Forthcoming, with minor changes, in the *Journal of Environmental Economics and Management*

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

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September 2013

ABSTRACT

This paper uses a large 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 hundreds of thousands of English 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 and Mark Wilson for technical assistance with the Alcuin Research Resource Centre cluster computing facility. We thank Associate Editor Chris Timmins and helpful referees for comments. We thank Spencer Banzhaf, Mary Evans, Don Fullerton, Dan Kaffine, Andrew Jones, Laure de Preux Gallone, Nigel Rice, Nick Sanders, 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, the University of Washington, the University of Maryland, Resources for the Future, and the NBER. Jason Edgar provided excellent research assistance.
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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 several 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). Pollution effects for children may be long lasting as early-life illness may impede long-term human capital development (Currie 2009). Finally, economic costs for children's respiratory illnesses are large. The CDC estimates that treatment costs alone amount to several billion dollars annually in the U.S.

Our analysis makes several contributions. First, our dataset is unusually large and detailed. We observe several years of early-life health treatments for hundreds of thousands of children (more than *328,000* children in one sample and *680,000* children in another sample). 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

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.

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.

2.3 Contribution

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 use a broad and quasi-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. We study relationships between pollution and non-fatal health outcomes for non-infant children.

Why might the impact of pollution exposure over the past year be of interest? Animal toxicology, in vitro mechanistic, and limited controlled human exposure studies 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).

Why might the impact of pollution on young children differ from the impacts of pollution on other groups? Pollution effects on children are likely driven by direct exposure, whereas pollution-caused infant mortality, preterm birth, and low birth weight likely reflect placental function or maternal health channels. Young children spend more time outdoors, exhibit greater activity levels, experience higher and more variable breathing rates, and display lower nasal particle deposition rates than most other age groups. Children also have lower body weight and less lung surface area than adults. Respiratory development, primarily through alveoli formation and cell differentiation, is especially rapid during early childhood (Dietert et al. 2000). Young children may be more susceptible to changes in lung function, cell proliferation, airway inflammation, and pulmonary injury than other subpopulations (USEPA 2006, USEPA 2009, USEPA 2010). Children are especially susceptible to viral conditions like respiratory syncytial virus (RSV) and chronic conditions like asthma, and pollution effects can aggravate or interact with these other respiratory conditions. In short, dose-response relationships for children may differ from those of other age groups.

Another reason the welfare impacts of pollution on young children may differ from the impact of pollution on other groups is that long-term consequences of health shocks may be especially large for young children. As surveyed in Currie (2009) and elsewhere, a growing literature suggests that childhood health can influence future labor supply and productivity in at least three key ways. First, poor child health may be associated with later poor adult health. Second, poor child health can have a direct effect on cognitive ability and neuro-behavioral development. Third, poor child health can have an indirect effect on skill acquisition via school absences and ability to learn while in school.

3. Data

To analyze the relationship between pollution and children's morbidity, we constructed an individual-by-month panel. Time invariant individual characteristics were not aggregated. Each individual's health outcomes were summed over days in the month. Monthly pollution and weather exposure data were 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 collected 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.[2] We first obtained birth records for children born in England between 1997 and 1999.[3] For 1.13 million of these births, or about 2/3 of total births in England over the time period, we observed an individual identifier, date of birth, and MSOA of residence at birth.[4] For about 50 percent of the birth records, we also observed sex, weeks of gestation at birth, birth weight in grams, and maternal characteristics.

We then obtained 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 observed health treatments from June 2001 through June 2006. We do 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

[2] NHS treatment centers are specialized diagnostic and treatment facilities. These facilities are often located on the grounds of full NHS hospitals and are designed to lower costs and wait times for scheduled, short-stay elective procedures (Bate et al. 2006). While services vary somewhat across facilities, NHS treatment centers handle inpatient and day cases. Diagnoses and procedures at NHS treatment centers are considerably more serious than seeing a general practitioner doctor. One can think of NHS treatment centers as "specialty hospitals."

[3] Cohort start dates were largely determined by data availability.

[4] 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.

outcomes.[5] We do not examine childhood NHS contacts after age 7 due to data availability and because age 6 or younger corresponds closely with common physiological definitions of "young children."

Since our focus is on air pollution and children's health, we analyze diagnosis codes related to diseases of the respiratory system (ICD-10 codes beginning with "J"). In our main analyses, outcome variables are defined over all such diseases. Related discharges include those related to acute upper respiratory infections (including sinutis), influenza and pneumonia, acute lower respiratory infections such as acute bronchitis and acute bronchiolitis, 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. When these diseases are included in outcome variables, potential measurement error conservatively biases our pollution impacts towards zero and reduces statistical precision.

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.[6] 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

[5] 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.

