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ABSTRACT

The Lifelong Costs of Urban Smogs*

Exposure to smoggy days is a common part of urban life, but can be avoided by vulnerable populations with municipal investment in warnings. This paper provides the first evidence on the long-term effects of early exposure to smog. Variation comes from exposure to the Great London Smog of 1952. Affected cohorts are tracked for up to sixty years using the Office of National Statistics Longitudinal Study. Exposure to the four day smog reduced the size of the surviving cohort by 2% and caused lasting damage to human capital accumulation, employment, hours of work, and propensity to develop cancer.

JEL Classification: Q53, I12, I18
Keywords: pollution, fetal origins, Great London Smog

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1 Introduction

Urban smogs occur whenever the normal dispersion of pollution stops, usually because of a change in the weather. In contrast to other environmental risks, urban smogs are brief occurrences that can be avoided with municipal investment in early warning systems. However, this investment is rare. The World Health Organisation has been able to document air quality for just eight percent of cities. Smogs are important because even short exposures to heightened levels of pollution are potentially dangerous, and the hypothesised effects on cancer and cardiovascular health could lie latent for decades. The focus of this study is an atmospheric temperature inversion that struck London in December of 1952, stopping the normal dispersion of pollution and resulting in an intense four-day smog of the kind experienced in low- and middle-income countries today. The Great London Smog is an ideal environment in which to gather evidence on the full, long-term effects of these events: exposure was clearly delineated both in space and time; pollution levels at the time were better documented than in many cities in the present day; reliable records on birth outcomes were available, making it possible to study short-term effects; and the timing of the event makes it possible to trace those exposed for just under sixty years.

As many hypothesised effects of fetal exposure to pollution can take decades to manifest, the ability to observe those affected for almost their entire working lives is particularly important. Those affected *in utero* or as infants are traced by their year and borough[1] of birth, and observed using the Office of National Statistics Longitudinal Study every decade from 1971, when those affected are around twenty, to 2011, when those affected are around sixty. Administrative data linked to the study makes it possible to observe effects on education, employment over the working life, absence from work due a permanent illness, cancer registrations, and mortality. This is the first study able to identify the effects of smog (or any pollution exposure) on these very-long-term outcomes, and the only study of the effects of fetal exposure on cancer registrations or mortality.

The study first investigated the effects of exposure on fetal loss using historical data from the Registrar General for England and Wales. Those *in utero* during the Smog would have been born in 1953. Results showed around 1280 fewer births in that year than would be expected, or a reduction of 2% in the size of the *in utero* cohort. Those observed later in life were 3% less likely to hold a degree-level qualification. There was no effect on unemployment, but there was a significant effect on hours worked in the early career, with those affected *in utero* working 8 fewer hours than unaffected cohorts, and those affected as infants working 4 fewer hours. There was no effect on cancer registration for those affected *in utero* but those affected as infants (and so exposed to the Smog directly) were 3% more likely to be diagnosed with cancer. Lastly, the treated

[1] Greater London is divided into 32 Boroughs, which average around 40 square kilometres in size, and had average populations of around 100,000 at the time of the smog. In the E.U. categorisation, one or two Boroughs form a NUTS3 region.

cohorts were both 2% less likely to die in sample - suggesting that those not surviving to be observed would have died before the age of sixty, in the absence of the Smog.

The evidence suggests that the benefits of avoiding exposure to smogs are considerably larger than current evidence suggests. People living in areas with high levels of ambient pollution experience smoggy days whenever winds slow, the weather is particularly cold, or a temperature inversion traps warm air at ground level. Given the costs to individuals and to the countries in which they live, it is striking that so few municipalities have invested in the infrastructure required to allow smog alerts. Pollution meters are cheap, avoidance is feasible, and the costs of exposure appear considerable.

2 Smogs and Long-Term Health

This section provides a background on pollution, how it measured, and how exposure to smoggy days might be expected to affect infant and fetal health.

2.1 Sources, measurement, exposure, and avoidance

The main source of urban pollution is the burning or combustion of fuel, producing primary pollutants like carbon monoxide, nitrogen and sulphur oxides, lead, and volatile organic compounds. Once in the atmosphere, pollutants can interact, generating secondary pollutants like ozone[2]. Common emitters produce a large number of pollutants simultaneously, and most legislators focus on a small number of pollutants as a proxy for general levels of pollution[3]. Particulate matter is the most commonly reported pollutant. The total amount of particulates suspended in the atmosphere (TSP) is measured in microgrammes per meter cubed ($\mu g/m^3$). Smaller particulates are particularly harmful because they can travel deep into the lungs, and gravimetric measures have emerged for fine particulate matter measuring less than 10 micrometers (PM_{10}) or less than 2.5 micrometers ($PM_{2.5}$). The black smoke (BS) method is a separate way of measuring particulate pollution used mainly in Europe. Here, particulates are measured by the reflectivity of a stain left by ambient air on white filter paper. Black smoke particulates are also measured in microgrammes per meter cubed. Measurements from the black smoke method are highly correlated with those from gravimetric measures, but due to the different techniques used, the conversion between black smoke and other particulate measures depends on the composition of the pollution.

Global exposures In the five years between 2008 and 2013, global pollution levels increased by 8%, with more than 80% of all urban residents exposed levels of pollution deemed unsafe by the World Health Organization. Exposures in low- and middle-income

[2]Information on major sources and health effects from the U.K. Department for Environment, Health, and Rural Affairs (DEFRA) website `www.uk-air.defra.gov.uk/`, and the U.S. Environmental Protection Agency website `www.epa.gov/airquality/urbanair/`

[3]In the U.S., for example, there is particular focus on the 'criteria' pollutants: carbon monoxide, sulphur dioxide, nitrogen oxide, ozone, lead, and particulate matter.

countries can be particularly extreme. Among the cities for which data is available, there were over a hundred and fifty cities with average annual levels over 100 $\mu g/m^3$ (or five times the WHO limit)[4]. Annual averages hide large fluctuations across time and space. Pollution exposure in a given day depends on wind-direction, temperature, and economic activity. The average annual level of pollution in Delhi is around $280\mu g/m^3$ for TSP, but between 2004 and 2010, there were fifty six occasions in which the Town Hall meter recorded levels of over $800\mu g/m^3$, and nine occasions when levels were over $1000\mu g/m^3$. Observations were recorded for only around one day in ten and the true figures are likely to be higher[5]. Meters in other residential areas of India have registered peaks of over 3000 $\mu g/m^3$[6]. Globally, the number of people exposed to extreme levels of atmospheric pollution is very large. Peshawar is home to over three million people and has annual average pollution levels almost twice those of Delhi.

Avoidance Actual exposure depends both on pollution in the air and measures taken to avoid it. Pollution reduces house prices (Chay & Greenstone 2005, Currie *et al* 2015, Sullivan 2016) and affects day-to-day behaviour (Janke 2014, Moretti & Neidell 2011)[7], showing that those with the resources to avoid pollution will do so. Smogs last at most a few days, and can be avoided by moving away, or by using masks to filter particulates, as is common in many Asian cities. The extent to which long-term levels of pollution can be avoided varies: sealing homes and offices against gaseous and fine particulate pollution can be difficult. Staying at home can be impractical or, in many cases, worse than the alternative. The daily average levels of PM_{10} caused by inefficient stoves ranges from $200\mu g/m^3$ to $5000\mu g/m^3$. The World Health Organization estimates that over 1.3 million people die prematurly each year as a result of indoor pollution caused by inefficient cooking apparatus[8].

2.2 How pollution affects health

A key source of damage from pollution arises from oxidative stress. Free radicals from pollutants attack cells at the molecular level, destroying proteins, lipids, and DNA. This process can kill or damage cells and is, in fact, an important part of how the immune system destroys pathogens. The main components of urban pollution either *are* free radicals (CO, NO_x) or produce them in the body (O_3, PM).

The body has both active and passive defences that respond to exposure. Some parts of the body - such as the lung lining fluid - naturally contain anti-oxidants that can protect critical systems from a degree of exposure by neutralising free radicals before they cause damage. In contrast, the effects of exposure can be magnified by the body's

[4]World Health Organization release based on data from the Global Urban Ambient Air Pollution Database. Available at `www.who.int/mediacentre/news/releases/2016/air-pollution-rising/en/`

[5]Data available from the Indian Central Pollution Control Board.

