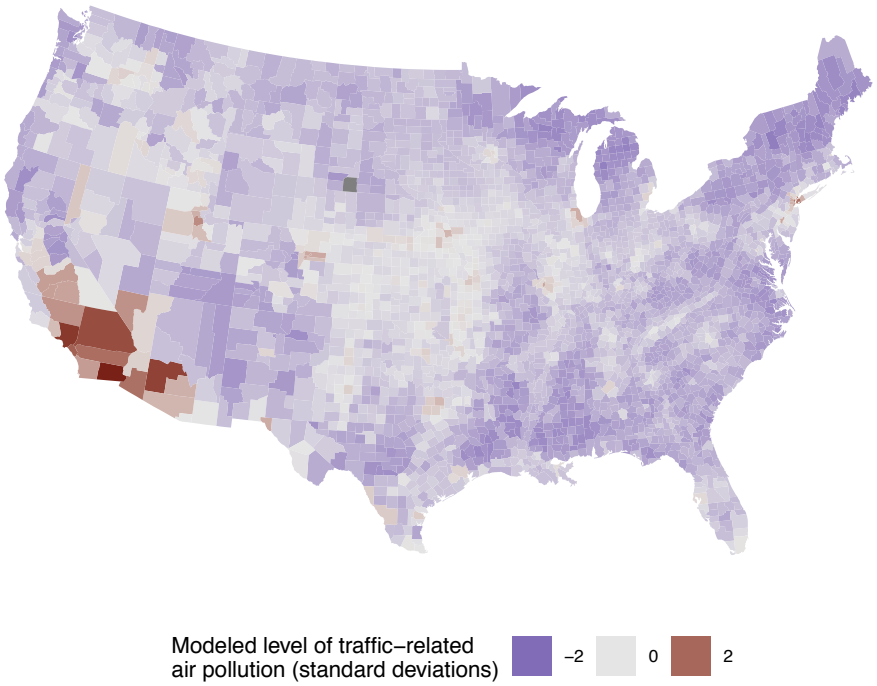
Winter, A. S., & Sampson, R. J. (2017). From Lead Exposure in Early Childhood to Adolescent Health: A Chicago Birth Cohort. *American Journal of Public Health*, *107*(9), 1496–1501.

Washington-State-Department-of-Health. (2015). *A Targeted Approach to Blood Lead Screening in Children, Washington State*. https://assets.documentcloud.org/documents/2644455/Expert-Panel-Childhood-Lead-Screening-Guidelines.pdf.

Zivin, J. G., & Neidell, M. (2018). Air pollution's hidden impacts: Exposure can affect labor productivity and human capital. *Science, 359*(6371), 39-40.

Figure 1. Maps of traffic-related air pollution and lead exposure risk by county, 2000