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

calculate a weighted pollution concentration for every MSOA-pollutant-hour using the weights 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 obtain raw data from the British Atmospheric Data Centers' MIDAS Land Surface Station database.[7] Weather data are observed from hundreds of weather stations (the exact number varies over time). Sites are distributed so that no station is more than roughly 50km from another station, and spatial coverage is especially high in urban areas (where our sample children are predominantly located). Sites are intended to be representative of the area around them. Observations on temperature and humidity are typically observed at the hourly level. We assign these data to local areas (MSOAs) on a monthly basis using a similar procedure to the one used to assign pollution concentrations to local area by month combinations. First, we identify the population-weighted center of each and every MSOA. Second, for each MSOA-weather metric-day combination, we identify all reporting stations within a 10 mile radius of the identified population-weighted centroid. Third, we assign each station a weight proportional to the inverse of its distance from the MSOA center. We calculate these weights daily, since some stations do not measure all weather metrics for all sample days. Fourth, we calculate a weighted weather metric for every MSOA-hour combination using the weights from step 3. Fifth, we calculate the monthly mean over the hourly measures to obtain weather observations for each MSOA-month combination. Final variables include monthly average temperature, monthly maximum temperature, monthly average humidity, and monthly maximum humidity.

3.3 Pollution summary statistics

The top panel of Table 1 summarizes overall pollution. 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 52.6 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.

[7] A detailed description of the dataset is available at: http://badc.nerc.ac.uk/data/ukmo-midas/ukmo_guide.html .

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 considerably. The top panel of Table 1 indicates that overall pollutant standard deviations were approximately 20 to 50 percent of mean pollution levels. The latter columns of the top panel suggest that dispersion is driven by variability across geographic areas and variability within areas across time. The middle panel of Table 1 explores seasonal variation. Seasonal peaks in CO occur in the fall and winter, when average levels are approximately 60 percent larger than average levels in the spring and summer. In contrast, seasonal peaks in O3 occur in spring and summer, when average levels are approximately 50 percent larger than average levels in the fall and winter. PM10 exhibits no strong seasonality.

Figure 2 graphically depicts longer-run temporal variation. CO displays a clear long-term downward trend over our sample period. The quarterly high in CO of 1.24 mg/m3 occurred in quarter 1 of 2001 while the quarterly low of 0.39 mg/m3 in quarter 3 of 2006. In contrast, O3 increased slightly on average over the sample period. The quarterly high in O3 of 74.4 µg/m3 occurred in quarter 2 of 2006 while the quarterly low of 33.3 µg/m3 occurred in quarter 4 of 2002. PM10 exhibited no obvious long-term trend over sample periods.

The bottom panel of Table 1 explores regional variability. Here, regions are defined following 1996 U.K. standard government office region conventions. Some regions of England, including the North East, the North West, Merseyside and the West Midlands, had relatively lower average CO levels of about 0.5 mg/m3. Other regions, including London and the South West, had relatively higher average CO levels of about 0.9 mg/m3. Regional variability in O3 was also observable, but proportionately somewhat lower than for CO. Some regions of England, including the North West, Yorkshire, the East Midlands, and London, had relatively lower average O3 levels of around 50 µg/m3. Other regions, including the North East and Merseyside,

had relatively higher average O3 levels of around 60 μg/m3 or higher. Regional variability in PM10 was small.

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 682,305 children, of which 320,082 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.

On average, 0.85 of 1000 children experienced a contact with an NHS hospital for diseases of the respiratory system in a given month. Table 3 shows that adverse health outcomes were strongly negatively correlated with child age. The mean of the outcome variable was 1.26

for 2 years olds and 0.50 for 6 year olds, and respiratory treatments fall monotonically as age increases. The mean of the outcome variable for boys was 1.02 while the mean of the outcome variable for girls is 0.67. The North West, Yorkshire and the Humber, and East Midlands regions have relative high mean children's respiratory treatment rates, in the range of 1.55 – 1.84. North East, London, Eastern, and South West regions have relative low mean children's respiratory treatment rates, in the range of 0.43 – 0.63.

In the data, we observe relatively few cases of children experiencing many repeat visits. If we restrict attention to the subsample of children treated at an NHS facility for diseases of the respiratory system at least once while in our dataset (i.e. between their 2nd and 7th birthdays), we find that 80% of the sample had one and only one respiratory treatment. 14% had two respiratory treatments, 4% had three respiratory treatments, and less than 3% had four or more respiratory treatments.

4. Relationships between pollution and children's health outcomes

Our basic empirical strategy is to regress children's health outcomes in a given month 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, Depro and Timmins 2012). 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; Barreca and Shimshack 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 an indicator for whether or not child *i* experienced one or more respiratory treatments in month *t.* More formally, our outcome variable is the indicator function $1[\text{treatment}_{it}]$. Outcome variables are initially defined over all respiratory treatments,

corresponding to ICD-10 codes beginning with the block code "J." Later sensitivity analyses disaggregate outcome variables into more narrowly defined diagnosis code groups.

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 month *t.* PM10 and O3 regression specifications are analogous.[8] In order to account for possible correlations between contaminants, we run regressions for all three pollutants and/or all lagged pollution measures simultaneously.