[6]Annual maximum exposures in residential areas, based on data on Indian cities between 1987 and 2007, collected by Greeenstone & Hanna (2014), and available on the AEA website.

[7]Almond & Mazumder 2013 review the literature on pollution avoidance behaviour.

[8]World Health Organization, available at *www.who.int/topics/airpollution/en*

immune response. Inflammatory cells flood affected and unaffected areas with free radicals which, in the absence of pathogens, attack native cells. This is one mechanism by which pollution exposure could result in damage to the heart, and other critical systems.

Health effects can emerge quickly. Ozone is very reactive, and exposure can result in adverse health effects in less than an hour, while effects from particulate matter have been recorded after less then four hours exposure (Rundel 2012). The second round of oxidative stress from the body's immune response can last from six to eighteen hours after exposure[9].

Kannan et al (2006) discuss the channels through which fetal exposure to pollution can affect health. One of the most well documented is the effect of polycyclic aromatic hydrocarbons (PAHs), which are found on particulate matter. PAHs are able to cross the placenta and form free radicals when metabolised, exposing the foetus to oxidative stress. Exposure in the first trimester, when the neural tube is formed, is expected to be most harmful. Exposure to PAHs has been linked to fetal mortality and – among those surviving to term – low birthweight, and smaller head circumference (Jayachandran, 2009).

2.3 Evidence on the short-run effects of exposure to pollution

A major concern in studying the effects of pollution on health is accounting for non-random assignment (Graff Zivin & Neidel 2013). The dominant approach in the economics literature is to study plausibly exogenous variation in pollution exposure from external shocks such as recessions or policy changes (Chay and Greenstone 2003), natural disasters (Jayachandran 2009), shocks to emissions (Pope 1986, Schlenker & Walker 2014) or external factors affecting location (Lleras-Muney 2010)[10].

Studying the effects of atmospheric pollution on fetal mortality presents the additional challenge that stillbirths are often under-reported and, should they occur early in the pregnancy, can even go unnoticed by the mother. Sanders and Stoecker (2011) take an indirect approach, exploiting the Trivers-Willard hypothesis that male foetuses are more vulnerable to external shocks than females. Using plausibly exogenous variation from the 1970 Clean Air Act and data from a 50% sample of U.S. birth certificates, the authors find that a one standard deviation drop in annual average particulates reduces the percentage of male live births by 3.1%. Jayachandran (2009) provides information on the effects of exposure to high levels of particulates. She overcomes the under-reporting of stillbirths by focusing instead on recorded births. Exploiting spatial and temporal variation from a wildfire that swept through Indonesia, the paper finds that exposure to a short, severe pollution shock led to 15,600 fewer births in subsequent months[11].

There is a growing literature on the effects of fetal exposure to pollution on health

[9] Unless otherwise stated, information on oxidative stress from Kelly (2003), who surveys the medical and epidemiology literatures on the health effects of pollution through the oxidative stress channel.

[10] This approach is also taken by some papers in the epidemiology literature - a notable example is Pope (1986), who studies the effect of the closure of a major emitter.

[11] Another paper taking this approach is Kwawand et al 2015, who study the effects of wildfires in California.

at birth. A fetal insult can have two effects on the relative health of a birth cohort. Exposure to pollution could lead to 'scarring' effects, whereby fetal exposure harms the health of each individual. Exposure could also lead to 'culling' effects, whereby the fetal insult affects the composition of the surviving cohort. When it is the strongest that survive a shock, these effects combine to make surviving cohorts appear healthier than they are[12]. Bozzoli, Deaton and Qintana-Domeque (2009) encounter this effect when studying the cross-country link between child mortality and adult height. The authors find the expected relationship among most countries, but find that child mortality is associated with an *increase* in the height of the surviving population in the poorest countries, where mortality was particularly high.

Nonetheless, there is strong evidence that fetal exposure to pollution affects infant health and mortality[13]. In utero exposure to pollution has been linked to prematurity, low birth weight and size (Liuchinger et al 2014, Yang & Chou 2016, Cho et al 2013). Currie and Walker (2011) study the effects of air pollution caused by traffic using variation from the introduction of the EZ-Pass scheme in New Jersey and Pennsylvania. This scheme allowed drivers to pass through toll gates without stopping, and resulted in a sharp reduction in carbon monoxide pollution in residential areas close to the tolls. The paper uses a differences-in-differences strategy, comparing the change in infant health of those *in utero* close to highway tolls to those *in utero* close to other parts of the highway system. Their results show that the introduction of the EZ-Pass scheme resulted in around an 11% reduction in prematurity, and a 12% reduction in birth weight - a common proxy of infant health.

Both *in utero* and neonatal exposure to pollution have been linked to increased infant mortality (Currie & Neidell 2005, Knittel et al 2011, Schlenker & Walker 2015, Clay et al 2016, Arceo et al 2016, Greenstone & Hanna 2014, Tanaka 2015, Khawand et al 2015) though it is often difficult to distinguish between the effects of in-utero and neonatal exposure, which tend to be highly correlated. Chay and Greenstone (2003) use variation in pollution caused by the 1981-82 U.S. recession to study the effects of particulate pollution on infant mortality. They find that a 1% decrease in pollution in a county results in a 0.35% reduction in the infant mortality rate. The strongest effects were found for infants less than one month old, suggesting that fetal exposure was an important factor. There is much less evidence from countries with high levels of ambient pollution, due mostly to the difficulty in obtaining information on health and pollution. Arceo-Gomez, Hanna & Oliva (2016) gather ten years of weekly data on health for forty eight municipalities in Mexico City, where data for pollution is also available. The authors adopt an IV strategy using temperature inversions - which prevent the dispersion of atmospheric pollution - as an instrument for exposure. The IV estimates show that a $1\mu g/m^3$ increase in particulates results in 0.24 infant deaths per 100,000

[12]See Almond and Currie (2011) for a fuller discussion of 'culling' in papers studying the effects of fetal shocks.

[13]See Zivin & Neidell (2013) for a survey of the health effects of pollution, including the effects of fetal exposure on infant mortality. Currie and Vogl (2013) provide an overview of the long-term effects of early shocks in developing countries, including those from atmospheric pollution.

births - a health effect similar to those found in the literature on the United States. Greenstone and Hanna (2014) construct a database of infant health and pollution levels in India in order to study the effectiveness of environmental regulations. The authors also test the effects of the most successful of the reforms, which promoted the use of catalytic converters, on infant mortality. Their results were suggestive of a decline in infant mortality, but were not statistically significant.

2.4 Evidence on the long-run effects of exposure to pollution

To the best of my knowledge, there is currently no evidence on the long-term effects of exposure to short urban smogs. There is also very little evidence on the longer-term effects of fetal exposure to long-term levels of pollution due to the difficulty in obtaining information on place of birth for individuals observed as adults[14]. I am aware of three studies of longer-term effects of exposure - though none track individuals for long enough to observe the more serious health effects (such as cardiovascular damage or cancer) that can result from pollution exposure.

The earliest, Sanders (2012) studies the effect of fetal exposure to pollution on performance in high school, using administrative data from Texas. Information on place of birth is not available, and *in utero* assignment of pollution is based on the county in which the high school is located. Exposure is calculated using county-level data on total suspended particulate matter, and is instrumented using county-level changes in relative manufacturing employment. The study finds that a standard deviation decrease in particulates is associated with a 2% increase in grades using OLS, and in a 6% increase using IV.

Isen, Rossin-Slater and Walker (2014) use linked administrative data from the U.S. census to investigate the effects of fetal exposure to particulates on income at the age of thirty. To identify the effect, the authors exploit a sharp drop in atmospheric pollution that followed the implementation of the 1970 Clean Air Act. Their results indicate that a 10 unit decrease in PM10 particulates resulted in a 1% decrease in earnings for individuals aged 29-30, mostly driven by a drop in labour force participation.