A. Traffic–related air pollution, 2000



B. Housing–derived lead exposure risk, 2000

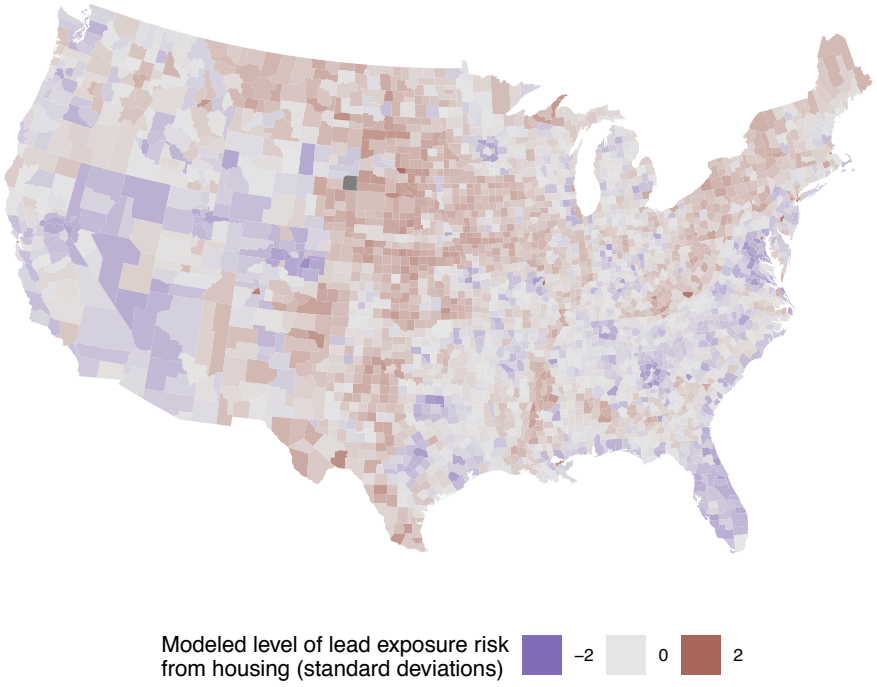


Figure 2. Maps of traffic-related air pollution and lead exposure risk by Census tract, Houston Commuting Zone, 2000

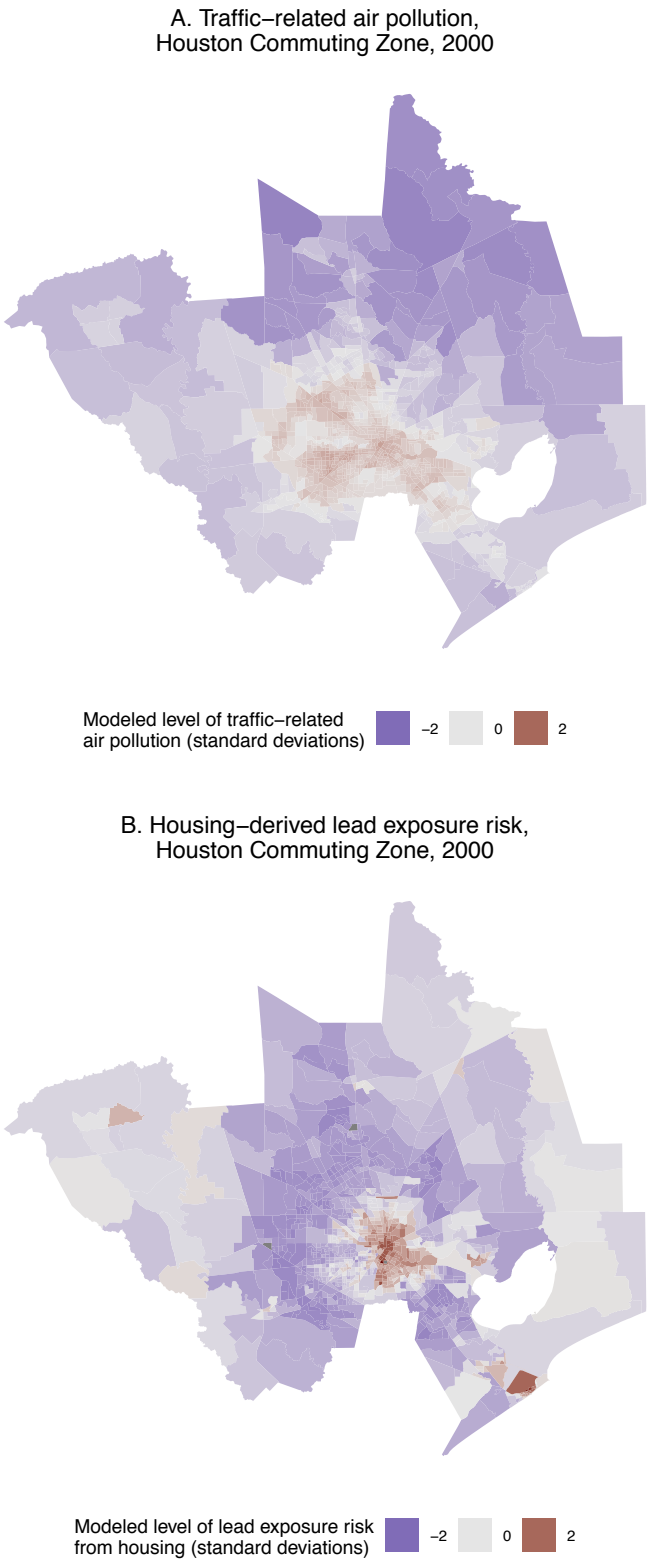


Figure 3. Histograms showing selected counterfactual simulations of exposure to pollution sources by race

