# Air Pollution and Children's Respiratory Health: A Cohort Analysis[1]

Timothy K.M. Beatty[†] and Jay P. Shimshack[‡]

November 2012

ABSTRACT

This paper uses a large and representative database of multiple birth cohorts to study relationships between air pollution exposure and non-infant children's respiratory health outcomes. We observe several years of early-life health treatments for each of nearly *700,000* children. Three distinct research designs account for potential socioeconomic, behavioral, seasonal, and economic confounders. We find that marginal increases in carbon monoxide and ground-level ozone are associated with statistically significant increases in children's contemporaneous respiratory treatments. We also find that carbon monoxide exposure over the previous year has an effect on children's health that goes above and beyond contemporaneous exposure alone.

KEYWORDS: air pollution, environmental health, public health, children's health, cohort
JEL I18, Q53, Q58

[1] Both authors contributed equally to this paper. Primary analysis of HES data was conducted by Beatty at the University of York, UK. We thank Mark Dusheiko for help with the HES database. We thank Spencer Banzhaf, Mary Evans, Don Fullerton, Dan Kaffine, Andrew Jones, Nigel Rice, and seminar participants at the 2009 Heartland Workshop, the 2010 ASSA annual meetings, the 2010 World Congress of Environmental and Resource Economists, the 2011 AERE conference, Georgia State University, and the University of Washington. Jason Edgar provided excellent research assistance.
[†] Dept of Applied Economics, Univ of Minnesota, 317e Ruttan Hall, St Paul, MN 55108. tbeatty@umn.edu .
[‡] Dept of Economics, Tulane University, 206 Tilton Hall, New Orleans, LA 70118. jshimsha@tulane.edu .

## 1. Introduction

Pollution regulations are controversial, and economists and policymakers continue to debate their efficiency and cost effectiveness. Discussions of the benefits typically focus on health considerations. In principle, controlled clinical experiments could conclusively estimate links between pollution and human health. In practice, however, much of this research is prevented by ethical and other considerations. Relationships between pollution and morbidity or mortality are most often inferred from observational data.

A literature published in epidemiological journals establishes statistical associations between air pollution and human health. Economists have recently contributed new datasets and empirical approaches to study links between pollution and morbidity and mortality. The aim is a more precise estimate of the causal effect of pollution. These latter studies enhance our understanding of the relationships between air quality and health by more completely controlling for potentially confounding unobserved factors.

This paper builds on the recent literature by constructing a rich database of multiple birth cohorts to examine relationships between air pollution exposure and children's morbidity. We focus on children's health for three reasons. Relationships between pollution and health outcomes for non-infant children are understudied and relatively poorly understood. Closely related studies often focus on links between pollution and infant mortality or pollution and adult outcomes. Children are also thought to be highly susceptible to damages from pollution. High-risk impacts are likely attributable to ongoing physiological respiratory development, smaller average lung size, and increased activity levels (Committee on Environmental Health 2004; Gauderman 2000). Finally, effects may be long lasting as early-life illness may impede long-term human capital development (Currie 2009).

Our analysis makes several contributions. First, our dataset is unusually large and detailed. We observe several years of early-life health treatments for each of nearly *700,000* children. We analyze health outcomes for the majority of children born in English cities between 1997 and 1999, and our sample is representative, at least for urban children. Second, we assess the health impacts of both contemporaneous pollution exposure and average pollution exposure over the previous year. Studies emphasizing causal effects typically only identify contemporaneous pollution impacts. However, we observe repeated observations for each individual and individuals from multiple birth cohorts, so plausible attribution of some non-

contemporaneous impacts is possible. Third, our pollution and weather data are observed at a fine geographic scale. Our geographic unit of analysis – English middle super output areas – average less than 1/3 of the size of the average California zip code. Fourth, we examine data from a universal health care system. This setting offers two advantages: we observe both inpatient treatments and day cases, and we minimize common selection bias concerns that arise due to differences in insurance coverage and ability to pay.

Even with a rich dataset, attributing health outcomes to pollution can be challenging. A household's location is not randomly assigned, so socioeconomic confounders may be correlated with both pollution exposure and health outcomes via mobility and Tiebout sorting. Several determinants of illness may be spuriously correlated with pollution through seasonality. Local trends in economic activity may influence both pollution and health. Our research design seeks to isolate causal impacts. We control for children's age, health at birth measures, seasonality, weather, and national time trends. We identify remaining relationships between pollution and non-infant children's health outcomes in three distinct ways: (1) Analyses include individual-level fixed effects. Identification of a given individual's dose-response relationship comes only from atypical deviations from that individual's own average pollution exposure, over all sample periods. Here, time invariant individual-level confounders like income, race, and persistent differences in local economic conditions will not bias estimates. Tiebout sorting correlated with long-run average differences in pollution will not bias estimates. (2) Analyses include local area-by-year fixed effects. Identification of a given individual's dose-response relationship comes only from atypical within-area deviations from that area's average pollution exposure, for that same year. Confounders cannot bias estimates unless they are correlated with unusual or anomalous pollution levels within an area and a year. Tiebout sorting correlated with neighborhood specific trends in pollution will not bias estimates. (3) Analyses include area-by-age fixed effects. Identification of an average individual's dose-response relationship comes from differences in pollution exposure for children of the same age and living in the same area but born at different times. The intuition is that children living in the same area but born several months to a few years apart are presumed similar and are presumed to have grown up in similar circumstances, but face somewhat different pollution exposures at a given age because they reach that age at a different point in time.

We find that marginal increases in carbon monoxide (CO) and ground-level ozone (O3) are associated with statistically significant increases in children's contemporaneous respiratory treatments. CO results are especially robust. We believe these findings are novel for two reasons. First, non-fatal morbidity impacts of carbon monoxide at common ambient levels remain poorly understood. The EPA's integrated science assessment emphasizes that only a limited number of studies link carbon monoxide and respiratory health, and that the present evidence is merely "suggestive that a causal relationship exists" (USEPA 2010). Second, associations between criteria pollutants and morbidity outcomes for non-infant children are understudied. Most studies stressing causal effects focus on infant mortality, infant morbidity, and adult mortality.

We also find that CO exposure over the previous year has an incremental effect on children's health that goes above and beyond contemporaneous CO exposure alone. While we do not claim to fully capture the cumulative effects of pollution on children's respiratory health, we do contribute additional evidence on the causal effects of longer-term pollution exposure. These are open questions; the EPA asserts that the "available evidence is inadequate to conclude that a causal relationship exists" between longer-term CO and respiratory morbidity (USEPA 2010). Our findings suggest that research that focuses only on the acute health impacts of pollution may understate the benefits of pollution reductions.

## 2. Background and Literature

We study the health impacts of particulate matter (PM10), carbon monoxide (CO), and ozone (O3) concentrations. Particulate matter consists of solids and liquids suspended in the air. Particulates smaller than 10 micrometers in diameter are designated PM10. Common PM10 sources include construction, on and off road vehicles, fires, and industrial facilities including power plants. Carbon monoxide is a colorless and odorless gas formed when carbon in fuel is incompletely burned. Vehicle emissions are the primary source of ambient carbon monoxide. Ground-level ozone is created from chemical reactions that occur between oxides of nitrogen and volatile organic chemicals in the presence of sunlight and heat. Primary ground-level ozone sources are vehicle emissions, gasoline vapors, and industrial facilities including power plants.

### 2.1 Pathways Linking Pollution and Respiratory Health in both the Short- and Longer-Run

Medical research, including animal toxicology and in vitro mechanistic studies, suggests several biological pathways that may link contaminants with respiratory health outcomes in humans. Deposition of inhaled particulate matter (PM10) induces acute and persistent airway

inflammation, lung inflammation, pulmonary injury, and reduced lung function. More precise mechanisms may include oxidative stress, reduced host defenses against infectious disease, respiratory surface permeability disruptions, and alterations in cell signaling activity (USEPA 2009). It is also believed that ozone (O3) causes lung inflammation, reduced lung function, and chronic lung disease, although specific mechanisms remain controversial (USEPA 2006). It has been long accepted that carbon monoxide (CO) exposure at extremely high levels induces hypoxic responses that can lead to severe morbidity or mortality (Raub and Benignus 2002). Plausible mechanisms linking health outcomes and more typical ambient levels of carbon monoxide were unknown until recently. Recent evidence suggests that carbon monoxide alters protein function at concentrations near those commonly observed. Precise pathways may include a combination of hypoxic stress, oxidative stress, and cell signaling changes (USEPA 2010). Note that outside of controlled experimental settings, health reactions to CO may also be attributable to high correlations between CO and currently unmeasured toxic air pollutants also common in vehicle emissions.