Lastly, Bharadwaj, Gibson, Graff Zivin, and Neilson (2016) study the effect of fetal exposure to pollution in Chile between 1990 and 2005. The levels of pollution in Chile dropped by more than fifty percent during the study period. The authors take a novel approach to the problem of geographic sorting, focusing on within-family fixed effects. They find that a one standard deviation increase in carbon monoxide exposure in the third trimester reduces language and mathematics scores by 0.042 and 0.038 standard deviations in the fourth grade.

[14]Evidence on other harmful exposures, such as from radioactive fallout or toxic waste, is discussed later in this section.

2.5 Evidence on other environmental and adverse life events

This paper is part of a larger literature on the effects of adverse environmental shocks. The focus of this paper is pollutants – such as the U.S. criteria pollutants – that are ordinarily present in the urban environment, but there is growing evidence that other kinds of man-made and natural environmental hazards can have a profound effect on health. Currie and Schmeider (2009), Agarwal et al (2010) and Persico et al (2016) study the effects of toxic releases. There is also growing evidence about exposure to water pollution (Gamber-Rabindran et al 2010), exposure to radiation (Almond et al 2009, Black et al 2013) and to dust clouds (Baek et al 2015, Arthi et al 2016).

This study is also related to a broader literature on the effects of *in utero* shocks. The earliest contribution in the economics literature is by Almond (2006), who studied the effect of fetal exposure to the the 1918 influenza epidemic. The later literature – see Almond & Currie (2011) for a review – has shown lasting effects from fetal exposure to poor health conditions (Bleakley 2007, Field et al 2009, Lin & Liu 2014, Barreca 2010, Kelly 2011) alcohol (Nilsson et al 2008), poor dietary conditions (Almond et al 2015, Chen & Zhou 2007), economic conditions (Wehby 2016, Burlando 2014), stress or bad news (Carlson 2014, Aizer et al 2015,), warfare (Lee 2014), or weather shocks (Groppo and Kraehnert 2015, Maccini & Yang 2009).

2.6 The Effects of Smog: Expectations

The focus of this paper is not the average annual level of pollution, but on smoggy days of the kind common when – due to a lack of wind, or other weather shock – the natural dispersion of pollution stops. This type of event can last from one to several days, leading to a doubling or tripling of ambient pollution levels. Medical and epidemiological evidence shows that only short exposures are required to produce damage to health and that, as exposure can lead to the death of cells, or damage to critical components, such as DNA, effects could persist long after exposure. Any single shock to pollution affects foetuses of between one and nine months of gestation. Consequently, the effects of a single day's exposure could be observed in birth outcomes for the nine subsequent months.

For short-term effects on fetal loss, the most relevant paper is by Jayachandran (2009), who studies a large increase in pollution caused by a wildfire that lasted from August to November. The air quality was very poor, with some areas experiencing 1000 $\mu g/m^3$ for PM_{10} for several days. The study found a reduction of 1.2% in the size of the affected cohort. To my knowledge, there is no evidence on the long-term effects of exposure to smoggy days. The evidence reviewed in the section above suggests that the effects of longer exposures over the entire pregnancy can persist at least until the thirties. This study follows those affected for sixty years, or almost to the end of their working life. The most serious effects of early exposure to pollution operate through damage to the cardiovascular system, and to cell components, which can increase the risk of cancerous growths. Both of these channels would tend to manifest towards the

age of fifty, and there is no evidence to date on outcomes late enough in life for these effects to manifest.

Fetal exposure to pollution can affect long-term outcomes in two ways. First, the average health of all members of the treated population would expected to be harmed. Second, exposures to shocks early in life can lead to miscarriage or infant mortality, meaning that the most vulnerable among those affected do not survive to be observed in later life. If, as in Jayachandran (2009), the latter effect is important, estimates will reflect an upward bias in health.

3 The Great London Smog of 1952

On the fifth of December of 1952, winds dropped and a high pressure weather system settled over London. Pollution from traffic and the burning of coal became trapped under an envelope of cold air, and a thick ground-level smog formed over the city[15]. Conditions persisted for four more days, during which time visibility dropped, and pollution levels increased threefold. London was accustomed to thick winter fogs and there was little panic. Reports at the time indicate that people stayed at home[16] or carried on with ordinary life. The Times of London reported that there were traffic problems as a result of reduced visibility. There were also reports about the cancellation of sporting fixtures, and an increase in housebreaking, but no reports relating to human health[17]. When official figures on deaths and hospitalisations arrived a week later, it became clear that there had been a dramatic effect on the population. A Ministry of Health (1954) report attributed four thousand premature deaths – mainly among the over sixty fives (see Figure 8) – to these five days of exposure[18]. Parliament responded with Clean Air Act of 1956, drafted with the goal of preventing any further smogs in London.

Historic pollution data for the UK goes back to around the mid-Forties, and it is possible to observe the Smog in context. Figure 3 shows black smoke particulates in London and urban areas of the UK from the period from 1950 to 1958. Annual averages are high in both series - at just over 200 $\mu g/m^3$ BS overall, and closer to 300 $\mu g/m^3$ BS during winter months[19]. In general, seasonal and annual dynamics are close. The

[15]Temperature inversions of the kind that affected London in 1952 are a common occurrence in many cities worldwide. Examples of cities in which inversions are particularly frequent are Los Angeles, Mexico City, Mumbai, Chengdu, Santiago, Sao Paolo, Tehran, and Milan.

[16]Houses in London were not (and are not) well sealed against the outside air.

[17]A report in the Times of December the 8th noted that some show cattle being transported to the Smithsfields show had encountered breathing difficulties.

[18]The four thousand excess deaths recorded in the next three months were initially attributed to influenza - but there was no evidence of influenza in the lungs of the diseased (Ministry of Health, 1954) and the Chief Medical Officer concluded that there was no major outbreak of influenza in 1952. Later studies have revised the number of excess deaths caused by the smog up to 12,000 Bell & Davis (2001)

[19]Conversion between Black Smoke particulates and TSP is imprecise and depends on local conditions. Bell and Davis (2001) also study the Great London Smog and convert Black Smoke particulates directly into TSP. The conversion rate during the fifties was likely close to 1:1 in winter months. The ratio changes in the summer, and TSP readings for annual averages are likely to be twenty percent higher than those for black smoke. Ball & Hume (1977) investigate traffic pollution in the 1970s in London using both Black Smoke and TSP meters, finding a winter conversion of around 1.14. However, the study is from twenty years later than the Smog, when black smoke stains were less pronounced. Commins & Waller (1966) also study particulates in London, using Black Smoke and TSP meters, for the period

London smog can be seen as a spike in December 1952, and a smaller London smog can also be seen in 1955. Variation in environmental conditions is not large in the UK compared to other countries, either over time or across space. Figure 3 shows minimum temperatures in London and for the rest of England. Minimum temperatures in the fifties were low, relative to long-run trends, but there was a not a lot of variation between years.

The Smog impact was well focused on London - figures 4, 5 and 6 show daily measurements taken during the first half of December for London, Great Britain, and for other large towns. Meters outside of London show a very small increase in pollution levels on the fourth and fifth of December, but nothing close to the scale of the London smog[20]. Exposure to pollution during the Smog cut across social lines. The most severely affected areas were both affluent (Kensington, Chelsea) and relatively deprived (South London). Figure 1 shows the effects of the smog by London borough[21]. The division of London into four levels of exposure is based on a report by The Ministry of Health (1954), which used information from 117 sulphur dioxide meters that were scattered around central and southern London. A Londoner in the five days of the smog experienced an average 'spike' in pollution exposure of over 1,100 $\mu g/m^3$ BS. This is a very severe pollution shock - closer in magnitude to serious pollution events in seen Mumbai and Beijing in recent years than to exposures seen in Europe or in the U.S[22].

The effect on mortality can be seen in the registrar data. Figure 7 shows the ratio of deaths in London to those in England and Wales. The impact of the smog is clearly visible, and there are no comparable incidents in the ten year period that the data covers. Deaths were concentrated among the over-forty fives. Figure 8 shows the total number of deaths recorded during the weeks following the smog, broken down by age.

4 Sources of Data

Once the risk of infant mortality has passed, the most serious effects of exposure to atmospheric pollution could take several decades to manifest. The main focus of this paper is to follow those exposed to the Great London Smog as infants or *in utero*, and gather evidence on their health and labour market outcomes over the next sixty

from 1955 to 1962, with results that imply a conversion factor of 1.25 for an annual average (and a lower number for readings taken during the winter.)