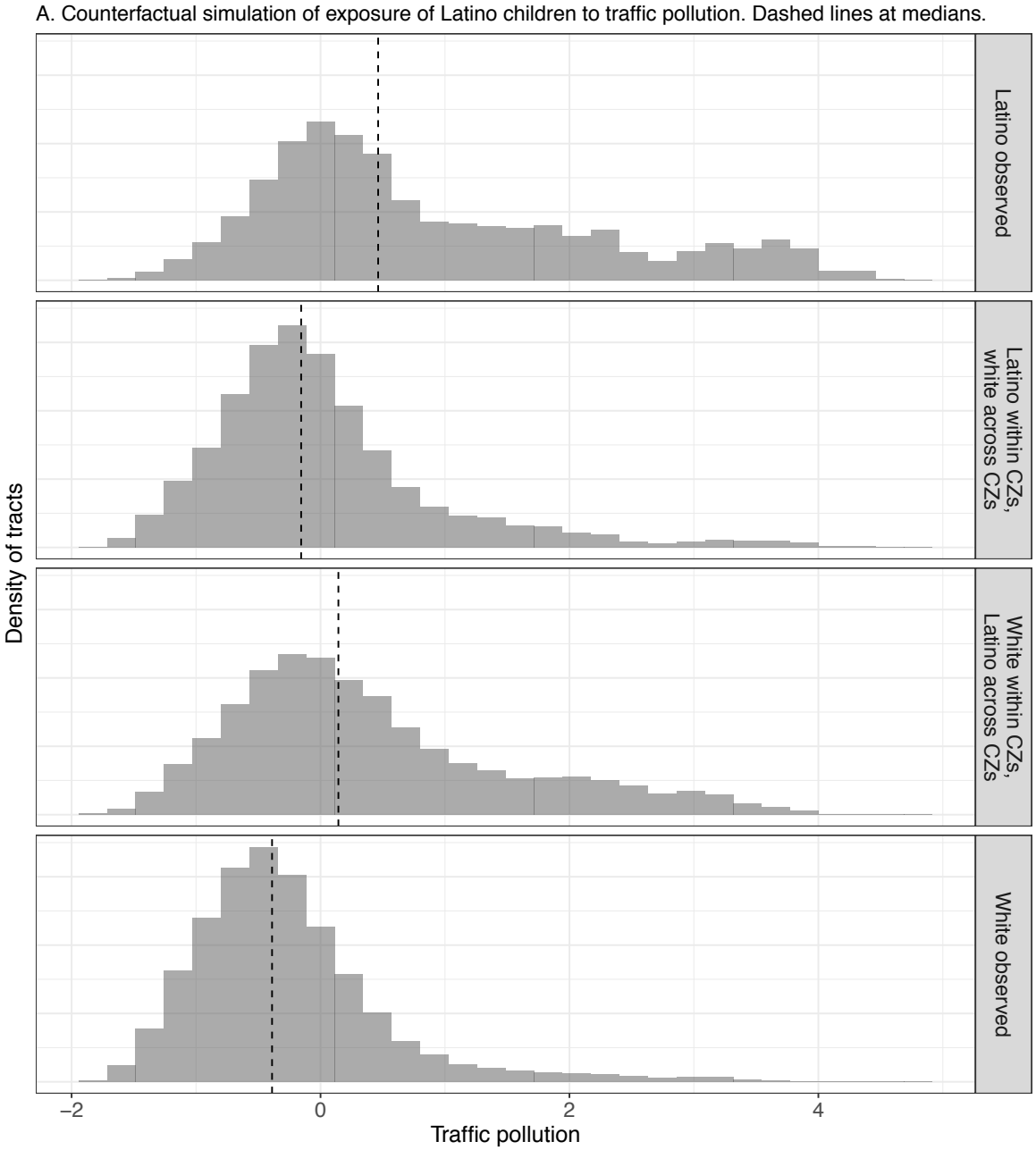


Figure 3 continued...