Critical controls include a nonlinear spline in age. The spline allows us to flexibly control for relationships between health and age at different points along the age distribution, but does not require us to impose a specific functional form ahead of time. Our spline is piecewise linear with 15 knots spread evenly over the observed life of each child15 evenly spaced knots (the piecewise linear segments join at ages 27, 31, 35, 39, 43, 47, 51, 55, 59, 63, 67, 71, 75, and 79 months). To us, one of the important advantages of individual-level data is the ability to adequately control for child age. Our summary statistics show strong correlations between child age and respiratory health. According to the US Census Bureau Statistical Abstract, child age is highly correlated with health care utilization and hospitalization more broadly. Age may also be correlated with pollution exposure through time spent outdoors and activity choice. Without flexible controls for age, the potential for omitted variable bias is high.

We also control for observable individual characteristics, seasonality, and common time trends. Observed time invariant individual characteristics include sex, birthweight, mother's age at birth, and gestation at birth; these may be correlated with health outcomes and treatment propensities during childhood and may be correlated with pollution exposure via activity choice and other mechanisms Month-of-year (January? February? etc.) dummy variables account for

[8] 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.

seasonal environmental and economic factors common across all MSOAs and all years, and nest other common approaches like season-of-year (winter? spring? etc.) dummy variables. We account for annual changes in economic activity, general welfare, and respiratory health that are common across MSOAs with year dummy variables (fixed effects) and/or region-specific time trends.

Our final set of explanatory variables control for weather variability. Weather may directly affect morbidity and mortality outcomes via virus survival, virus transmission, activity choice, and other mechanisms (Basu and Samet 2002, Braga et al. 2002, Lowen et al. 2007, Shaman and Kohn 2009, Barreca 2012, Barreca and Shimshack 2012). Weather may also be correlated with pollution through effects on chemical reactions, mixing, dispersal, and dilution. We therefore include variables for monthly average temperature, monthly average humidity, monthly maximum temperature, and monthly maximum humidity. We choose temperature and humidity since they are observed reliably and frequently in the MIDAS database. We focus on monthly averages for consistency with our other variables, for consistency with the related literature, and because fully modeling pollution transport is beyond the scope of the present analysis.[9]

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 an indicator for whether or not child *i* of age *a* living in MSOA *m* had a treatment in month *t* of season *s* and year *y* as:

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

$$(2)\quad 1[treatment_{imtsy}] = P_{mt}\mathrm{B} + X_i\Gamma + A_i(t) + W_{mt}\Upsilon + \alpha_s + \xi_{ym} + \mu_{imtsy}\,.$$

$$(3)\quad 1[treatment_{iamtsy}] = P_{mt}\mathrm{B} + X_i\Gamma + W_{mt}\Upsilon + \eta_y + \omega(t) + \alpha_s + \tau_{am} + \varepsilon_{iamtsy}\,.$$

P_{mt} denotes one or more pollution measures, X_i denotes a vector of time invariant demographic characteristics, $A_i(t)$ denotes a piecewise linear spline in child *i*'s age at time *t*, W_{mt} denotes a vector of weather variables, η_y denotes year dummies (year fixed effects), $\omega(t)$ denotes region-specific linear time trends, α_s denotes month-of-year season dummies (month fixed effects), π_i

[9] Modeling weather with these variables alone is somewhat unsophisticated, but we share the view of Currie and Neidell (2005) that our approach should broadly capture the first-order effects of unusual or unseasonal weather swings that are not already captured by seasonality controls and local area or area-by-year fixed effects. Note that earlier versions of this paper included wind speed measures; including or omitting these factors had no substantive impact on estimated relationships between pollution and health.

denotes individual-level fixed effects, ξ_{ym} denotes MSOA-by-year fixed effects, τ_{am} denotes MSOA-by-age fixed effects, and μ denotes a standard idiosyncratic error term.[10]

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. They also control for time invariant neighborhood characteristics like general economic conditions, access to health care facilities, and local geography. 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 an area's time invariant average socio-demographic characteristics like income, education, and race. They also control for time invariant neighborhood characteristics like general economic conditions, access to health care facilities, and local geography. More notably, MSOA-by-year fixed effects control for unobserved annual shocks common to all individuals within an MSOA. These shocks may affect localized economic activity, health care access, public policy outcomes, and many other factors. 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

[10] 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) as these controls are implicit in those fixed effect specifications.

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.[11]

4.2 Estimation Notes

Estimation of all specifications involves large numbers of observations. Our primary analysis sample consists of 329,082 children for whom detailed individual covariates are observed. Each child is observed over 60 months each, yielding 19,744,920 observations in total. A larger robustness sample consists of 682,305 children for whom fewer individual covariates are observed. Each child is again observed over 60 months each, yielding 40,938,300 observations in total. All specifications involve thousands of fixed effects.