[20] A notable exception is Leeds (not included in figure 6, or in the main analysis), which experienced peaks in pollution exposure comparable to those in London.

[21] Greater London is divided into 32 Boroughs, which average around 40 square kilometres in size, and had average populations of around 100,000 at the time of the smog. In the E.U. categorisation, one or two Boroughs form a NUTS3 region.

[22] Kernel density plots based on readings from the Town Hall in Mumbai (from the Indian Central Control Board) and the U.S. Embassy in Beijing suggest that readings over 800 $\mu g/m^3$ are likely to occur almost every year in central Mumbai, and almost every other year in Beijing. Readings of over 1000 $\mu g/m^3$ are likely to occur almost every other year in Mumbai, and about one year in five in Beijing. Data on cities are rarely available at daily frequencies for low- and middle-income countries, and are particularly difficult to obtain for cities with high levels of pollution, where the incentives to collect and distribute data can be weak. Data collected by Greenstone & Hanna (2014) for India between 1987 and 2007 shows that residential areas of India have experienced daily peaks of over 3000$\mu g/m^3$ TSP - though pollution spikes of this size are rare.

years. This section outlines the data sources for long-term outcomes, key independent variables, and on birth outcomes used in the subsequent analysis.

4.1 Long-term labour market and health

Information on long-term outcomes comes from the Office of National Statistics Longitudinal Study. This is a study based on a 1% sample of the decennial census for England and Wales, with inclusion into the study determined by being born on one of four dates in a year. Participants are observed in the 1971, 1981, 1991, 2001, and 2011 censuses, and additional information on major events such as births, migrations, cancer registrations, and deaths have been linked by the Office of National Statistics. The full study holds information on just under a million individuals born in England and Wales. For the purpose of this paper, individuals affected by the smog *in utero* are identified by their year and borough[23] of birth. The full dataset used in this paper contains information from around 37,000 people born between 1950 and 1958, and observed in each round of the census. Around 6,600 of these people were born in London, with an average of 730 individuals born in any one year. Summary statistics can be seen in Table 1. As discussed in Bertrand *et al* (2004), performing the analysis with individual-level data could lead to inconsistent standard errors if there existed a random effect at the area-year level. Following the authors, the dataset is collapsed into area-birthyear cells, resulting in a balanced panel with fifty one geographic areas observed from 1950 to 1958.

4.2 Exposure to pollution and extreme weather

Both severe weather and pollution are expected to affect registered births in a given quarter. Data on weather conditions comes from the U.K. Meteorological Office, and includes monthly series for London and the rest of England and Wales for minimum temperatures, maximum temperatures, days of frost, and precipitation. A graph showing the temperature series for London and the UK can be seen in Figure 3. Seasonal changes tend to affect different parts of the U.K. symmetrically. Winter temperatures in London during 1952 were low relative to long-term trends, but almost identical to those in the five year period from 1951 to 1955. Data on black smoke particulates comes from the official publications of the Fuel Research Board[24]. There were around two hundred black smoke meters active in England and Wales during the period, though not all meters can be used to construct long series. Figure 3 shows a series for urban areas of England and Wales based on measurements from fifty eight meters, and a series for London, based on data from fifteen meters. In general, seasonal and annual dynamics are similar in different parts of England and Wales.

[23]Greater London is divided into 32 Boroughs, which average around 40 square kilometres in size, and had average populations of around 100,000 at the time of the smog. In the E.U. categorisation, one or two Boroughs form a NUTS3 region.

[24]Available in the UK National Archives.

4.3 Evidence on fetal loss

Before proceeding to the main analysis, the paper will look at the extent to which exposure to the Great London Smog affected fetal loss. Information on births outcomes is based on historic data from the Registrar General for Births, Deaths, and Marriages. This is the official record of births for the United Kingdom, and should contain the universe of registered births[25]. Two datasets have been constructed from the information available. The first is a quarterly time-series for births in London and the rest of England and Wales spanning from 1947 to 1964, which makes it possible to observe the Great London Smog in context. Figure 10 gives a long-view of births over this period, while Figure 11 shows the ratio of the two series. Overall, London and the rest of England and Wales share seasonal and broad long-term trends. There were two serious pollution events reported during the period. The first was a London smog in the winter of 1948, and the second was the Great London Smog in the winter of 1952. Both appear to register in the ratio series - with a dip in births coincident with when first trimester births exposed in 1948 would have been registered, and a long dip in 1953, highlighted in gray. (Those affected by the Smog *in utero* in December of 1952 would be born in 1953.) The second dataset is a quarterly panel broken down to the Borough[26] level. This data spans from the first quarter of 1947 till the fourth quarter of 1954. Summary statistics from this panel are presented in the following section.

5 The Effect of the Smog on Fetal Loss and Infant Mortality

Although the Smog lasted just a few days, the impact on fetal loss could be observed for up to nine months. This is because mothers were exposed at different stages in their pregnancies: fetal loss from exposure in the first trimester would be observed six to nine months later, while the effects of exposure in the third trimester would be observed soon after December. As discussed in earlier sections, early exposure to pollution is likely to be particularly serious, as this is when all critical systems are formed. A loss of the foetus at this stage would not be included in stillbirth records[27], and could potentially go unnoticed by the mother. Following from Jayachandran (2009), the approach of this section is to study the effect of the smog by observing 'missing' births in subsequent months. This measure is a good proxy for fetal loss, and has the advantage of being able to capture stillbirths, un-reported stillbirths, and fetal losses happening earlier in the pregnancy,

Data on births comes from the Registrar General for England and Wales, and takes

[25] Registration of births has been compulsory in the U.K. since the Births and Deaths Act of 1874.

[26] Greater London is divided into 32 Boroughs, which average around 40 square kilometres in size, and had average populations of around 100,000 at the time of the smog. In the E.U. categorisation, one or two Boroughs form a NUTS3 region.

[27] A stillbirth is defined as a fetal loss after 24 weeks of pregnancy.

the form of a quarterly panel broken down to the Borough[28] level. This data spans from the first quarter of 1947 till the fourth quarter of 1954. Summary statistics for the dependent variable and key controls are shown in Table 1[29]. The dependent variable is births per one thousand people B_{jgt} in borough j, and quarter t. It is modelled as a function of environmental exposures E_{jt}, a vector containing levels and squares of in-utero minimum temperatures, and average pollution exposure when in utero. Births show a clear seasonality, and a vector of dummies q_t is included in the model controls for quarter of birth. In order to control for unobserved nation-wide shocks, a differences-in-differences strategy is used. Below, D_j^L is a dummy for boroughs in London exposed the smog, and D_t^{53} is a dummy for the year 1953, when births affected by the December 1952 smog would be registered. Some specifications also include borough-level fixed effects λ_j, and year-level fixed effects μ_t.

$$B_{jt} = \alpha + \beta_1 D_j^L + \beta_2 D_t^{53} + \beta_3 (D_j^L D_t^{53}) + \beta_4 E_{jt} + q_t + \lambda_j + \mu_t + \epsilon_{jt} \tag{1}$$

The coefficient of interest is β_3, which can be interpreted as the change in births per one thousand people as a result of exposure to the Great London Smog. A key identifying assumption is that the effects of the Great London Smog did not extend out to the rest of England and Wales. Daily exposures to pollution during the Smog can be seen in Figures 5 and 6. Pollution levels in the rest of the UK and in other big towns do not show evidence of increased intensity during the period of the smog. When taking account of unobserved nation-level effects, a second key assumption is that London and the rest of England and Wales follow common trends in 1953. Trends relative to the UK were estimated using data from years unaffected by the Great London Smog. It was not possible to reject the null hypothesis that births-per-capita in London and the UK followed a common trend. The statistical model is estimated by ordinary least squares, with robust standard errors clustered at the borough-level. Results for the key parameters can be seen in Table 4. The estimated coefficient for β_3 was -0.09 for all models. The population of the affected Boroughs was around 4,750,000 in the fourth quarter of 1952. The estimated reduction in births per thousand people implies that there were around 1280 fewer births in London in the three quarters following the Great London Smog. This represents around a 2% reduction in the size of the affected cohort.