B. Counterfactual simulation of exposure of black children to lead risk. Dashed lines at medians.

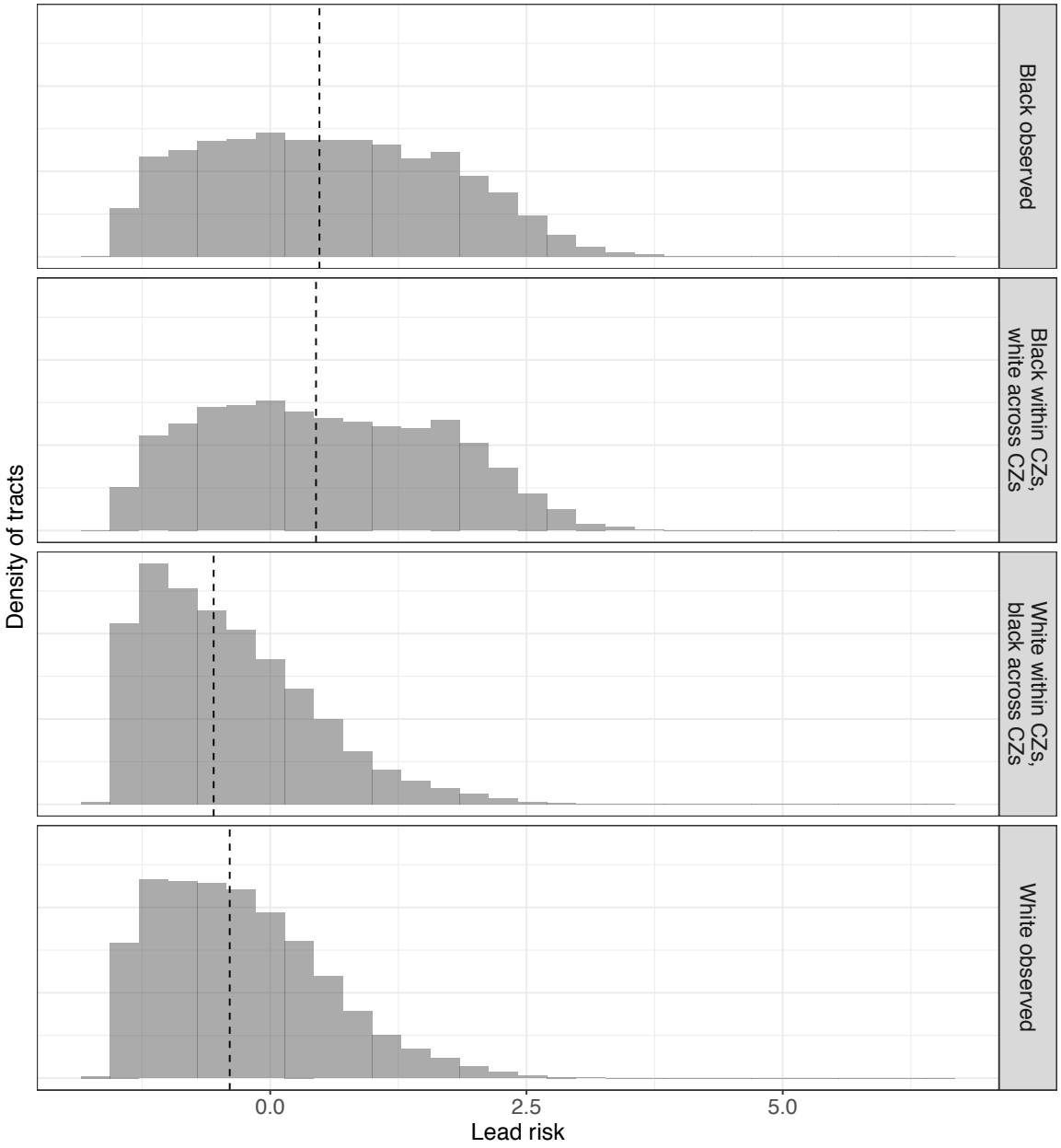


Table 1. Regression results, models predicting adult income rank, incarceration, and teen birth