Animal toxicology, in vitro mechanistic, and limited controlled human exposure studies also suggest that the effects of contemporaneous or shorter-run exposure may differ from the effects of longer-term exposure. Shorter-run or acute pollution exposure may be more likely to be associated decrements to pulmonary functions like breathing rate and volume, pulmonary inflammation, oxidative injury, and exacerbation of existing allergies (USEPA 2006; USEPA 2009; USEPA 2010). Longer-run pollution exposure may be more likely to be associated with pulmonary injuries related to wall thickness, protein structure and protein function, lung growth and development, cell signaling changes, airway remodeling, and the progression of allergies (USEPA 2006; USEPA 2009; USEPA 2010).

**2.2 Observational Studies Linking Air Pollution and Health Outcomes**

Numerous studies establish statistical associations between air pollution and health outcomes. Early epidemiologic research often examined time-series relationships between pollution concentrations and morbidity or mortality outcomes for a single city. More recent studies investigated independent time-series associations for several cities, and then used meta-analyses to estimate average relationships over a larger study area (Spix et al. 1998; Samet et al. 2000; Dominici 2003). Research published in epidemiology journals increasingly uses multi-city cohort or repeated cross-section approaches (Dockery et al. 1993; Pope et al. 1995; Peters et al.

1999; Pope et al. 2002; Gauderman 2007; Jarrett et al. 2009; Sheffield et al. 2011). These studies often employ a two-step research design: First, individuals' health outcomes over several periods are regressed on community identifiers and individual-level covariates. Second, the estimated community-level fixed effects, referred to as relative risks, are regressed on long-term community-level average pollution measures.

These recent advances have contributed significantly to the state of knowledge. The widely used two-step approach accounts for the fact that air quality exposure is usually observed at the community-level. However, published estimates may be affected unobserved factors that confound causal identification, as common research designs in the epidemiology-oriented literature often ultimately exploit purely cross-sectional or purely temporal statistical identification (Chay et al. 2003; Chay and Greenstone 2003). In response to these concerns, environmental and health economists have begun to contribute additional datasets and statistical tools to the study of pollution and health, with the goal of isolating causal relationships. Economists typically use one of three research designs: The first design links contaminant exposure to self-reported health outcomes and detailed individual-level characteristics collected from surveys. The second design involves natural experiments or instrumental variable approaches. The third involves fixed effect approaches that exploit within-area pollution variation. Studies also vary based on the health outcomes they consider (morbidity vs. mortality), the unit of observation (individuals vs. areas), and the population of interest (infants, children, adults, the elderly, etc.).

Several notable studies consider pollution and mortality. Pope et al. (1992) exploited the closing and reopening of a steel mill in Utah Valley to identify the effect of PM10 exposure on adult mortality. Chay et al. (2003) examined the relationships between early 1970's suspended particulates and adult mortality using county-by-year variation induced by the Clean Air Act. Chay and Greenstone (2003) used a natural experiment stemming from the 1981-1982 recession to examine the relationship between total suspended particulates and infant mortality at the county-by-year level. Janke et al. (2009) explored relationships between several air pollutants and population mortality rates with local authority-by-year data from the UK during the late 1990's and early 2000's.

Other well-cited studies investigated relationships between pollution and morbidity. Neidell (2004) used seasonal pollution variation within California zip codes to examine the

connection between several air pollutants and children's asthma hospitalizations during the 1990's. Moretti and Neidell (2011) used boat traffic at the port of Los Angeles as an instrument to estimate the impacts of ozone on zip-code level hospitalizations in Southern California during the 1990's. Schlenker and Walker (2011) used exogenous changes in daily airport traffic in California to investigate relationships between changes in short-run pollution exposure and changes in unplanned hospitalizations among those who live near airports.

The studies discussed in the preceding paragraphs typically analyze spatially aggregated data, largely because pollution exposure is not observed at the individual level. An alternative approach uses individual health outcome data. This allows for different statistical approaches and may allow for individual-level controls. Much of this work focuses on infant outcomes. Currie and Neidell (2005) and Currie et al. (2009) used individual-level data and extensive fixed effect structures to examine relationships between pollution and infant outcomes in California and New Jersey during the 1990's. Knittel et al. (2009) used road traffic as an instrument for pollution exposure to investigate relationships between pollution and infant mortality in California during the early 2000's. Currie and Walker (2011) exploited the introduction of EZ-pass toll collection systems to explore the relationships between traffic congestion and prematurity and low birthweight.

An alternative means of collecting individual information is survey data. The use of survey methods allows detail to be collected about individual characteristics, outcomes, and behaviors. Krupnick et al. (1990) matched daily variation in air pollution with daily variation in self-reported health status for individuals living in Southern California. Evans and Smith (2005) used survey data from several birth cohorts to explore relationships between long-term pollution exposure and the onset of previously unreported serious health conditions in older adults.

One thing to note is that the literature emphasizing causal effects has largely focused on adults and infants. Work on children is somewhat less common. Notable studies include Pope (1989), which used the closure and reopening of a steel mill to identify the effects of PM10 on hospital admissions in Utah Valley. Lleras-Muney (2010) leveraged changes in location due to military transfers to study the impact of pollution on hospitalizations for military children. Beatty and Shimshack (2011) exploited differential timing of school bus retrofit programs in the Puget Sound area of Washington to explore the relationships between localized air pollution programs and children's respiratory outcomes during the early 2000's.

This paper builds on the studies reviewed above, as well as the larger literature exploring pollution and health. We use a birth cohort research design. We study the relationship between pollution and non-fatal health outcomes for non-infant children. We use a broad and representative sample. Our unique dataset also allows us to consider the effects of both contemporaneous pollution exposure and the average pollution exposure over the past year.

## 3. Data

To analyze the relationship between pollution and children's morbidity, we construct an individual-by-month panel. Time invariant individual characteristics are not aggregated. Each individual's health outcomes are summed over days in the month. Monthly pollution and weather exposure data are calculated for the middle super output area (MSOA) of the individual's residence. MSOA's are fine geographic units; for perspective, the average MSOA is less than 1/3 the size of the average California zip code.

### 3.1 Individual data

We collect comprehensive health outcome data from England's Hospital Episodes Statistics Database (HES). The HES tracks individuals' contacts with National Health Service (NHS) hospitals and treatment centers funded by the NHS. We first obtain birth records. For each of 1.13 million children born between 1997 and 1999, or about 2/3 of all children born in the country during those years, we observe an individual identifier, date of birth, and MSOA of residence at birth.[2] For about 50 percent of the birth records, we also observe sex, weeks of gestation at birth, birth weight in grams, and maternal characteristics.

We then obtain individual-level inpatient and day case discharge data from all NHS hospitals and treatment centers funded by the NHS. Each discharge observation consists of an individual identifier, treatment date, patient age, patient MSOA at time of treatment, and a detailed diagnosis code. Consistent individual identifiers allow us to match birth records with health events and allow us to track each child's complete NHS contacts over many years. We track each child for 60 months, from their 2nd birthday until their 7th birthday. For example, for a child born in June 1999, we observe health treatments from June 2001 through June 2006. We do

[2] We do not observe children born in private hospitals or private homes in England. We omit stillborn children and children who die immediately following birth. We also omit children with birth records that are missing MSOA of residence. Comparisons with national statistics suggest that our 1.13 million children represent approximately 2/3 of all children born in England between 1997 and 1999.

not study early childhood NHS contacts since infant outcomes are well studied in the literature, and because morbidity outcomes during these years are confounded with mortality outcomes.[3]

Since our focus is on air pollution and children's health, we analyze diagnosis codes related to diseases of the respiratory system (ICD-10 code J01). Discharges include those related to upper respiratory infections, influenza and pneumonia, acute lower respiratory infections such as bronchitis, chronic respiratory infections including asthma and chronic bronchitis, and other diseases of the respiratory system. A few respiratory ailments, like pleurisy, have never been associated with air pollution. Any potential measurement error from including such illnesses conservatively biases our pollution impacts towards zero and reduces statistical precision. For every child, our individual-by-month measure is a dummy variable equal to one if a respiratory treatment occurred and zero otherwise.

**3.2 Pollution and weather data**

We collect comprehensive pollution data from the UK Air Quality Archive for January 1997 through December 2006. We obtain monitor-by-hour readings on particulate matter (PM10), ozone (O3), and Carbon Monoxide (CO). For each contaminant, approximately 60-80 monitors assess concentrations at any given time. A spatial distribution map is presented in Figure 1. Monitors measure pollution in every region of England, but monitor density is highest where population density is highest. For example, multiple monitors are clustered within the metro areas of London, Birmingham, Leeds, Manchester, Liverpool, and Newcastle/Sunderland.

We assign concentrations for each pollutant to each MSOA-month following Currie and Neidell (2005). However, since urban monitor density is higher in England than it is in most of the United States, we choose a smaller pollution exposure radius than is common in the literature. The goal is to reduce exposure measurement error.[4] The assignment procedure is as follows. First, we identify the population-weighted center of each and every MSOA. Second, we identify, for each MSOA-pollutant-day combination, all reporting pollution monitors within a 10 mile radius of the identified population-weighted centroid. Third, we assign each monitor a weight proportional to the inverse of its distance from the MSOA center. We calculate these weights daily, since some monitors do not measure all pollutants for all sample days. Fourth, we calculate a weighted pollution concentration for every MSOA-pollutant-hour using the weights

[3] Children who die cannot be later observed in a hospital or primary care facility. Children's deaths from respiratory conditions after the second birthday are extremely rare.