There is a significant body of evidence linking early exposure to pollution with infant mortality. In order to investigate this possibility, the exercise was repeated with deaths among infants less than one year old per 1000 people as the dependent variable. Unlike the analysis on missing births, it is not possible to distinguish cleanly between the effects of *in utero* and infant exposure when looking at infant deaths with quarterly data. This is because a child born in a given quarter could potentially appear in the infant mortality

[28]Greater London is divided into 32 Boroughs, which average around 40 square kilometres in size, and had average populations of around 100,000 at the time of the smog. In the E.U. categorisation, one or two Boroughs form a NUTS3 region.

[29]Figures for the control variables vary from the summary statistics presented in the data section because the number of years covered is smaller.

statistics for four successive quarters[30]. Results can be seen in the Table 5. The variable 'infant deaths after the Smog' measures the increase in deaths among infants less than one year old, per 1000 people, in the year following the Smog. The variable 'Infant deaths in quarter of the Smog' shows the number of deaths among infants born in the final quarter of 1952. This group is treated separately because only around a fifth would have been in-utero during the smog. Results for both variables show an increase of 0.01 - this is equivalent to an increase of fifty infant deaths per quarter, or a total of around 250. The number of infant deaths per 1000 people was around 0.135 per quarter in previous years, and so the percentage change in infant deaths is around 7%. Although a large percentage increase in mortality, the effect on the size of the surviving cohort is small, at around 0.2%.

6 Long-term effects for survivors

The most serious effects of pollution exposure can take decades to manifest. While the effects of education and employment can be observed after twenty or thirty years, an increased chance of developing cancer or cardiovascular problems would usually be observed from the late-forties onwards. The goal of this section is to follow those affected by the Great London Smog, and observe long-term effects on educational outcomes, employment through the life-cycle, and vulnerability to serious risks among the surviving population.

Data on long-term outcomes comes from a balanced area-birthyear panel of labour market and health outcomes, which covers cohorts born in 1950 through to 1958, and is based on the Office of National Statistics Longitudinal Study[31]. The outcome variable Y_{jt} is derived from information on individual labour market and health outcomes collapsed to the area j and birth-year t level. All specifications contain the variable $SMOG_{jt}$, a dummy that takes the value of one if the cohort jt was affected by the Great London Smog; $\mathbf{E_{jt}}$, which contains levels and squares of in-utero minimum temperature and pollution exposure, and area and year fixed effects. Some specifications also contain $\mathbf{X_{jt}}$, containing collapsed individual information on gender and ethnicity; and treatment-level time trends.

$$Y_{jt} = \alpha + \beta SMOG_{jt} + \mathbf{E_{jt}}\boldsymbol{\gamma} + \mathbf{X_{jt}}\boldsymbol{\delta} + \zeta_j + \eta_t + \tau_j t + \epsilon_{jt} \tag{2}$$

The coefficient of interest β can be interpreted as a differences-in-differences estimate, measuring the change in the outcome Y_{jt} caused by exposure to the Great London Smog. The estimated effect is on the cohort, and incorporates both direct effects on the health of children and, potentially, later parental responses to poor health.

Estimation is by ordinary least squares, with robust standard errors clustered at the area-level. The unaffected areas in the differences-in-differences design are other

[30] Thus, 1952q4 statistics contain information from only those affected in utero, 1953q4 contains information from those only affected as infants, and quarters in-between contain a mix of the two.

[31] For more detail on data used and summary statistics, see Section 4.

urban areas of England and Wales[32]. A key condition for the identification of β_2 is that outcomes in affected areas of London and those in unaffected urban areas of England and Wales follow parallel trends. This assumption will be tested for each of the outcomes presented using information from years unaffected by the smog. A second condition is that the effects of the Great London Smog did not extend out to the rest of England and Wales. Daily exposures to pollution during the Smog can be seen in figures 5 and 6. Pollution levels in the rest of the UK and in other big towns do not show evidence of increased intensity during the period of the smog.

Fetal exposure to pollution can affect long-term outcomes in two ways. First, the average health of all members of the treated population would expected to be harmed, shifting the whole health distribution of those observed to the left. Second, exposures to shocks early in life can lead to fetal loss or infant mortality, meaning that the most vulnerable among those affected do not survive to be observed in later life. This would remove the 'left tail' of the health distribution, so that people who would have encountered serious health issues later in life do not survive to be observed. The total effect would be of a treated population that is less healthy on average, but in which very sick people are no longer observed. Evidence on fetal loss from the previous section suggests that around 2% of the treated population did not survive till birth, which would be expected to create a shift towards healthier outcomes in β_2 for the affected cohort. As in all studies of long-term outcomes from early shocks, estimates must therefore be treated as causal effects for the cohort, but lower bounds for individuals. It will be possible to provide evidence on the two mechanisms described later in the analysis. Although most outcomes, such as education and employment, are based on averages in the population, some measures capture changes in the lower tails of health. In particular, it will be possible to observe whether people die young, and it will be possible to observe if people are permanently out of work due to a limiting illness - another measure of serious ill health.

6.1 The Surviving Population

There is evidence from historic registrar data that exposure to the Great London Smog had a significant effect on the size of the surviving population. A natural first step is to gather evidence on the surviving cohort. There are two outcomes in the data, gender and ethnicity, that would be fixed at conception, making it possible to gather evidence on the effect of the Smog on the composition of the surviving population.

Gender

As discussed in the literature review, the hypothesis that females are more likely to survive shocks has found empirical support in both the epidemiological and economics literatures. Sanders and Stoecker (2011), for example, studied the effects of pollution ex-

[32]Rural areas were defined as those with population density less than 400 people per square kilometer, and removed from the unaffected group.

posure on the gender of survivors, finding that a standard deviation increase in pollution exposure decreased the percentage of births that are male by 3.1%. Before proceeding to the main analysis, a test was run to check whether gender followed common trends in London and urban areas of England and Wales - it was not possible to reject the null of common trends. Results for the differences-in-differences estimate for β_2 are presented in Table 6. A graph of gender in London and urban areas of England and Wales can be seen in Figure 12. The cohort affected by the smog *in utero* are 3% less likely to be male, but the difference is not statistically significant. The cohort affected as infants are 6% less likely to be male.

Ethnicity

The second characteristic fixed at conception is ethnicity. Before proceeding to the main analysis, trends in London and urban areas of England and Wales were tested. There was a difference in trends of 0.002 that was significantly different from zero. This can also be seen as a slow divergence between the series in Figure 13. Results can be seen in Table 7. The cohort affected by the smog *in utero* were 1% less likely to be white, but this result is not robust to allowing for differential trends. Those affected as infants are 3% less likely to be white - accounting for trends reduces this to 2%, but the difference remains significant.

6.2 Education

Educational attainment can capture the effects of weaker health problems that might not appear in hospital records and mortality statistics. The series for London and for urban areas of England and Wales can be seen in Figure 14. The series appear to follow each other closely, except for the cohorts affected by the smog. It was possible to reject the hypothesis of common trends, but the estimated coefficient was very close to zero. Results can be seen in Table 8. Those affected in utero are 3% less likely to hold a degree. The result is only significant (at the 5% level) in the two specifications that take trends into account. The estimate for infants also show a difference of 2%, but are not statistically significant.

6.3 Unemployment

Fetal exposure to pollution could affect employment directly through its effects on health, or indirectly through its effects on educational attainment. As with education, changes in employment can be a good measure of the kind of health effects that would not appear in statistics on hospitalisations or mortality. The studied cohorts entered the labour market under very similar conditions. Figure 9 shows that average levels of unemployment on entry were 2.8% while those for the 1953 cohort were 3.1%, and so any employment effects are unlikely to be driven by issues of timing. Individuals are first observed in 1971, when the treated population were aged twenty and may still be

at university. They are then observed at four points during the normal working life: in 1981 when thirty, in 1991 when forty, and in 2001 when fifty.

The unaffected years were tested for differential trends. It was possible to reject the null of common trends for all by the 2011 cohort, for which there was a difference of 0.002: this is significantly different from zero, but too small to affect the analysis. Results be seen in Table 9. Overall, there was no significant difference in unemployment for the *in utero* cohorts. The results for the infant cohort are similar, except for in 1991 (when the cohort were around forty years old.) In this year, the infant cohort appear 2% less likely to be unemployed.