We estimate a linear probability 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).[12]

Within the linear probability model framework, we present multiple specifications for multiple identification strategies. We identify "preferred" specifications, however, using statistical intuition and more formal Bayesian Information Criterion (BIC). Since CO, O3, and PM are often correlated spatially and temporally, we consider specifications as preferred if they that simultaneously assess the impact of all three pollutants. Since evidence is mounting that longer-term pollution exposure impacts may differ from short-term pollution exposure impacts,

[11] 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.

[12] One 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. A second alternative approach would be a duration model, but duration models that allow for multiple spells and censoring while controlling for unobserved heterogeneity can be difficult to interpret, can require strong assumptions, and are computationally intractable given our large dataset.

we consider specifications as preferred if they simultaneously assess the impact of contemporaneous exposure and average exposure over the last year. BIC statistics consistently support our intuition; models with all pollution measures included simultaneously are preferred to alternatives on BIC grounds, even though BIC significantly penalizes adding unimportant information.

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 and null hypotheses can be easy to reject (McCloskey and Ziliak 1996), even when combined with extensive fixed effect structures. 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 4-6. Results in Tables 4-6 come from analysis of the 329,082 children (19,744,920 observations) for whom we observe more complete health-at-birth information. As discussed later in this section, key results are robust to the larger but less complete sample of 682,305 children (40,938,300 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 specifications and not statistically significant in others. When significant, temperature is positively associated with respiratory treatments and relative humidity is negatively associated with respiratory treatments.

5.1 Effects of Contemporaneous Pollution Exposure

Columns 1, 3, and 5 of Tables 4-6 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 4-6 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 4-6 show coefficients on CO exposure range from 0.18 to 0.19. Table 2 indicates that the sample baseline probability of respiratory treatment in any given month is 0.85. 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 2.1 - 2.2 percent.[13] 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 2.5 – 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 more than twice as large 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 0.9 percent or less.

5.2 Effects of Pollution Exposure over the Previous Year

Columns 2, 4, and 6 of Tables 4-6 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 0.53 to 1.73, 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

[13] (0.19/0.82)*10 approximates the percentage effect of a 10% change in CO pollution. Later coefficients are interpreted similarly.

approximately 6.2 – 20.4 percent.[14] 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 4 - 6 for the full sample of 682,305 children (40,938,300 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 7. 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 - 2.4 percent. A ten percent increase in the previous year's average CO pollution increases the typical child's probability of a respiratory treatment in a given month by 3.0 – 15.3 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 0.7 - 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 7, we find a statistically significant negative coefficient on cumulative O3 effects. This result is not robust across the many specifications in Tables 4-7. It is possible that co-linearity may make separate identification of both cumulative and contemporaneous effects difficult. Also, avoidance

[14] 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.

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 as outcome variables. More precisely, we replicate our analysis with a dependent variable that indicates one or more treatments for "injury" by child i in month t. Corresponding ICD-10 codes represent all blocks beginning with "S," including treatments for injuries to the head, neck, back, arm, leg, elbow, shoulder, wrist, ankle, knee, and hip. The mean of the outcome variable is 0.26 and the standard deviation is 16.1.

Table 8 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 treatments.

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.[15] To be precise, max PM10 measures represent the highest daily mean of PM10 over 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 9 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.

[15] 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.

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 4-7. 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 Specification 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.

As noted, our distinct fixed effect specifications have their own strengths and weaknesses. As a sensitivity analysis of research design, we replicated the analysis using specifications with individual-by-year fixed effects. Results are robust. Patterns of statistical significance and practical importance in regressions with individual-by-year fixed effects are similar to those in all other regressions (Tables 4 - 7). Moreover, magnitudes of significant coefficients in regressions with individual-by-year fixed effects lie between the magnitudes of corresponding coefficients in the regressions with individual-level fixed effects and regressions with MSOA-by-year fixed effects. This is perhaps unsurprising ex-post, as results from regressions with individual-by-year fixed effects should be similar to results from regressions with area-by-year fixed effects, provided individuals within local areas are relatively homogeneous.

6.5 Disaggregating Respiratory Disease Classifications

Our analyses consider outcomes defined over all diagnosis codes for diseases of the respiratory system (ICD-10 codes beginning with "J"). Such an aggregation affords us the most statistical variability and corresponds closely to aggregate social welfare considerations. However, the aggregate outcome variables may obscure the individual respiratory ailments

driving the results. We therefore replicated our analyses using more disaggregated diagnoses.[16]

Table 10 present results. We find that short-run CO results appear to be largely driven by impacts on acute upper respiratory infections (including sinusitis) and acute lower respiratory infections (including acute bronchitis and acute bronchiolitis). Our longer-run CO results appear to be largely driven by impacts on acute upper respiratory infections (including sinusitis), acute lower respiratory infections (including acute bronchitis and acute bronchiolitis), and acute and chronic diseases of the upper respiratory tract (including rhinitis). Shorter and longer-run CO results do not appear to be driven by impacts on influenza and pneumonia or chronic lower respiratory diseases including asthma. We also find that short-run O3 results appear to be largely driven by impacts on acute upper respiratory infections (including sinusitis) and influenza and pneumonia. We continue to find no robust and statistically significant relationships between PM-10 and childhood respiratory outcomes.[17]

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,

[16] The relevant ICD-10 codes for these investigations are: J00-J06 for acute upper respiratory infections (including sinutis), J10-J18 for influenza and pneumonia, J20-J22 for other acute lower respiratory infections (including acute bronchitis and acute bronchiolitis), J30-J39 for other diseases of the upper respiratory tract (including rhinitis), and J40-J47 for chronic lower respiratory diseases (including asthma).