[4] As discussed in a later sensitivity section, results are robust to larger pollution radii as well.

from step 3. Fifth, we calculate the monthly mean over the hourly measures to obtain pollution concentrations for each MSOA-contaminate-month combination.

We also assign weather data to each MSOA-month combination. We first obtain raw data from the British Atmospheric Data Centers' MIDAS Land Surface Station's database. We then construct data at the appropriate level for analysis following the pollution algorithm described above. In short, we assign weather outcomes to MSOA's using inverse distance weighted averages from stations within a 10 mile radius of the MSOA center. Variables include monthly average temperature, monthly maximum temperature, monthly average humidity, monthly maximum humidity, monthly average wind speed, and monthly maximum wind speed.

**3.3 Pollution summary statistics**

Table 1 summarizes pollution means and variability. For the period 1997-2006, average CO for urban and suburban areas in England was 0.71 milligrams per cubic meter (mg/m3). Average PM10 and average O3 were 25.6 and 53.4 micrograms per cubic meter (µg/m3), respectively. For perspective, UK health-based air quality regulations were based in part on standards of (1) a 10 mg/m3 8-hour running mean for CO, (2) a 50 µg/m3 daily mean for PM10, and (3) a 40 µg/m3 annual mean for PM10. Ozone regulations did not exist over the sample period, but published ozone air quality objectives were based on a 100 µg/m3 8-hour running mean.

Pollutant concentrations throughout England during the late 1990s and early 2000s were substantially lower than well-studied US pollutant concentrations during the 1990s. While direct comparisons are difficult, our CO, PM10, and O3 concentrations are approximately one-fifth to one-half of the US national concentrations over the same period as reported by the US Environmental Protection Agency (USEPA 2012). Relationships between these lower average pollution levels and health are important because many pollutants are declining throughout the industrialized world. Understanding current and *future* marginal benefits of pollution regulations requires an understanding of links between lower pollution exposures and health.

For the period 1997-2006, pollution varied significantly. Table 1 indicates that overall pollutant standard deviations were approximately 20 to 50 percent of mean pollution levels. Results in the last two table columns of the table indicate that the dispersion is driven by both variability across geographic areas and variability within areas across time. Figure 2 explores temporal variability in more detail. CO displays a clear long-term downward trend over our

sample period. In contrast, O3 increased slightly on average and PM10 experienced no obvious long-term trend over the sample period. Seasonal variability, especially for CO and O3, is pronounced. Seasonal peaks in CO occur in the late fall and early winter while seasonal peaks in O3 occur in the late spring and early summer. Figure 2 also displays significant variability unexplained by long-run trends and seasonality alone.

Our three pollutants are correlated with one another. The correlation coefficient between CO and O3 is -0.55. As noted above, this is at least partially driven by divergent long-term trends and opposite seasonal peaks. The correlation coefficient between CO and PM is +0.38. Since CO and PM generally do not experience similar trends over time, this correlation may be largely driven by geographic clustering. The correlation coefficient between PM and O3 is a relatively modest -0.09.

**3.4 Individual-level summary statistics**

Our final data construction step merges all data to the individual-by-month level. For each individual and each month, we assign pollution and weather outcomes based on last known residence. MSOA of residence is directly observed at every contact with a hospital or treatment center funded by the NHS, including birth, but not directly observed between contacts. We therefore infer a child's residence in any given month based on last known residence. Potential issues arising from relocation are discussed in detail below, although we note here that we rarely observe a sample child relocating to another MSOA.

We retain all individuals living in MSOAs with complete pollution data for all three contaminants and all months spanning birth through 2006. This procedure yields a final sample of 681,958 children, of which 328,920 have full control variables such as health-at-birth measures. Since we only retain children living within 10 miles of pollution and weather monitors, our sample children are predominantly located in urban and suburban areas.

Table 2 presents individual-level summary statistics for the full analysis sample and for the subsample with individual-level covariates like health-at-birth indicators. Overall summary statistics are all consistent with English national health statistics. 49 percent of sample young children are boys. The average sample child was born at 3300 grams after 39.2 weeks of gestation to a mother who averaged 28.2 years of age. We observe no statistical differences in environmental exposures or respiratory health outcomes for the full sample and the subsample with more complete individual-level controls.

## 4. Relationships between pollution and children's health outcomes

Our basic empirical strategy is to regress the probability of child *i*'s respiratory treatment in month *t* on one or more pollution measures. In principle, coefficients on these pollution measures represent the impact of marginal changes in pollution exposure on children's respiratory outcomes. In practice, however, a number of challenges arise because pollution exposure is not randomly assigned. First, pollution exposure may be correlated with individual characteristics that directly influence childhood morbidity like demographics and maternal behavior. One notable concern is that household income and other socio-demographics may be correlated with pollution exposure through Tiebout sorting, as environmental quality may be reflected in housing prices (Chay et al. 2003; Banzhaf and Walsh 2008; Bayer, Keohane, and Timmins 2009). Second, while pollution exposure is highly seasonal, respiratory health outcomes may also be seasonal for reasons other than pollution. For example, evidence suggests that weather directly influences disease transmission and virus survival (Lowen et al. 2007; Lowen et al. 2008; Shaman and Kohn 2009; Barreca 2012). Third, pollution exposure may be correlated with increased local area economic activity, which may feedback to health care quality and health outcomes (Knittel et al. 2009).

Our dependent variable is the probability of a respiratory treatment $R_{it}$ for child $i$ in period $t$. Our key explanatory variables are pollution measures $P_{mt}$. We first consider average pollution over the contemporaneous month for each contaminant individually. For example, the explanatory variable may be the log of mean CO in individual $i$'s MSOA $m$ during month $t$. Individual PM10 and O3 regression specifications are analogous. We augment individual contemporaneous exposure regressions with average monthly exposure over the previous year. For example, a CO regression may contain an additional explanatory variable representing the log of mean CO in individual $i$'s MSOA $m$ over the 12 months preceding $t$. PM10 and O3 regression specifications are analogous.[5] In order to account for possible correlations between contaminants, we run regressions for all three pollutants and/or all lagged pollution measures simultaneously.

[5] Models with variables reflecting average exposure over the previous year are equivalent to distributed lag models with monthly lags and coefficients constrained to be equal. An alternative approach is to regress health on a variable for each and every month over the last year, which allows the effects of different lags to have differential effects on health. However, as a practical matter, individual lagged pollution measures are highly collinear and separate identification is difficult.

Other critical explanatory variables include a nonlinear spline in age with 15 knots spread evenly over the observed life of each child. One of the important advantages of individual-level data is the ability to adequately control for child age. According to the US Census Bureau Statistical Abstract, childhood age is highly correlated with health care utilization and hospitalization. Age may also be correlated with pollution exposure through time spent outdoors and activity choice. Without flexible nonlinear controls for age, the potential for omitted variable bias is high.

We also control for time invariant individual characteristics such as sex, birthweight, mother's age at birth, and gestation at birth. These may be correlated with health outcomes and treatment propensities during childhood. We control for seasonality, weather, and common national time trends. Month-of-year dummy variables account of seasonal environmental and economic factors common across all MSOAs. Time variant weather variables include MSOA-level monthly average and monthly maximum temperature, humidity, and wind speed. We account for annual changes in economic activity, general welfare, and respiratory health that are common across MSOAs with year dummies.

**4.1 Three empirical approaches**

We attempt to minimize remaining endogeneity concerns with three different empirical designs, each with its own strengths and weaknesses. We model the probability of a respiratory treatment $R$ for child $i$ of age $a$ living in MSOA $m$ in time period $t$ of season $s$ and year $y$ as:

$$(1)\quad R_{imtsy} = P_{mt}\mathrm{B} + A_i(t) + W_{mt}\Upsilon + \eta_y + \omega(t) + \alpha_s + \pi_i + \mu_{imtsy}\,.$$

$$(2)\quad R_{imtsy} = P_{mt}\mathrm{B} + X_i\Gamma + A_i(t) + W_{mt}\Upsilon + \alpha_s + \xi_{ym} + \mu_{imtsy}\,.$$

$$(3)\quad R_{iamtsy} = P_{mt}\mathrm{B} + X_i\Gamma + W_{mt}\Upsilon + \eta_y + \alpha_s + \tau_{am} + \varepsilon_{iamtsy}\,.$$

$P_{mt}$ denotes pollution measures, $X_i$ denotes time invariant demographic characteristics, $A_i(t)$ denotes a piecewise linear spline in child $i$'s age at time $t$, $W_{mt}$ denotes weather variables, $\eta_y$ denotes year dummies, $\omega(t)$ denotes regional time trends, $\alpha_s$ denotes month-of-year season dummies, $\pi_i$ denotes individual-level fixed effects, $\xi_{ym}$ denotes MSOA-by-year fixed effects, $\tau_{am}$ denotes MSOA-by-age fixed effects, and $\mu$ denotes a standard idiosyncratic error term.[6]

[6] Regions are defined by the U.K. standard Government Office Regions. We do not include time invariant demographics in empirical model (1) as these controls are implicit in that fixed effect specification. We do not include region-specific time trends in empirical models (2) and (3) as these controls are implicit in those fixed effect specifications.