6.4 Hours Worked

Hours worked were observed in 1971, 1991 and 2001, when the affected cohorts were around twenty, forty, and fifty years old. Graphs of the three series can be seen in Figures 15, 16, and 17. The two series follow each other closely in unaffected years. Working hours shown in the graph for 1971 census tail off for birth years later than 1956 as many are too young to work legally. (The working age in the U.K. is sixteen.) It was not possible to reject the null of common trends for all years. Results can be seen in Table 10. Those affected by the smog *in utero* work eight fewer hours than their peers in 1971, and between one and two fewer hours in 1991. There is no significant difference by 2001. Those affected by the smog as infants work four fewer hours in 1971. The estimate for 1991 is around negative one, but not significant, and there is no difference in hours by 2001.

6.5 Permanently out of the workforce

Fetal loss or infant mortality as a result of early exposure ot the Great London Smog would tend to be concentrated among those most likely to become unhealthy adults. In the data, this would be equivalent to truncating the left-hand tail of the health distribution. The employment data in the ONS Longitudinal Study includes a question that could shed light on whether there are fewer adults with very poor health in the sample. Respondents are asked to answer the following question: 'I am permanently out of employment for health reasons.' This question was asked in a consistent way over the respondents' life-cycle and, being a more concrete measure than self-reported health, avoids some conceptual issues with that measure[33]. It was not possible to reject the null hypothesis of parallel trends for all census years. Results can be seen in Table 11. The cohorts affected by the smog *in utero* were similar to unaffected cohorts in 1971, 1981, and 1991. In the first two decades, the estimate was negative and significant, but very

[33]Deaton (2008) discusses three key issues. The first is that people might not fully perceive the impacts of a health shock. Someone with small respiratory problems may not fully contemplate the career as a professional footballer they might have had in full health. The second is that people grow accustomed to their ailments, and no longer consider them to be day-to-day problems. The third is that there are cross-country differences in how this kind of question is answered due to both cultural differences, and differences in the average health of comparison groups.

small. They were 2% less likely to be permanently sick in 2001, and 1% less likely in 2011, though this latter result is not significantly different from zero. For those affected as infants, there are no significant differences in severe illness.

6.6 Cancer

Exposure to atmospheric pollution has been linked to cancer in numerous studies, but there has been no evidence to date on the effects of fetal exposures, because it can take many years for cancers to develop. A graph of cancer incidence in London and urban areas of England and Wales can be seen in Figure 18. The two lines follow each other closely. It was not possible to reject the null of common trends for cancer incidence. Results can be seen in Table 12. For the cohort affected when *in utero*, there was no effect on cancer registration. The cohort affected as infants were 3% more likely to develop a cancer, though this estimate was only statistically significant in the specifications incorporating trends.

6.7 Mortality

The final outcome observed is mortality, defined as dying before 2011, when the affected cohorts would be around sixty years old. A graph of mortality series for London and urban areas of England and Wales can be seen in Figure 19. It was not possible to reject the null of parallel trends for mortality. Results can be seen in Table 19. They show that cohorts affected *in utero* and those affected as infants are both 2% less likely to have died by 2011 than unaffected peers. The section on missing births showed around a 2% reduction in the *in utero* cohort due to fetal loss, and evidence on gender within the infant cohort also suggests an effect on the size of the surviving cohort. Taken together, it may be that those not surviving to be observed are less healthy than the average in the cohort, and would have been more likely to die before sixty.

7 Discussion

Smogs are a common phenomenon in urban environments. The goal of this study was to investigate the long-term effects on a cohort of early exposure to a severe smog, of the kind seen in low- and middle-income countries. This section will first summarise the results above, providing some interpretation, and then discuss implications for policy.

7.1 Early Exposure to Smog

The results showed that smoggy days, and the brief but intense increases in pollution exposure that accompany them, can have two serious effects on the affected *in utero* and infant cohorts: early loss among the most vulnerable, and lasting damage to the surviving population.

Early loss among the most vulnerable The first effect of the smog was an increase in fetal and infant mortality. Evidence from the Registrar General showed around 1280 fewer births in the year following the Smog than would be expected, or a reduction of 2% in the size of the *in utero* cohort. Infant deaths were also 7% higher following the smog, though it was not possible to determine if this increase was among those affected *in utero* or as infants. There is evidence in the epidemiological and economic literatures suggesting that males are more vulnerable to *in utero* health shocks[34]. The surviving population were first observed in 1971 at around twenty years old. For the cohort affected *in utero*, there was no significant evidence that fetal loss was concentrated among males. The cohort affected as infants were 5% less likely to be male, indicating a considerable difference in survival between male and female cohorts affected as infants.

Fetal or infant mortality would also be expected to be concentrated among the most vulnerable. Evidence from two outcomes, both associated with serious poor health, provided support for this hypothesis. Those exposed *in utero* were significantly less likely to be 'permanently out of work for reasons of poor health' than their peers, though the difference was small, peaking at 2% at around the age of fifty, and not always statistically significant. Stronger, was the evidence on dying before the age of around sixty. The cohorts affected *in utero* and as infants were both 2% less likely to have died young, suggesting that those not surviving fetal or infant exposure would have been more likely to die young, in the absence of the smog[35]. The observed effects were robust to controls for the proportion of men and ethnic minorities in the cohort, and environmental controls including levels and squares of temperature and black smoke particulate pollution.

Lasting damage to the surviving population Evidence on other outcomes indicates that exposure to the four day smog created lasting damage to health that affected labour market outcomes, human capital accumulation, and health. The strongest differences were in the twenties and thirties, the years in which labour market trajectories are largely determined. In 1971, those affected *in utero* worked eight hours less than unaffected cohorts, and those affected as infants worked four fewer hours. This effect faded slowly, either as the affected cohorts adjusted, or the unaffected cohorts 'caught up' in terms of poor health. By 1991, when the affected cohorts were around forty, those affected *in utero* worked between one and two hours less, and there was no significant difference for those affected as infants. The evidence was not consistent with a difference in unemployment, but the two richest specifications showed a significant reduction in human capital accumulation, with those affected by the smog *in utero* three percent less likely to hold a degree-level qualification. Lastly, estimates from the two richest specifications showed those exposed to the smog directly as infants were 3% more likely to develop a cancer, implying that some of the damage from the smog lay latent for several decades.

[34]See, for example, Sanders and Stoecker (2011), and the papers cited within,

[35]The alternative explantation, that pollution has beneficial effects on those affected ('what doesn't kill you make you stronger'), is not consistent with evidence from the wider medical and epidemiological literature. Pollution always has a deleterious effect on health.

The observed effects were, again, robust to controls for the proportion of men and ethnic minorities in the cohort, and environmental controls including levels and squares of temperature and black smoke particulate pollution.

7.2 Implications for policy

Smogs occur whenever the normal dispersion stops or slows down, such as when winds stop, the weather is particularly cold, or a temperature inversion traps warm air at ground level. The key difference between smogs and general levels of pollution is that the former are mercifully brief, usually lasting just a few days. With proper investment in education and municipal smog warnings, reducing the harm to vulnerable populations is a feasible and affordable possibility. The effects of shocks to air quality on the most vulnerable among the adult population is well documented. To my knowledge, this study is the first to document the lifelong costs to those affected either *in utero* or as young infants. The returns to investment in pollution meters and other infrastructure that could be used to warn vulnerable populations are surely high, but these investments are rare. The World Health Organisation was able to collect pollution data from only around 8% of cities - with missing values most common in areas expected to have high levels of atmospheric pollution.

Data on cities are rarely available at daily frequencies for low- and middle-income countries, and are particularly difficult to obtain for cities with high levels of pollution, where the incentives to collect and distribute data can be weak. Evidence from cities that *have* invested in monitoring networks suggests that severe smogs are not rare events. Kernel density plots based on readings from the Town Hall in Mumbai (from the Indian Central Control Board) and the U.S. Embassy in Beijing suggest readings over 800 $\mu g/m^3$ TSP occur almost every year in central Mumbai, and almost every other year in Beijing. Readings of over 1000 $\mu g/m^3$ TSP are likely to occur almost every other year in Mumbai, and about one year in five in Beijing. Data collected by Greenstone & Hanna (2014) for India between 1987 and 2007 shows that some residential areas of India have experienced daily peaks of over $3000\mu g/m^3$ TSP - though smogs of this severity are very uncommon.