[17] In one specification, we find a statistically significant negative coefficient on a pollutant's longer-term exposure variable. These results are not robust across the many specifications in Table 10, nor in the many specifications of Tables 4-9. 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 pollution levels caused individuals to stay indoors more frequently, pollution could plausibly be negatively associated with respiratory health outcomes.

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.[18] 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

[18] Typical weather variables are not suitable as instrumental variables since they may directly influence health and thus violate exclusion restrictions. The atmospheric inversions used as instruments in Arceo-Gomez et al. (2012)'s exploration of pollution in Mexico City are not widespread in the U.K., and thus not available as instruments.

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.[19]

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.[20] 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.[21] Second, our research design does not permit an evaluation of the full effects of cumulative lifetime pollution exposure on children's respiratory health. Separately identifying

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

[20] An additional explanation for failing to reject a null of no significant relationship between PM10 and children's health outcomes is the lack of statistical variability. Summary statistics in Table 1 highlight that PM10 in our sample is proportionally less variable than CO and O3 across both space and time.

[21] 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 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.

This paper suggests promising avenues for future research that are beyond the present scope. First, our analysis estimates the first-order relationships between pollution and young children's respiratory health, but we do not explore the heterogeneity of pollution impacts in detail. A nuanced understanding of the welfare effects of pollution requires evaluating how pollution effects differ across age, income, education, and other demographic and socioeconomic factors. Second, our analysis estimates the aggregate impact of pollution on the total number of children's respiratory health treatments, but we do not evaluate repeat treatments differently than first-time treatments. Repeat treatments are rare in our sample. Nevertheless, completely specified individual-level dynamics represents an important area of the ongoing research agenda exploring pollution and 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 2.1 - 2.2 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 6.2 – 20.4 percent. How large are these detected impacts? The baseline number of monthly respiratory hospitalizations for our ~700,000 sample children was ~595 (0.85 treatments per 1000 children 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 30 – 120 cases per month lower due to the change in longer-term exposure, ceteris paribus.[22]

[22] Few published studies generate results that are directly comparable to our results. Lleras-Muney (2010) finds that annual O3 exposure impacts military children's hospitalizations. That study also finds suggestive, but less definitive, evidence for impacts of annual CO exposure. Impact magnitudes, however, are comparable across studies. In Lleras-Muney (2010), a 15 increase in a year's O3 exposure increases the probability of US children's hospital admissions

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.

by 8-23 percent. We find that a 10 increase in year's CO increases the probability of UK children's hospital admissions by 2-3 percent due to acute effects and an additional 6-20 percent due to longer-term impacts.

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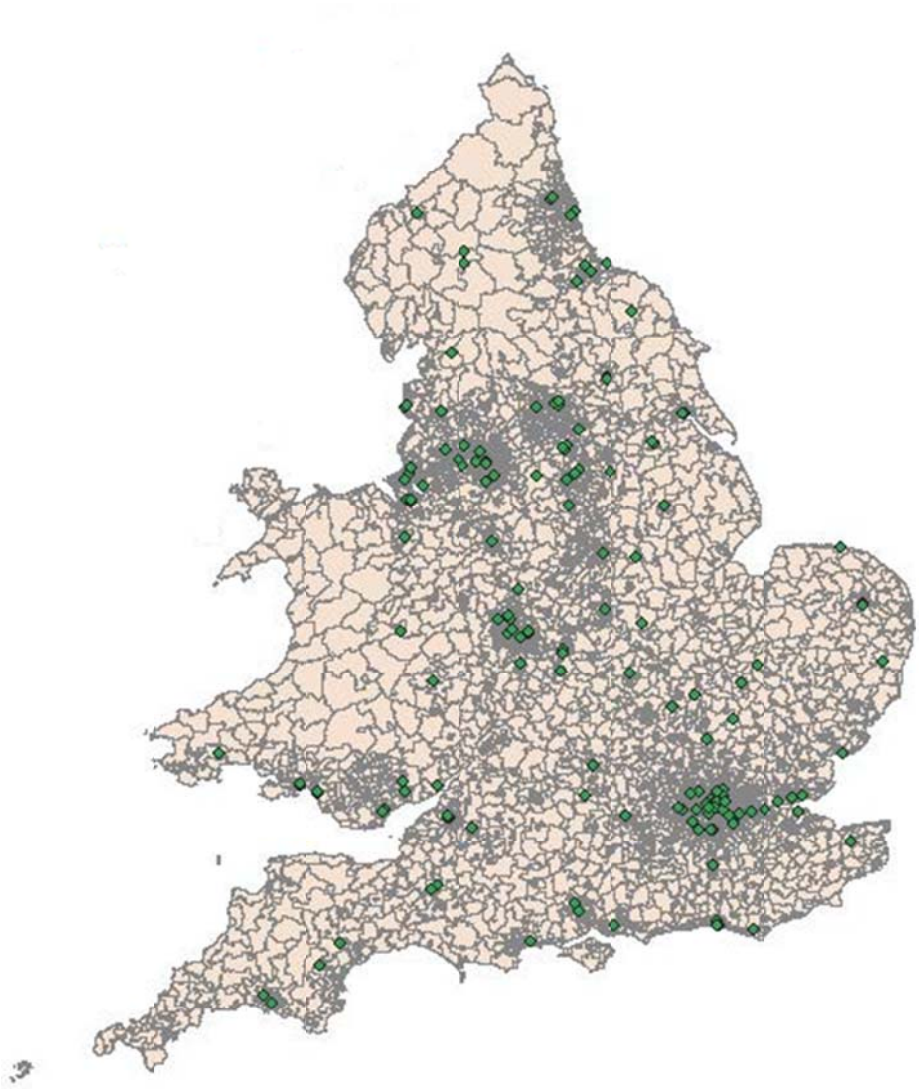