Specification (1) focuses on addressing potential concerns about unobserved individual-level heterogeneity. Recall that we control for several observed individual-level characteristics, weather, seasonality, and regional trends. Net of these effects, identification of the relationship between a given individual's pollution exposure and morbidity comes from atypical deviations from that individual's own average pollution exposure over all sample periods. Individual-level fixed effects control for income, race, education, health consciousness, maternal characteristics, prenatal pollution exposure, etc. Individual-level fixed effects also prevent bias due to Tiebout sorting driven by, or correlated with, average differences in pollution across MSOAs.

In specification (2), identification of a given individual's relationship between pollution and morbidity comes only from atypical within-MSOA deviations from area-average pollution exposure for that same year (again, net of observed individual-level characteristics, weather, seasonality, etc.). MSOA-by-year fixed effects control for unobserved time invariant average differences like income and education, as well unobserved annual shocks common to all individuals within an MSOA. Such shocks may affect localized economic activity, health care access, and public policy outcomes. Note also that MSOA-by-year fixed effects allow for neighborhood specific trends in pollution. As such, specification (2) prevents bias due to Tiebout sorting driven by, or correlated with, average differences in pollution across MSOAs or even MSOA-specific trends in pollution .

Specification (3) leverages the multiple cohort and large *t* longitudinal nature of our dataset to combine advantageous aspects of specification (1) and specification (2). Here, identification of the relationship between pollution and morbidity comes from within-MSOA differences in pollution exposure for children of the same age but born at different times (yet again, net of observed individual-level characteristics, weather, seasonality, etc.). The intuition is that children living in the same area but born several months to a few years apart are presumed similar and presumed to have grown up in similar circumstances, but face different pollution exposures at a given age because they reach that age at a different point in time.[7]

[7] Consider two children living in same neighborhood. Because they grew up in the same area, they may be relatively similar. However, child A is born in January 1998 and Child B is born a year later in January 1999. Child A is then 36 months old in January 2001 and Child B is 36 months old in January 2002. When these two children are the same age (i.e. 36 months old), they experience different contemporaneous pollution exposures and they have experienced different pollution exposure histories over the previous year. In sum, they may experience similar background characteristics but differ in the probability of illness at a given age (i.e. 36 months old) due to differences in pollution exposure.

### 4.2 Estimation Notes

Estimation of all specifications involves very large numbers of observations. Our primary analysis sample consists of 328,920 children for whom detailed individual covariates are observed. Each child is observed over 60 months each, yielding 19,739,160 observations in total. A larger robustness sample consists of 681,958 children for whom fewer individual covariates are observed. Each child is again observed over 60 months each, yielding 40,917,480 observations in total. All specifications involve thousands of fixed effects.

We estimate a linear probability (LP) model, largely for reasons of computational tractability. Our goal is to assess the marginal effects of pollution on children's health outcomes. While non-linear models, such as the logit or probit, may more accurately fit the conditional expectation function, linear probability and non-linear models frequently generate very similar marginal effects (Angrist and Pischke 2009; Angrist and Evans 1998). An additional advantage is that we are not required to arbitrarily choose a non-linear functional form (Deaton 1997).[8]

In order to control for serial correlation within cross-sectional units, as well as the heteroskedasticity that arises in linear probability models, we cluster all standard errors at the MSOA-level. Large sample sizes imply considerable statistical power, i.e. null hypotheses are particularly easy to reject (McCloskey and Ziliak 1996). To this end, we base all inference on a 1 percent level of significance.

## 5. Results

Regression results, corresponding to specifications (1) through (3), are presented in Tables 3-5. Results in Tables 3-5 come from analysis of the 328,920 children (19,739,160 observations) for whom we observe more complete health-at-birth information. As discussed later in the section, key results are robust to the larger but less complete sample of 681,958 children (40,917,480 observations).

Before interpreting our key pollution results, we note the impact of control variables. Older children have far fewer respiratory treatments than younger children. Boys are more likely to be treated for illnesses of the respiratory system than girls. As expected, infants born to younger mothers, after longer gestation periods, and with higher birthweight have fewer respiratory treatments during childhood. Weather variables are statistically significant in some

[8] An alternative approach to an LP model is to use case control sampling and estimate a probit, logit, or other non-linear model. However, Knittel et al. (2009) demonstrated that pollution and health relationships are highly sensitive to case control choices, and case control estimates can be challenging to interpret.

specifications and not statistically significant in others. When significant, temperature is positively associated with respiratory treatments, relative humidity is negatively associated with respiratory treatments, and wind speed is positively associated with respiratory treatments.

**5.1 Effects of Contemporaneous Pollution Exposure**

Columns 1, 3, and 5 of Tables 3-5 present contemporaneous exposure results for pollutants evaluated separately. In all specifications with pollutants considered individually, increases in CO, PM10, and O3 exposure are positively associated with contemporaneous increases in children's respiratory treatments. However, only the effects of carbon monoxide (CO) are statistically significant across all specifications. Column 7 of Tables 3-5 reveals the importance of considering pollutants simultaneously. CO and PM10 are positively correlated, and independent estimates of the effects of PM10 on respiratory illness overstate that pollutant's contribution to children's health outcomes. O3 is negatively correlated with both CO and PM10, and independent estimates of the effects of O3 on respiratory illness understate that pollutant's contribution to children's health outcomes.

When contemporaneous effects of pollution exposure are considered simultaneously, we find that both CO and O3 are statistically significant predictors of children's respiratory treatments. It is relatively straightforward to interpret the magnitude of these results. Column [7] results in Tables 3-5 show coefficients on CO exposure range from 0.00019 to 0.00021. Table 2 indicates that the sample baseline probability of respiratory treatment in any given month is 0.00082. The CO coefficients then imply that a ten percent increase in a month's CO pollution increases the average child's probability of respiratory treatment in that month by approximately 2.3 - 2.6 percent.[9] The O3 coefficients imply that a ten percent increase in a month's O3 pollution levels increases the average child's probability of respiratory treatment in that month by approximately 2.2 – 3.3 percent. In contrast to the effects of CO and O3, PM10 coefficients are not statistically significant. On average, point estimates would have to be approximately 1.8 to 2.3 times greater than present magnitudes to be statistically significant. Point estimates currently imply that a ten percent increase in a month's PM10 pollution increases the average child's contemporaneous probability of respiratory treatment in that month by 1.2 percent or less.

[9] (0.00019/0.00082)*10 approximates the percentage effect of a 10% change in CO pollution. Later coefficients are interpreted similarly.

### 5.2 Effects of Pollution Exposure over the Previous Year

Columns 2, 4, and 6 of Tables 3-5 present results for contemporaneous exposure and average monthly exposure over the previous year for pollutants evaluated separately. The only coefficient on exposure over the previous year that is statistically significant across all specifications is the coefficient on average CO exposure 1-12 months ago. Column 8 results, from specifications where pollutants are considered simultaneously, are similar. Again, the only longer-term coefficient that is statistically significant across the three identification approaches is the coefficient on average CO exposure 1-12 months ago. These coefficients range from 0.00055 to 0.00175, suggesting that a ten percent increase in average monthly CO pollution over the past year increases the typical child's probability of a respiratory treatment in a given month by approximately 6.7 – 21.3 percent.[10] Note that this effect occurs above and beyond the contemporaneous effect.

## 6. Sensitivity Analysis

Our results for the effects of contemporaneous CO and O3 on children's respiratory outcomes are robust to three different research designs, each with different strengths and weaknesses. Contemporaneous results are robust to multiple specifications within each design. Results for the effects of CO exposure over the previous year on children's subsequent respiratory outcomes are also robust to multiple research designs and specifications. In this section, we present results from additional sensitivity analyses designed to explore robustness further.