As gestation is around nine months, once yearly shocks would affect three quarters of the babies born in the subsequent year. The severity of smogs is linked to average levels of pollution, and the number of people exposed to high average levels of pollution is very large. In 2012, over a hundred of the cities taking measurements had average levels of over $100\mu g/m^3$. Average levels in Delhi were $286\mu g/m^3$, while those in Peshwar, home to over three million people, were over $500\mu g/m^3$.

The policy implications of this study are direct - smogs do not last long, and their harmful effects can be avoided (relatively) easily by vulnerable populations when proper warnings are available. Evidence of lasting effects on labour market outcomes, human capital accumulation, and health suggest the returns to cheap monitoring networks are very high.

8 Conclusion

Smogs are a common occurrence in many cities around the world, but can be avoided by the most vulnerable with proper investment in municipal warning systems. The goal of this paper was to study the effects of the kind of severe smogs common in low- and middle-income countries by following up on the cohorts affected as infants and *in utero* by the Great London Smog of 1952. This smog was an ideal environment in which to gather evidence about these events: the pollution shock was clearly delineated both in space and time, making it possible to cleanly separate *in utero* from neonatal exposures; the government at the time kept good records of health outcomes, and exposures to pollution; and the timing of the event makes it possible to study hypothesised effects on long-run health that would be otherwise impossible.

The study first investigated the effects of exposure on fetal loss using historical data from the Registrar General for England and Wales. Those *in utero* during the Smog would have been born in 1953. Results showed around 1280 fewer births in that year than would be expected, or a reduction of 2% in the size of the *in utero* cohort. Those observed later in life were 3% less likely to hold a degree-level qualification. There was no effect on unemployment, but there was a significant effect on hours worked in the early career, with those affected *in utero* working 8 fewer hours than unaffected cohorts, and those affected as infants working 4 fewer hours. There was no effect on cancer registration for those affected *in utero* but those affected as infants (and so exposed to the Smog directly) were 3% more likely to be diagnosed with cancer. Lastly, the treated cohorts were both 2% less likely to die in sample - suggesting that those not surviving to be observed would have died before the age of sixty, in the absence of the Smog.

The results of this paper imply that the costs of short smogs can last a lifetime for those affected young. As shocks such as these happen with some frequency in low- and middle-income countries, the short and long-term benefits of investing in the protection of those *in utero* from exposure to airborne pollution are likely to be particularly high.

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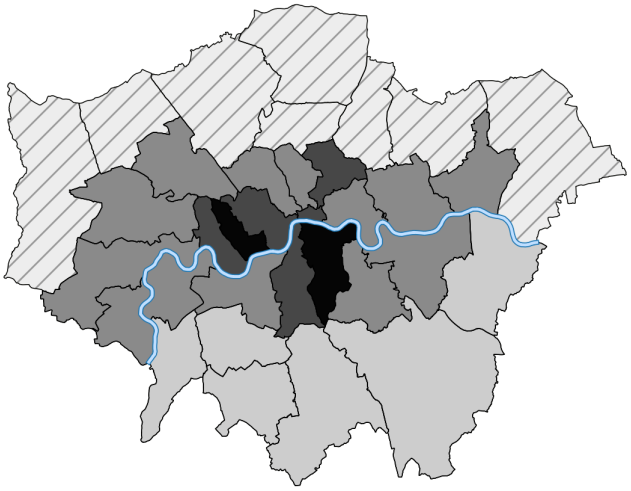
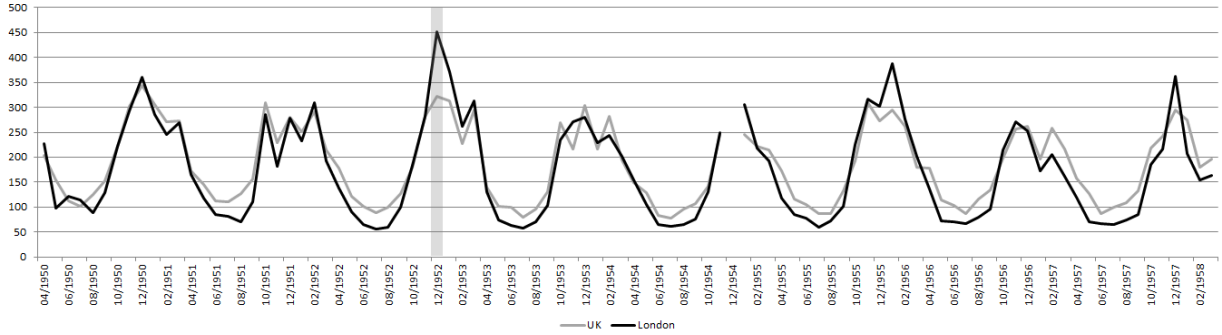
Figure 1: Impact of the Smog in December 1952



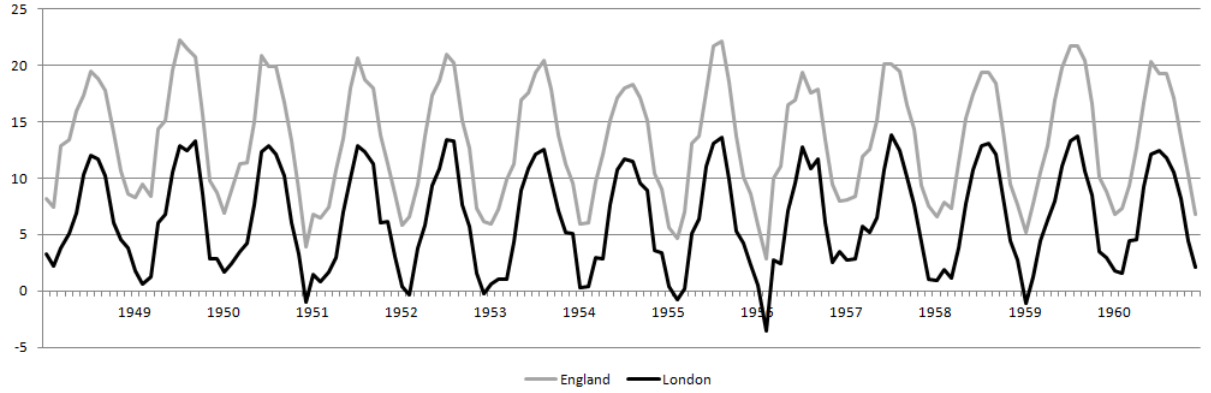
Notes: Figure shows Greater London divided into Boroughs, with the Thames running from west to east, and more affected regions of London shaded in darker gray. For reference, London Boroughs average around 40 square kilometres in size, and had average populations of around 100,000 at the time of the smog. The two darkest regions include the London Boroughs of Kensington and Chelsea, Southwark, Lambeth, Westminster, Hackney, Hammersmith and Fullham, and the City of London. The two lighter regions include Newham, Wansworth, Barking and Dagenham, Brent, Camden, Ealing, Greenwich, Hounslow, Islington, Lewisham, Richmond, Tower Hamlets, Bexley, Bromley, Croydon, Kingston, Merton, and Sutton. There is no information on exposure in the striped area to the north. (Though the area was affected by the Smog.) Pollution data from the Fuel Research Board and Wilkins (1954), mapping data from the Ordinance Survey.