Figure 1. MSOAs and air quality monitors in England.

Figure 2. Trends in Pollution Over Time

Table 1. Pollution Summary Statistics

OVERALL SUMMARY	Mean	Std. Dev	Between MSOA std. dev.	Within MSOA std. dev.
Monthly Average CO (mg/m3)	0.71	0.33	0.20	0.26
Monthly Average PM10 (µg/m3)	25.6	5.08	3.19	3.95
Monthly Average O3 (µg/m3)	52.6	15.7	4.39	15.1

SEASONAL VARIABILITY	January-March	April - June	July - September	October – December
Mean Average CO (mg/m3)	0.81	0.55	0.57	0.92
Mean Average PM10 (µg/m3)	27.1	25.2	25.3	25.4
Mean Average O3 (µg/m3)	47.3	69.1	58.0	36.6

REGIONAL VARIABILITY	[A]	[B]	[C]	[D]	[E]	[F]	[G]	[H]	[J]	[K]
Mean CO (mg/m3)	0.54	0.54	0.46	0.64	0.61	0.54	0.78	0.96	0.77	0.83
Mean PM10 (µg/m3)	23.7	24.4	22.4	25.5	23.3	22.3	27.1	29.3	25.7	25.2
Mean O3 (µg/m3)	63.5	50.7	59.7	52.7	52.0	57.0	53.5	49.5	55.3	57.5

NOTES: All summarized data originally observed at the MSOA by month level. Regions are defined following U.K. Standard Government Office Regions Conventions for 1996-1998: A: North East; B: North West; C: Merseyside: D: Yorkshire & Humber; E: East Midlands: F: West Midlands; G: Eastern: H: London; J: South East

Table 2. Full Summary Statistics

	Sample w/ Covariates (329,082 children)		Full Sample (682,305 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	0.72	0.34
Monthly Average PM10 (µg/m3)	25.6	5.1	25.7	5.1
Monthly Average O3 (µg/m3)	52.6	15.7	52.5	15.7
Monthly Mean Temp. (°C)	10.98	4.85	10.88	4.82
Monthly Max Temp. (°C)	19.18	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
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	5.77	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. Respiratory Health Summary Statistics by Child Age

Child Age (in years)	Mean of treatment in month t	Std Dev. Of treatment in month t
2	1.26	35.5
3	0.99	31.4
4	0.85	29.1
5	0.65	25.3
6	0.50	22.4

NOTES: Outcome variable is an indicator for a respiratory treatment by child *i* in month *t,* expressed in #/1000.

TABLE 4. 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)	0.166*	0.136*					0.179*	0.124*
	(0.042)	(0.043)					(0.045)	(0.046)
Log (mean CO 1-12 months ago)		0.482*						0.527*
		(0.105)						(0.108)
Log (mean PM this month)			0.095	0.087			0.075	0.106
			(0.059)	(0.059)			(0.063)	(0.062)
Log (mean PM 1-12 months ago)				-0.319				-0.413
				(0.190)				(0.211)
Log (mean O3 this month)					0.209*	0.210*	0.283*	0.292*
					(0.064)	(0.064)	(0.065)	(0.066)
Log (mean O3 1-12 months ago)						-0.087		0.147
						(0.182)		(0.208)
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	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920