### 6.1 Robustness to Sampling Choices

Our first sensitivity check replicates the analyses in Tables 3 - 5 for the full sample of 681,958 children (40,917,480 observations). This sample contains fewer individual-level controls, but analyzes relationships between pollution and health for approximately twice as many children. Summary results are presented in Tables 6. Results for CO are robust. Interpreting the contemporaneous CO coefficients in columns 2, 4, and 6 implies that a ten percent increase in a month's CO pollution increases the average child's probability of respiratory treatment in that month by approximately 1.0 - 2.7 percent. A ten percent increase in the previous year's average CO pollution increases the typical child's probability of a respiratory

[10] Note that, in our specifications, increasing total pollution by ten percent over the past year is equivalent to increasing average monthly pollution over the past year by ten percent.

treatment in a given month by 3.2 – 15.4 percent. Results for the contemporaneous effects of O3 appear to be somewhat less robust. Results are frequently not statistically significant, and magnitudes are systematically smaller. Interpreting the contemporaneous O3 coefficients in columns 2, 4, and 6 implies that a ten percent increase in a month's O3 pollution increases the average child's probability of respiratory treatment in that month by approximately 0.5 - 1.8 percent. Generally, effects of contemporaneous and previous year PM10 are neither statistically significant nor practically important. In the MSOA-by-AGE specification in column 6 of Table 6, we find a statistically significant negative coefficient on cumulative O3 effects. This result is not robust across the many specifications in Tables 3-6. It is possible that co-linearity may make separate identification of both cumulative and contemporaneous effects difficult. Also, avoidance behavior biases coefficients in a negative direction. If especially high cumulative O3 levels caused individuals to stay indoors more frequently, O3 levels could be negatively associated with respiratory health outcomes.

**6.2 Robustness to Falsification Tests**

Our second sensitivity check involves falsification tests that replicate previous analyses for injuries and fractures. Table 7 summarizes falsification test results. We find no evidence of statistically significant relationships between contemporaneous pollution and children's injuries and fractures. Coefficients are practically small in magnitude. Moreover, we find no evidence of statistically significant relationships between pollution exposure over the past year and children's injuries and fractures. These placebo test results suggest that our primary results are unlikely to be driven by omitted variables correlated with both air pollution and general health or health treatment outcomes.

**6.3 Robustness to Pollution Exposure Specifications**

We analyze the health effects of pollution exposure defined by monthly averages. One natural concern is that especially high pollution concentrations, rather than average pollution concentrations, drive health outcomes. We therefore replicated our analysis using monthly maximum, rather than monthly average, exposures as the key contemporaneous explanatory variables.[11] To be precise, max PM10 measures represent the highest daily mean of PM10 over

[11] An alternative approach involves including both average and maximum contemporaneous exposure variables in the same specification. However, average and maximum exposure measures are sufficiently collinear that separate identification is difficult. Note that we do not replicate exposure over the previous year with maximums, as the goal of analyzing those variables is to pick up persistent effects.

all days in month *t*; max CO measures represent the highest 8-hour running mean of CO over all periods in month *t*; and max O3 measures represent the highest 8-hour running mean of O3 over all periods in month *t*. Table 8 demonstrates that CO results are broadly similar to results using average pollution concentrations. We continue to find a statistically significant and practically meaningful impact of contemporaneous CO exposure on children's respiratory health outcomes. However, we do note that the empirical magnitudes are somewhat smaller, perhaps suggesting that monthly average CO exposures influence health outcomes more than spikes in CO exposures, at least given the support of our data. We find no evidence that monthly maximum PM10 and O3 exposure adversely influences children's respiratory health outcomes.

We also replicated all analyses with pollution exposure variables defined over 20 mile radii, rather than 10-mile radii. Results are qualitatively and quantitatively similar to those in Tables 3-6. This similarity is perhaps not surprising ex-post, as urban pollution monitors are relatively dense in England and as we employ inverse distance weighting for exposure measures.

**6.4 Robustness to Functional Form Choices**

We log pollution exposure variables since the distribution of pollution across space and time is skewed. However, the precise functional relationship between pollution and health is unknown a priori. As a sensitivity analysis of functional form, we replicate all analyses using *levels* of pollution exposure – rather than *logs* of pollution exposure - for all air quality variables. We continue to find robust impacts of both short-term and longer-term CO exposure on children's respiratory treatment outcomes.

**7. Discussion and Conclusion**

What have we learned? We find that: (1) observed health effects of CO and O3 for non-infant children's respiratory health outcomes are significant, and (2) exposure to CO over the previous year has a significant effect on observed children's health that goes above and beyond contemporaneous exposure alone. Since the literature emphasizing the isolation of causal effects of pollution typically focuses on short-term outcomes for infants and adults, we believe our results add to the literature. Existing evidence emphasizing causal influences of criteria air pollution on non-fatal morbidity impacts for non-infant children is limited, existing evidence establishing causal influences of CO on respiratory outcomes is limited, and existing evidence supporting causal impacts of longer-term pollution exposure (especially CO exposure) is limited.

Our results are robust across three distinct research designs that are designed to account for potential socioeconomic, behavioral, seasonality, and economic confounders. Results are also robust to multiple sampling choices, falsification tests, and specification checks. Nevertheless, reliable identification and causal attribution can be difficult at the scale of analysis used in this paper, and we cannot rule out all possible confounding factors or other threats to internal validity. We are unable to identify a national-level natural experiment for our sample period. We know of no instrumental variable that plausibly satisfies exclusion restrictions for an entire nation over a lengthy period. Endogeneity concerns could possibly bias our estimates if omitted factors or measurement errors are correlated with anomalous, rather than typical, pollution outcomes at the highly local-level.

We note other possible limitations to internal validity. First, we do not reliably observe mortality. Pollution may also cause deaths in young children; to the extent that these outcomes are important, our current estimates are understated. Second, we do not reliably observe children leaving England or the NHS system. Our research methods generate biased results only if such attrition is correlated with local trends in pollution. Moreover, net emigration from the U.K. is small, averaging less than half a percent of the population during our sample period (UKONS 2012). Third, as is the case for virtually any observational study linking pollution and health, we do not perfectly observe individual-level pollution exposure. The density of air pollution monitors in urban areas of England is high, however, so we are able to examine smaller pollution radii than in many related studies. A similar concern, which is particular to our dataset, is that we observe an individual's exact place of residence only when they come into contact with an NHS facility. In principle, a child that moves could be assigned the pollution exposure of their former MSOA for several periods – i.e. until they have a new health contact. In practice, however, few children move. In the subsample of children for whom we directly observe place of residence late in our sample window, more than 70 percent continue to reside in their MSOA of birth. Further, a stylized fact is that the overwhelming majority of UK moves are local. In our sample, the median observed move was 1.8 miles, as measured from MSOA centroid to MSOA centroid. 95 percent of observed moves were less than 18 miles from centroid to centroid. Across all observed moves, the resulting change in pollution exposure was practically small and statistically zero.

We do not observe adaptive behavior. Unobserved short-run avoidance behavior, such as children remaining indoors on high pollution days, may bias our results downwards relative to idealized estimates. Again, this implies current estimates are conservative. Unobserved long-run avoidance behavior, like relocation within England due to pollution, is important in other contexts but is unlikely to influence results here. As discussed above, moves that significantly change a household's pollution exposure are rare. Given fixed effects specifications (including area-by-year fixed effects), only relocation due to unexpected or atypical within-year / within-area pollution levels could bias our results. If households base location decisions on local average pollution levels, or even changes in local annual pollution levels, our results are unbiased. Further, estimates that are conditional on adaptive behaviors, such as ours, may be most directly relevant for some policy purposes. Our estimates capture the net effect of pollution in a real world complicated by human behavior. This differs from the effect of pollution one might observe in a fully controlled human exposure or toxicological study.[12]

We also note caveats to external validity. First, we only model relationships between pollution and health outcomes for children living in urban and suburban areas. Results should not be extrapolated to children living in rural areas. Second, England is a developed country. Results should not be extrapolated to less developed nation contexts. Third, we observe lower average pollution concentrations than exist in many other urban areas of the developed world. For example, the average pollution concentrations in our sample are 20 to 50 percent of U.S. pollution concentrations over the same period. Results should be extrapolated to urban areas of other developed nations with caution.

Other cautionary notes relate to our specific exposure measures. First, variable definitions are constrained by observation choices made by UK air quality authorities over our sample period. While we find no statistically significant link between PM10 and health outcomes, it is possible that unobserved finer particulates such as PM2.5 significantly affect children's respiratory health. Our observed health reactions to carbon monoxide may be driven by high correlations between CO and unmeasured toxic air pollutants also common in vehicle emissions.[13] Second, our research design does not permit an evaluation of the full effects of

[12] We thank a helpful reader for noting this interpretation.

[13] This distinction is less important from a policy perspective, however, as most public programs and control technologies targeting CO are likely to reduce correlated air toxics as well.

cumulative lifetime pollution exposure on children's respiratory health. Separately identifying cumulative lifetime pollution exposure with our individual-level fixed effects or our cohort-by-MSOA fixed effects is not possible. So, while we do contribute original evidence on longer-term causal relationships between pollution and children's respiratory health, our model may miss some potentially important truly long-run effects of pollution on children's respiratory health.