Panel A: Main Specification	Statistic	African Americans			Whites			Latinos		
		Income	Jail	Teen birth	Income	Jail	Teen birth	Income	Jail	Teen birth
Poverty rate	beta	-0.194	0.103	0.210	-0.172	0.026	0.151	-0.330	0.128	0.311
	p	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Share foreign born	beta	0.338	-0.185	-0.308	0.107	-0.015	-0.085	0.248	-0.148	-0.219
	p	0.000	0.000	0.000	0.000	0.018	0.000	0.000	0.000	0.000
Share college educated	beta	0.207	-0.099	-0.196	0.335	-0.140	-0.362	0.090	-0.042	-0.181
	p	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Share African American	beta	0.081	-0.124	-0.084	-0.061	0.038	0.063	-0.067	-0.019	0.048
	p	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.010	0.000
Population density	beta	-0.021	-0.059	0.004	-0.059	-0.028	-0.027	-0.092	0.002	0.037
	p	0.000	0.000	0.584	0.000	0.000	0.000	0.000	0.855	0.000
Share commuting <15 minutes	beta	-0.074	0.055	0.064	-0.066	0.041	0.066	-0.030	0.002	0.031
	p	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.829	0.000
Share of homes built before 1940	beta	0.002	0.023	-0.003	0.108	-0.046	-0.060	0.008	0.000	-0.007
	p	0.772	0.058	0.771	0.000	0.000	0.000	0.311	0.974	0.440
Share employed in manufacturing	beta	-0.004	-0.011	0.058	-0.023	0.021	0.087	0.011	0.073	0.089
	p	0.515	0.270	0.000	0.000	0.001	0.000	0.052	0.000	0.000
Traffic-related air pollution	beta	-0.110	0.124	0.125	-0.055	0.035	0.067	0.022	-0.082	-0.132
	p	0.000	0.000	0.000	0.000	0.000	0.000	0.013	0.000	0.000
Lead exposure risk	beta	-0.190	0.125	0.085	-0.137	0.072	0.038	-0.081	0.049	-0.044
	p	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.003	0.000
CZ fixed effects		Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
N (Census tracts)		33,964	21,622	25,273	67,493	64,216	64,826	37,285	22,316	25,673
R-sq		0.663	0.393	0.625	0.600	0.129	0.579	0.560	0.311	0.628
adj. R-sq		0.659	0.383	0.619	0.596	0.119	0.575	0.554	0.300	0.623

Panel B: Alternate Specifications[a]	Statistic	African Americans			Whites			Latinos		
		Income	Jail	Teen birth	Income	Jail	Teen birth	Income	Jail	Teen birth
B1: Lead exposure risk: specification excluding poverty rate and share of homes built before	beta	-0.343	0.223	0.251	-0.137	0.046	0.068	-0.285	0.129	0.148
	p	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
B2: Particulate matter - specification with two air pollution factors	beta	0.054	-0.049	0.052	0.201	-0.094	-0.073	0.070	[b]	0.030
	p	0.000	0.001	0.000	0.000	0.000	0.000	0.000	[b]	0.017
B2: NO2 / CO - specification with two air pollution factors	beta	-0.210	0.224	0.087	-0.266	0.136	0.150	-0.040	[b]	-0.168
	p	0.000	0.000	0.000	0.000	0.000	0.000	0.000	[b]	0.000

[a] With the exception noted in B1, control variables are the same as in Panel A, but their coefficients and standard errors are not shown.

[b] The regressions predicting jail among Latinos with two air pollution factors did not converge.