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

TABLE 5. 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)	0.188*	0.247*					0.194*	0.246*
	(0.048)	(0.049)					(0.053)	(0.054)
Log (mean CO 1-12 months ago)		1.790*						1.727*
		(0.254)						(0.247)
Log (mean PM this month)			0.120	0.141			0.067	0.102
			(0.060)	(0.062)			(0.066)	(0.067)
Log (mean PM 1-12 months ago)				0.397				0.328
				(0.222)				(0.298)
Log (mean O3 this month)					0.129	0.127	0.206*	0.160
					(0.065)	(0.065)	(0.067)	(0.068)
Log (mean O3 1-12 months ago)						-0.362		-0.394
						(0.221)		(0.303)
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
MSOA -by- YEAR Fixed Effects	YES	YES	YES	YES	YES	YES	YES	YES
Observations	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920

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

TABLE 6. 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)	0.172*	0.161*					0.185*	0.178*
	(0.045)	(0.045)					(0.048)	(0.049)
Log (mean CO 1-12 months ago)		0.699*						0.671*
		(0.137)						(0.136)
Log (mean PM this month)			0.091	0.091			0.054	0.067
			(0.059)	(0.060)			(0.063)	(0.064)
Log (mean PM 1-12 months ago)				0.013				0.031
				(0.203)				(0.238)
Log (mean O3 this month)					0.163*	0.163*	0.233*	0.226*
					(0.064)	(0.065)	(0.065)	(0.066)
Log (mean O3 1-12 months ago)						-0.385		-0.313
						(0.197)		(0.237)
Individual Controls	YES	YES	YES	YES	YES	YES	YES	YES
Region-specific Time Trends	YES	YES	YES	YES	YES	YES	YES	YES
Weather Variables	YES	YES	YES	YES	YES	YES	YES	YES
Year Dummies	YES	YES	YES	YES	YES	YES	YES	YES
Month of Year Dummies	YES	YES	YES	YES	YES	YES	YES	YES
MSOA -by- AGE Fixed Effects	YES	YES	YES	YES	YES	YES	YES	YES
Observations	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920

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

TABLE 7. 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)	0.110*	0.082*	0.166*	0.205*	0.174*	0.163*
	(0.029)	(0.029)	(0.033)	(0.033)	(0.030)	(0.031)
Log (mean CO 1-12 months ago)		0.255*		1.260*		0.577*
		(0.069)		(0.162)		(0.083)
Log (mean PM this month)	0.025	0.043	-0.001	0.025	-0.042	-0.031
	(0.039)	(0.040)	(0.042)	(0.043)	(0.040)	(0.040)
Log (mean PM 1-12 months ago)		-0.114		0.370		0.072
		(0.125)		(0.198)		(0.147)
Log (mean O3 this month)	0.156*	0.156*	0.108	0.063	0.089	0.076
	(0.045)	(0.045)	(0.045)	(0.047)	(0.044)	(0.045)
Log (mean O3 1-12 months ago)		0.094		-0.555		-0.548*
		(0.137)		(0.222)		(0.169)
Piecewise Linear Spline in Age	YES	YES	YES	YES	NO	NO
Weather Variables	YES	YES	YES	YES	YES	YES
Month of Year Dummies	YES	YES	YES	YES	YES	YES
Year Dummies	YES	YES	NO	NO	YES	YES
Region-specific Time Trends	YES	YES	NO	NO	YES	YES
Observations	40,938,300	40,938,300	40,938,300	40,938,300	40,938,300	40,938,300

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

TABLE 8. 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)	-0.031	-0.042	-0.009
	(0.025)	(0.028)	(0.026)
Log (mean CO 1-12 months ago)	0.080	0.248	0.125
	(0.058)	(0.136)	(0.071)
Log (mean PM this month)	-0.025	0.001	-0.026
	(0.035)	(0.037)	(0.035)
Log (mean PM 1-12 months ago)	0.129	0.257	0.212
	(0.099)	(0.161)	(0.128)
Log (mean O3 this month)	-0.055	-0.066	-0.066
	(0.036)	(0.038)	(0.036)
Log (mean O3 1-12 months ago)	-0.101	-0.281	-0.240
	(0.108)	(0.174)	(0.138)
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	19,744,920	19,744,920	19,744,920

Notes: * indicates statistical significance at the 1 percent level. Corresponding ICD-10 codes represent all blocks beginning with "S," including treatments for injuries to the head, neck, back, arm, leg, elbow, shoulder, wrist, ankle, knee, and hip.

TABLE 9. 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)	0.128*	0.095*	0.131*	0.112*	0.131*	0.124*
	(0.032)	(0.020)	(0.033)	(0.021)	(0.032)	(0.021)
Log (maximum PM this month)	-0.024	-0.006	-0.018	-0.001	-0.031	-0.024
	(0.035)	(0.023)	(0.036)	(0.023)	(0.036)	(0.023)
Log (maximum O3 this month)	0.104	-0.006	0.005	-0.080	0.018	-0.075
	(0.056)	(0.038)	(0.058)	(0.039)	(0.056)	(0.038)
Additional Individual Controls	NO	NO	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	19,744,920	40,938,300	19,744,920	40,938,300	19,744,920	40,938,300

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

TABLE 10. Effect of Pollution on Children's Respiratory Treatments: Disaggregated Diagnosis Codes