Subject to the above caveats, our results imply that a ten percent increase in a month's CO pollution increases the average child's probability of respiratory treatment in that month by approximately 2.3 - 2.6 percent. A ten percent increase in average monthly CO pollution over the past year also increases the typical child's probability of a respiratory treatment in a given month by approximately 6.7 – 21.3 percent. How large are these detected impacts? The baseline number of monthly respiratory hospitalizations for our ~700,000 sample children was ~575 (0.00082 treatments per child per month). On average, carbon monoxide levels declined by approximately 7-10% each year throughout urban areas of England between 1997 and 2006. As an approximate guide to the magnitude of our results, consider a thought experiment in which the average annual CO reduction occurred in a single day. Starting immediately, results suggest that sample children's respiratory hospitalizations would decline by roughly 10 – 15 per month due to the change in acute pollution exposure, ceteris paribus. One year later, results suggest that sample children's respiratory hospitalizations would be an additional 20 – 60 cases per month lower due to the change in longer-term exposure, ceteris paribus.

Our analysis investigates the impact of pollution on respiratory outcomes that are severe enough to warrant hospital or clinical treatment; impacts for less severe respiratory outcomes are unobserved. Further, calculations in the preceding paragraph extrapolate regression estimates to a non-marginal context, do not fully account for transition dynamics, and apply only to a hypothetical one-time average pollution change. Relationships between health outcomes and costs of treatment, pain and suffering, and long-term human capital costs are complex. Thus, addressing the full welfare effects of pollution on children's respiratory health is beyond the scope of this study. Nevertheless, our results do suggest that the understudied influence of criteria air pollutants on non-infant children's respiratory health may be important. Further, our results are derived from a research setting where average air pollution concentrations are low relative to many urban areas of the developed world. This may suggest that the gross benefits of

pollution reduction programs may remain high even as pollution continues to decline in the United States and elsewhere.

.

**References**


Angrist, J.D. and W.N. Evans. “Children and their Parent’s Labor Supply: Evidence from Exogenous Variation in Family Size.” *American Economic Review*. 88: 450-477. 1998.

Angrist, J.D. and J. Pischke. *Mostly Harmless Econometrics: An Empiricist’s Companion*. Princeton, NJ: Princeton University Press. 2009.

Banzhaf, H.S. and R.P. Walsh. “Do People Vote with their Feet? An Empirical Test of Tiebout’s Mechanism.” *American Economic Review*. 98(3): 843-863. 2008.

Barreca, A. "Climate Change, Humidity, and Mortality in the United States." *Journal of Environmental Economics and Management*. 63(1): 19-34. 2012.

Bayer, P., N. Keohane, and C. Timmins. “Migration and Hedonic Valuation: The Case of Air Quality.” *Journal of Environmental Economics and Management*. 58(1): 1-14. 2009.

Beatty, T.K.M. and J.P. Shimshack. “School Buses, Diesel Emissions, and Respiratory Health.” *Journal of Health Economics*. 30(5): 987-999. 2011.

Chay, K.Y., C. Dobkin, and M. Greenstone, “The Clean Air Act of 1970 and Adult Mortality,” *Journal of Risk and Uncertainty*. 27(3): 279-300. 2003.

Chay, K.Y. and M. Greenstone. “The Impact of Air Pollution on Infant Mortality: Evidence from geographic variation in pollution shocks induced by a recession,” *Quarterly Journal of Economics*. 118(3): 1121-1167. 2003.

Clancy, L., et al. “Effect of air pollution control on death rates in Dublin, Ireland: an intervention study,” *Lancet*. 360(9341): 1210-1214. 2002.

Committee on Environmental Health, “Ambient Air Pollution: Health Hazards to Children,” *Pediatrics.* 114: 1699-1707. 2004.

Currie, J. “Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development,” *Journal of Economic Literature*. 47(1): 87-122. 2009.

Currie, J. and M. Neidell, “Air Pollution and Infant Health: What can we learn from California’s recent experience?” *Quarterly Journal of Economics.* 120(3):1003-1030. 2005.

Currie, J. and W.R. Walker, “Traffic Congestion and Infant Health: Evidence from E-ZPass,” *American Economic Journal: Applied Economics*, 3(1): 65-90. 2011.

Currie et al., “Air Pollution and Infant Health: Lessons from New Jersey,” *Journal of Health Economics*, 28(3): 688-703. 2009.

Deaton, A. *The Analysis of Household Surveys: A Microeconomic Approach to Development Policy*. Baltimore, MD: Johns Hopkins University Press for the World Bank. 1997.

Dockery, D.W., et al., "An association between air pollution and mortality in 6 U.S. cities," *New England Journal of Medicine,* 329(24): 1753-1759. 1993.

Dominici, F., et al., "Health Effects of Air Pollution: A Statistical Review," *International Statistical Review,* 71(2): 243-276.

Evans, M. and V.K. Smith, "Do New Health Conditions Support Mortality-Air Pollution Effects?" *Journal of Environmental Economics and Management* 50: 496-518. 2005.

Gauderman, W.J. et al., "Association between air pollution and lung function growth in southern California children." *American Journal of Respiratory and Critical Care Medicine*. 162(4): 1383-1390. 2000.

Gauderman, W.J., et al., "Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study," *Lancet* 369: 571-577. 2007.

Janke, K., C. Propper, and J. Henderson, "Do Current Levels of Air Pollution Kill? The Impact of Air Pollution on Population Mortality in England," *Health Economics* 18(9): 1031-1055. 2009.

Jarrett, M., et al, "Long-term Ozone Exposure and Mortality," *New England Journal of Medicine* 360(11): 1085-1095. 2009.

Knittel, C., D. Miller, and N. Sanders, "Caution, Drivers! Children Present. Traffic, Pollution, and Infant Health," UC Davis Working Paper. 2009.

Krupnick, A, W. Harrington, and B. Ostro, "Ambient Ozone and Acute Health Effects: Evidence from Daily Data," *Journal of Environmental Economics and Management* 18: 1-18. 1990.

Lleras-Muney, A. "The Needs of the Army: Using Compulsory Relocation in the Military to Estimate the Effect of Air Pollutants on Children's Health," *Journal of Human Resources,* 45(3): 549-590. 2010.

Lowen AC, Mubareka S, Steel J, et al. Influenza virus transmission is dependent on relative humidity and temperature. *PLoS Pathog* 2007;3(10):1470-76.

Lowen AC, Steel J, Mubareka S, et al. High temperature (30 degrees C) blocks aerosol but not contact transmission of influenza virus. *J Virol* 2008;82(11):5650-52.

McCloskey, D. and S.T. Ziliak, "The Standard Error of Regressions," *Journal of Economic Literature,* 34: 97-114. 1996.

Moretti, E. and M. Neidell, "Pollution, Health, and Avoidance Behavior: Evidence from the Ports of Los Angeles," *Journal of Human Resources*, 46(1): 154-175. 2011.

Neidell, M. "Air Pollution, Health, and Socio-Economic Status: the Effect of Outdoor Air Quality on Childhood Asthma," *Journal of Health Economics*. 23: 1209-1236. 2004.

Peters, J.M., et al., "A study of twelve California communities with differing levels and types of air pollution," *American Journal of Respiratory Critical Care Medicine*, 159(3): 760-767. 1999.

Peters, J.M. et al., "Epidemiological investigation to identify chronic effects of ambient air pollutants in Southern California". California Air Resources Board and the California Environmental Protection Agency, Contract No. 94-331. 2004.

Pope, C.A. "Respiratory disease associated with community air pollution and a steel mill, Utah Valley," *American Journal of Public Health,* 79(5): 623-628. 1989.

Pope, C.A. "Particulate Pollution and Health: A Review of the Utah Valley experience," *Journal of Exposure Analysis and Environmental Epidemiology* 6(1): 23-34. 1996.

Pope, C.A. et al., "Daily Mortality and PM10 Pollution in Utah Valley." *Archives of Environmental Health*, 47(3): 211-217.

Pope, C.A., et al., "Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults," *American Journal of Respiratory Critical Care Medicine*, 151(3): 669-674. 1995.

Pope, C.A., et al., "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution," *Journal of the American Medical Association*, 287(9): 1132-1141. 2002.

Raub, J. and V. Benignus, "Carbon monoxide and the nervous system," *Neuroscience and Biobehavioral Reviews* 26: 925–940. 2002.

Samet, J.M. et al., "Fine particulate air pollution and mortality in 20 U.S. cities, 1987-1994," *New England Journal of Medicine* 343(24): 1742-1749. 2000.

Schlenker, W. and W.R. Walker, "The Effect of Airports on Air Quality and Respiratory Problems," *Columbia University Working Paper.* 2011.

Seaton, A. et al., “Particulate Air Pollution and Acute Health Effects,” The Lancet 354: 176-178. 1995.

Shaman J, Kohn M. Absolute humidity modulates influenza survival, transmission, and seasonality. *Proc Natl Acad Sci* 106(9):3243-48. 2009.

Sheffield, P., et al. “Fine particulate matter pollution linked to respiratory illness in infants and increased hospital costs,” *Health Affairs*, 30(5): 871-878. 2011.

Spix, C. et al., “Short-Term Effects of Air Pollution on Hospital Admissions of Respiratory Diseases in Europe: A Quantitative Summary of APHEA Study Results,” *Archives of Environmental Health*. 53(1): 54-64. 1998.

UKONS, “Migration Statistics Quarterly Report May 2012.” May 24, 2012.

USEPA, “Air Quality Criteria for Ozone and Related Photochemical Oxidants,” EPA 600/R-05/004aF. February 2006.

USEPA, “Integrated Science Assessment for Particulate Matter,” EPA/600/R-08/139F. 2009.

USEPA, “Integrated Science Assessment for Carbon Monoxide (Final Report),” EPA/600/R-09/019F, 2010.

USEPA, “Our Nation’s Air: Status and Trends through 2010,” EPA/454/R-12/001. February 2012.

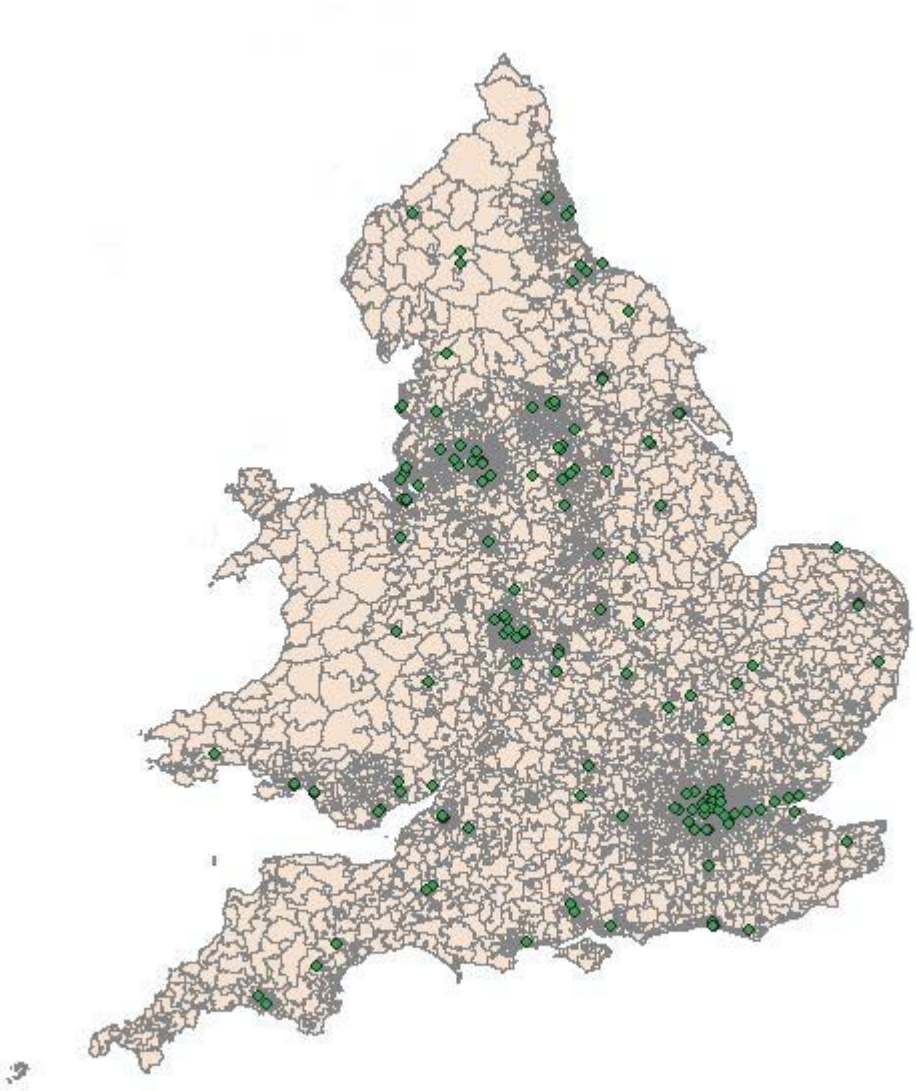

**Figure 1.** MSOAs and air quality monitors in England.


**Figure 2.** Trends in Pollution Over Time

**Table 1. Pollution Summary Statistics**

| Variable | Mean | Std. Dev | Between MSOA std. dev. | Within MSOA std. dev. |
|---|---|---|---|---|
| Monthly Average CO (mg/m3) | 0.71 | 0.35 | 0.20 | 0.29 |
| Monthly Average PM10 (µg/m3) | 25.6 | 4.85 | 2.90 | 3.88 |
| Monthly Average O3 (µg/m3) | 53.4 | 15.7 | 5.0 | 14.9 |

NOTES: Data observed at the MSOA by month level.

**Table 2. Full Summary Statistics**

| | Sample w/ Covariates (328,920 children) | | Full Sample (681,958 children) | |
|---|---|---|---|---|
| Variable | Mean | Std. Dev | Mean | Std. Dev. |
| Respiratory Treatments (#/1000) | 0.85 | 29.1 | 0.82 | 28.7 |
| Monthly Average CO (mg/m3) | 0.71 | 0.33 | 0.73 | 0.34 |
| Monthly Average PM10 (µg/m3) | 25.6 | 5.1 | 25.7 | 5.1 |
| Monthly Average O3 (µg/m3) | 52.6 | 16.7 | 52.5 | 15.7 |
| Monthly Mean Temp. (°C) | 10.98 | 4.85 | 10.88 | 4.82 |
| Monthly Max Temp. (°C) | 19.19 | 6.40 | 18.93 | 6.31 |
| Monthly Mean Humid. (rel. hum.) | 78.60 | 8.31 | 79.07 | 8.15 |
| Monthly Max Humid. (rel. hum.) | 97.77 | 3.07 | 97.93 | 2.93 |
| Monthly Mean Wind (knots) | 7.30 | 2.39 | 7.41 | 2.46 |
| Monthly Max Wind (knots) | 20.53 | 9.09 | 21.03 | 8.92 |
| Age (months) | 53.5 | 17.3 | 53.5 | 17.3 |
| Sex (male 1, female 2) | 1.49 | 0.50 | 1.49 | 0.50 |
| Birthweight (grams) | 3308 | 573 | n/a | n/a |
| Maternal Age at Birth (years) | 28.17 | 5.76 | n/a | n/a |
| Gestation at Birth (weeks) | 39.19 | 2.00 | n/a | n/a |

NOTES: Data observed at the child by month level.

**TABLE 3. Effect of Pollution on Children's Respiratory Treatments**
**Individual-Level Fixed Effect Specifications (Equation 1)**

| | [1] | [2] | [3] | [4] | [5] | [6] | [7] | [8] |
|---|---|---|---|---|---|---|---|---|
| Log (mean CO this month) | 0.00019* | 0.00016* | | | | | 0.00019* | 0.00014* |
| | (0.00004) | (0.00005) | | | | | (0.00005) | (0.00005) |
| Log (mean CO 1-12 months ago) | | 0.00051* | | | | | | 0.00055* |
| | | (0.00011) | | | | | | (0.00011) |
| Log (mean PM this month) | | | 0.00012 | 0.00011 | | | 0.00009 | 0.00012 |
| | | | (0.00006) | (0.00006) | | | (0.00007) | (0.00007) |
| Log (mean PM 1-12 months ago) | | | | -0.00031 | | | | -0.00040 |
| | | | | (0.00019) | | | | (0.00021) |
| Log (mean O3 this month) | | | | | 0.00021* | 0.00021* | 0.00027* | 0.00028* |
| | | | | | (0.00006) | (0.00006) | (0.00007) | (0.00007) |
| Log (mean O3 1-12 months ago) | | | | | | -0.00008 | | 0.00016 |
| | | | | | | (0.00018) | | (0.00021) |
| Region-specific Time Trends | YES | YES | YES | YES | YES | YES | YES | YES |
| Piecewise Linear Spline in Age | YES | YES | YES | YES | YES | YES | YES | YES |
| Weather Variables | YES | YES | YES | YES | YES | YES | YES | YES |
| Month of Year Dummies | YES | YES | YES | YES | YES | YES | YES | YES |
| Year Dummies | YES | YES | YES | YES | YES | YES | YES | YES |
| Individual-Level Fixed Effects | YES | YES | YES | YES | YES | YES | YES | YES |
| Observations | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 |

Notes: * indicates statistical significance at the 1 percent level.