	J00-J06			J10-J18			J20-J22		
	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]
Log (CO this month)	0.083*	0.138*	0.115*	0.026	0.021	0.021	0.031	0.049*	0.038*
	(0.209)	(0.034)	(0.030)	(0.012)	(0.014)	(0.013)	(0.013)	(0.015)	(0.014)
Log (CO 1-12 months ago)	0.188*	0.514*	0.146	0.027	0.031	0.012	0.083*	0.263*	0.102*
	(0.070)	(0.141)	(0.080)	(0.030)	(0.066)	(0.036)	(0.027)	(0.066)	(0.035)
Log (PM this month)	0.122*	0.104	0.077	0.005	0.006	0.004	-0.010	-0.020	-0.014
	(0.039)	(0.043)	(0.040)	(0.017)	(0.018)	(0.017)	(0.017)	(0.018)	(0.017)
Log (PM 1-12 months ago)	0.020	0.450	0.249	0.012	-0.007	-0.033	0.017	-0.035	0.014
	(0.124)	(0.179)	(0.144)	(0.053)	(0.081)	(0.062)	(0.056)	(0.073)	(0.062)
Log (O3 this month)	0.195*	0.133*	0.139*	0.071*	0.076*	0.074*	0.045	0.036	0.049*
	(0.042)	(0.044)	(0.043)	(0.018)	(0.019)	(0.018)	(0.018)	(0.018)	(0.018)
Log (O3 1-12 months ago)	0.026	-0.324	-0.309	-0.095	-0.130	-0.067	-0.185*	-0.146	-0.172*
	(0.122)	(0.182)	(0.146)	(0.059)	(0.083)	(0.064)	(0.058)	(0.073)	(0.059)
Individual Controls	NO	YES	YES	NO	YES	YES	NO	YES	YES
Region-specific Time Trends	YES	NO	YES	YES	NO	YES	YES	NO	YES
Spline in Age	YES	YES	NO	YES	YES	NO	YES	YES	NO
Weather Variables	YES	YES	YES	YES	YES	YES	YES	YES	YES
Month of Year Dummies	YES	YES	YES	YES	YES	YES	YES	YES	YES
Year Dummies	YES	NO	YES	YES	NO	YES	YES	NO	YES
Observations	19744920	19744920	19744920	19744920	19744920	19744920	19744920	19744920	19744920

Notes: * indicates statistical significance at the 1 percent level. The relevant ICD-10 codes for these investigations are: J00-J06 for acute upper respiratory infections (including sinutis), J10-J18 for influenza and pneumonia, J20-J22 for other acute lower respiratory infections (including acute bronchitis and acute bronchiolitis), J30-J39 for other diseases of the upper respiratory tract (including rhinitis), and J40-J47 for chronic lower respiratory diseases (including asthma).

TABLE 10 (Continued). Effect of Pollution on Children's Respiratory Treatments: Disaggregated Diagnosis Codes

	J30-J39			J40-J47		
	[1]	[2]	[3]	[1]	[2]	[3]
Log (CO this month)	0.010	0.066*	0.020	-0.017	-0.017	-0.006
	(0.021)	(0.024)	(0.022)	(0.021)	(0.024)	(0.022)
Log (CO 1-12 months ago)	0.091	0.749*	0.269*	0.118	0.132	0.126
	(0.049)	(0.129)	(0.066)	(0.048)	(0.092)	(0.056)
Log (PM this month)	0.018	0.026	0.037	-0.024	-0.016	-0.033
	(0.030)	(0.031)	(0.030)	(0.031)	(0.031)	(0.030)
Log (PM 1-12 months ago)	-0.179	-0.093	-0.041	-0.242*	0.009	-0.120
	(0.091)	(0.155)	(0.115)	(0.090)	(0.128)	(0.107)
Log (O3 this month)	-0.010	-0.036	-0.019	-0.020	-0.051	-0.018
	(0.030)	(0.033)	(0.031)	(0.030)	(0.030)	(0.030)
Log (O3 1-12 months ago)	0.141	0.065	0.035	0.220	0.128	0.169
	(0.095)	(0.164)	(0.120)	(0.093)	(0.130)	(0.108)
Individual Controls	NO	YES	YES	NO	YES	YES
Region-specific Time Trends	YES	NO	YES	YES	NO	YES
Spline in Age	YES	YES	NO	YES	YES	NO
Weather Variables	YES	YES	YES	YES	YES	YES
Month of Year Dummies	YES	YES	YES	YES	YES	YES
Year Dummies	YES	NO	YES	YES	NO	YES
Observations	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920	19,744,920

Notes: * indicates statistical significance at the 1 percent level. The relevant ICD-10 codes for these investigations are: J00-J06 for acute upper respiratory infections (including sinutis), J10-J18 for influenza and pneumonia, J20-J22 for other acute lower respiratory infections (including acute bronchitis and acute bronchiolitis), J30-J39 for other diseases of the upper respiratory tract (including rhinitis), and J40-J47 for chronic lower respiratory diseases (including asthma).