**TABLE 4. Effect of Pollution on Children's Respiratory Treatments**
**MSOA-by-YEAR Fixed Effect Specifications (Equation 2)**

| | [1] | [2] | [3] | [4] | [5] | [6] | [7] | [8] |
|---|---|---|---|---|---|---|---|---|
| Log (mean CO this month) | 0.00022* | 0.00027* | | | | | 0.00021* | 0.00026* |
| | (0.00005) | (0.00005) | | | | | (0.00005) | (0.00005) |
| Log (mean CO 1-12 months ago) | | 0.00181* | | | | | | 0.00175* |
| | | (0.00025) | | | | | | (0.00025) |
| Log (mean PM this month) | | | 0.00017* | 0.00019* | | | 0.00010 | 0.00013 |
| | | | (0.00006) | (0.00006) | | | (0.00007) | (0.00007) |
| Log (mean PM 1-12 months ago) | | | | 0.00039 | | | | 0.00033 |
| | | | | (0.00022) | | | | (0.00030) |
| Log (mean O3 this month) | | | | | 0.00011 | 0.00011 | 0.00018* | 0.00013 |
| | | | | | (0.00007) | (0.00007) | (0.00007) | (0.00007) |
| Log (mean O3 1-12 months ago) | | | | | | -0.00038 | | -0.00040 |
| | | | | | | (0.00022) | | (0.00030) |
| Individual Controls | YES | YES | YES | YES | YES | YES | YES | YES |
| Piecewise Linear Spline in Age | YES | YES | YES | YES | YES | YES | YES | YES |
| Weather Variables | YES | YES | YES | YES | YES | YES | YES | YES |
| Month of Year Dummies | YES | YES | YES | YES | YES | YES | YES | YES |
| Year Dummies | YES | YES | YES | YES | YES | YES | YES | YES |
| MSOA -by- YEAR Fixed Effects | YES | YES | YES | YES | YES | YES | YES | YES |
| Observations | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 |

Notes: * indicates statistical significance at the 1 percent level.

**TABLE 5. Effect of Pollution on Children's Respiratory Treatments**
**MSOA-by-AGE Fixed Effect Specifications (Equation 3)**

| | [1] | [2] | [3] | [4] | [5] | [6] | [7] | [8] |
|---|---|---|---|---|---|---|---|---|
| Log (mean CO this month) | 0.00021* | 0.00019* | | | | | 0.00021* | 0.00020* |
| | (0.00005) | (0.00005) | | | | | (0.00005) | (0.00005) |
| Log (mean CO 1-12 months ago) | | 0.00069* | | | | | | 0.00066* |
| | | (0.00014) | | | | | | (0.00014) |
| Log (mean PM this month) | | | 0.00014 | 0.00014 | | | 0.00008 | 0.00009 |
| | | | (0.00006) | (0.00006) | | | (0.00007) | (0.00007) |
| Log (mean PM 1-12 months ago) | | | | -0.00010 | | | | -0.00002 |
| | | | | (0.00021) | | | | (0.00024) |
| Log (mean O3 this month) | | | | | 0.00013 | 0.00013 | 0.00020* | 0.00020* |
| | | | | | (0.00006) | (0.00006) | (0.00007) | (0.00007) |
| Log (mean O3 1-12 months ago) | | | | | | -0.00057* | | -0.00046 |
| | | | | | | (0.00020) | | (0.00023) |
| Individual Controls | YES | YES | YES | YES | YES | YES | YES | YES |
| Piecewise Linear Spline in Age | YES | YES | YES | YES | YES | YES | YES | YES |
| Weather Variables | YES | YES | YES | YES | YES | YES | YES | YES |
| Month of Year Dummies | YES | YES | YES | YES | YES | YES | YES | YES |
| Year Dummies | YES | YES | YES | YES | YES | YES | YES | YES |
| MSOA -by- AGE Fixed Effects | YES | YES | YES | YES | YES | YES | YES | YES |
| Observations | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 | 19,739,160 |

Notes: * indicates statistical significance at the 1 percent level.

**TABLE 6. Robustness: Sample Selection**
**Full Sample without Individual-Level Control Variables**

| | Specifications with Individual-Level FEs | | Specifications with MSOA-by-YEAR FEs | | Specifications with MSOA-by-AGE FEs | |
|---|---|---|---|---|---|---|
| | [1] | [2] | [3] | [4] | [5] | [6] |
| Log (mean CO this month) | 0.00011* | 0.00008* | 0.00017* | 0.00022* | 0.00021* | 0.00018* |
| | (0.00003) | (0.00003) | (0.00003) | (0.00003) | (0.00003) | (0.00003) |
| Log (mean CO 1-12 months ago) | | 0.00026* | | 0.00126* | | 0.00065* |
| | | (0.00007) | | (0.00016) | | (0.00009) |
| Log (mean PM this month) | 0.00004 | 0.00006 | 0.00004 | 0.00006 | 0.00001 | 0.00001 |
| | (0.00004) | (0.00004) | (0.00004) | (0.00004) | (0.00004) | (0.00004) |
| Log (mean PM 1-12 months ago) | | -0.00012 | | 0.00037 | | 0.00010 |
| | | (0.00013) | | (0.00020) | | (0.00015) |
| Log (mean O3 this month) | 0.00015* | 0.00015* | 0.00008 | 0.00004 | 0.00007 | 0.00005 |
| | (0.00005) | (0.00005) | (0.00005) | (0.00005) | (0.00005) | (0.00005) |
| Log (mean O3 1-12 months ago) | | 0.00011 | | -0.00055 | | -0.00059* |
| | | (0.00014) | | (0.00022) | | (0.00017) |
| Piecewise Linear Spline in Age | YES | YES | YES | YES | YES | YES |
| Weather Variables | YES | YES | YES | YES | YES | YES |
| Month of Year Dummies | YES | YES | YES | YES | YES | YES |
| Year Dummies | YES | YES | YES | YES | YES | YES |
| Observations | 40,917,360 | 40,917,360 | 40,917,360 | 40,917,360 | 40,917,360 | 40,917,360 |

Notes: * indicates statistical significance at the 1 percent level.

**TABLE 7. Robustness: Placebo Tests**
**Effect of Pollution on Children's Treatments for Fractures and Injuries**

| | Specifications with Individual-Level FEs | Specifications with MSOA-by-YEAR FEs | Specifications with MSOA-by-AGE FEs |
|---|---|---|---|
| Log (mean CO this month) | -0.00004 | -0.00002 | -0.00004 |
| | (0.00003) | (0.00003) | (0.00003) |
| Log (mean CO 1-12 months ago) | 0.00007 | 0.00013 | 0.00023 |
| | (0.00006) | (0.00007) | (0.00014) |
| Log (mean PM this month) | -0.00004 | -0.00005 | -0.00001 |
| | (0.00004) | (0.00004) | (0.00004) |
| Log (mean PM 1-12 months ago) | 0.00013 | 0.00011 | 0.00022 |
| | (0.00010) | (0.00013) | (0.00016) |
| Log (mean O3 this month) | -0.00005 | -0.00006 | -0.00006 |
| | (0.00004) | (0.00004) | (0.00004) |
| Log (mean O3 1-12 months ago) | -0.00011 | -0.00020 | -0.00029 |
| | (0.00011) | (0.00014) | (0.00017) |
| Piecewise Linear Spline in Age | YES | YES | YES |
| Weather Variables | YES | YES | YES |
| Month of Year Dummies | YES | YES | YES |
| Year Dummies | YES | YES | YES |
| Observations | 19,739,160 | 19,739,160 | 19,739,160 |

Notes: * indicates statistical significance at the 1 percent level.

**TABLE 8. Robustness: Specification**
**Effect of Maximum Pollution Exposure on Children's Respiratory Treatments**

| | Specifications with Individual-Level FEs | | Specifications with MSOA-by-YEAR FEs | | Specifications with MSOA-by-AGE FEs | |
|---|---|---|---|---|---|---|
| | [1] | [2] | [3] | [4] | [5] | [6] |
| Log (maximum CO this month) | 0.00013* | 0.00010* | 0.00013* | 0.00012* | 0.00013* | 0.00013* |
| | (0.00003) | (0.00002) | (0.00003) | (0.00002) | (0.00003) | (0.00002) |
| Log (maximum PM this month) | -0.00002 | 0.00001 | -0.00001 | 0.00002 | -0.00002 | -0.00001 |
| | (0.00004) | (0.00002) | (0.00004) | (0.00002) | (0.00004) | (0.00002) |
| Log (maximum O3 this month) | 0.00009 | -0.00001 | -0.00002 | -0.00009 | -0.00001 | -0.00008 |
| | (0.00006) | (0.00004) | (0.00006) | (0.00004) | (0.00006) | (0.00004) |
| Additional Individual Controls | - | - | YES | NO | YES | NO |
| Piecewise Linear Spline in Age | YES | YES | YES | YES | YES | YES |
| Weather Variables | YES | YES | YES | YES | YES | YES |
| Month of Year Dummies | YES | YES | YES | YES | YES | YES |
| Year Dummies | YES | YES | YES | YES | YES | YES |
| Observations | 19,738,020 | 40,918,740 | 19,738,020 | 40,918,740 | 19,738,020 | 40,918,740 |

Notes: * indicates statistical significance at the 1 percent level.