Figure 2: Levels of Particulate Pollution (Black Smoke) in London and the UK



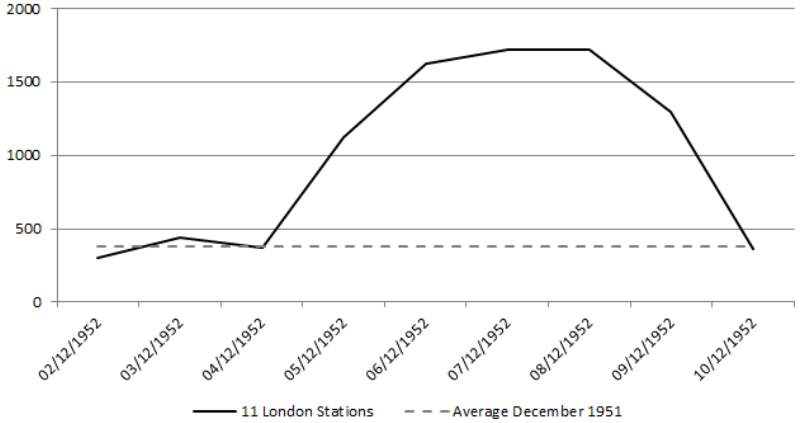
Notes: Particulate pollution measured in microgrammes per meter cubed, black smoke. London series based on fifteen meters in the Greater London area. UK series based on fifty eight urban meters, with all regions represented. Data from the Fuel Research Board.

Figure 3: Daily minimum temperatures in London and England, degrees Celsius



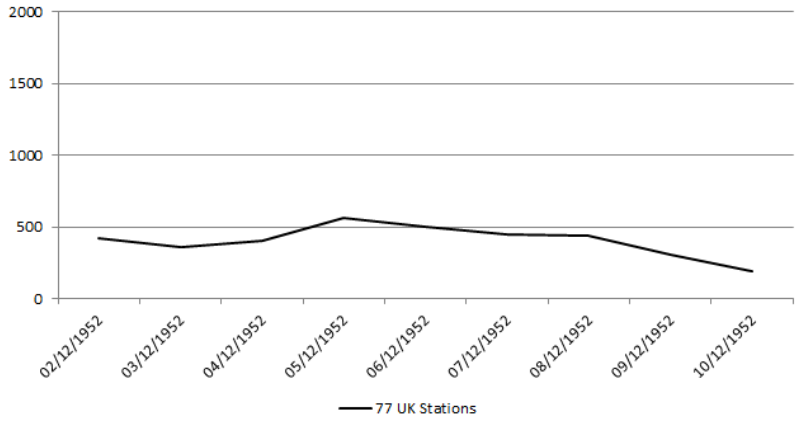
Notes: Temperatures measured in degrees Celsius. Data from the Met Office.

Figure 4: Pollution in December 1952 - London



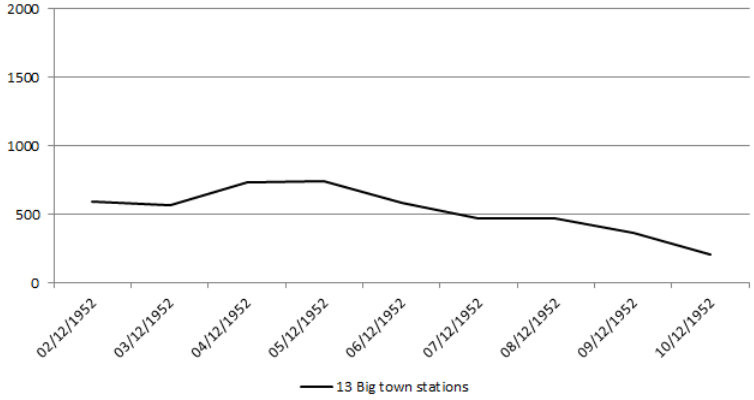
Notes: Pollution data is for black smoke particulates and is measured in micro-grammes per cubic metre. Solid black line based on an average from 11 monitoring stations in London. Data from the Fuel Research Board.

Figure 5: Pollution in December 1952 - Great Britain (Excluding London)



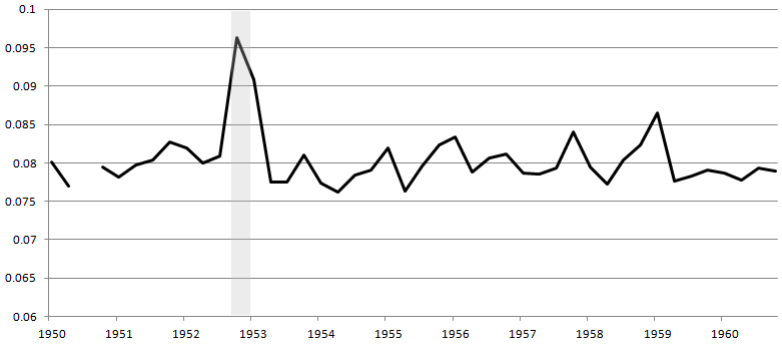
Notes: Pollution data is for black smoke particulates and is measured in micro-grammes per cubic metre. Solid black line based on an average from 77 monitoring stations in Great Britain. Data from the Fuel Research Board.

Figure 6: Pollution in December 1952 - Other Big Towns (Excluding London)



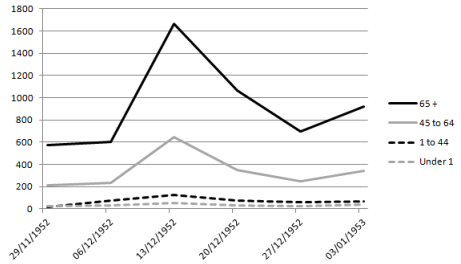
Notes: Pollution data is for black smoke particulates and is measured in micro-grammes per cubic metre. Solid black line based on an average from 16 monitoring stations other large towns in the UK. These are: Bradford, Bristol, Cardiff, Glasgow, Leicester, Liverpool, Manchester, Newcastle, Nottingham, and Sheffield. Data from the Fuel Research Board.

Figure 7: Ratio of Deaths in London to Deaths in England and Wales



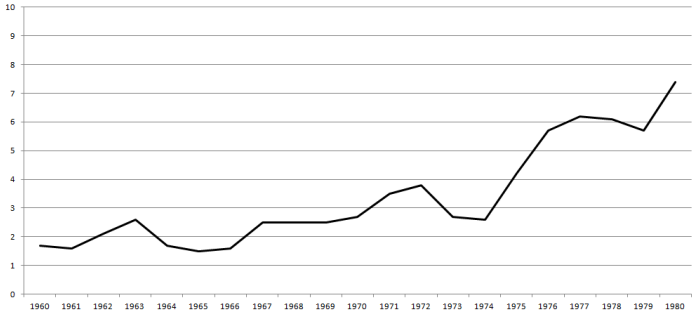
Notes: Ratio of quarterly deaths in London to quarterly deaths in England and Wales. Data from the Registrar General for England and Wales

Figure 8: Deaths in London during the Smog of 1952, by Age



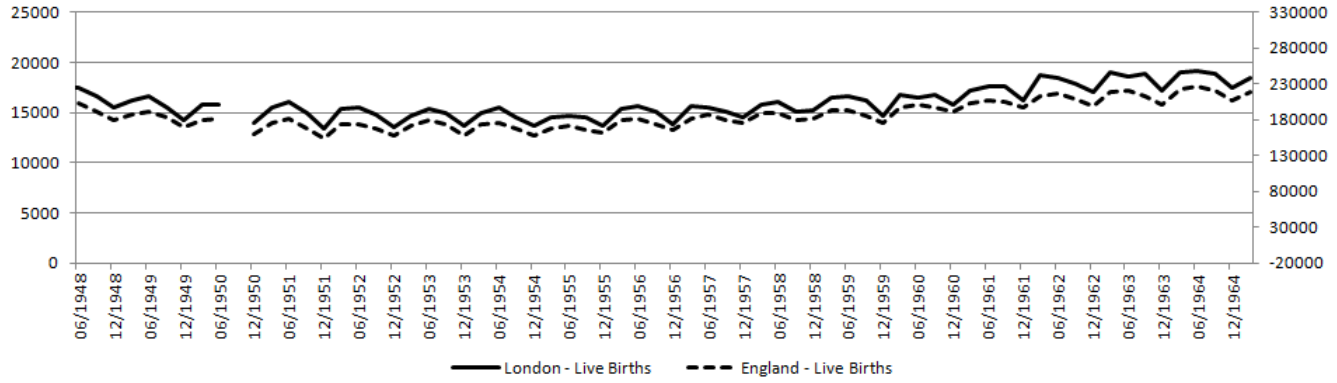
Notes: Deaths reported at the end of each week to the London administrative county, recorded in Ministry of Health (1954)

Figure 9: Labour Market Conditions (Unemployment) in the UK



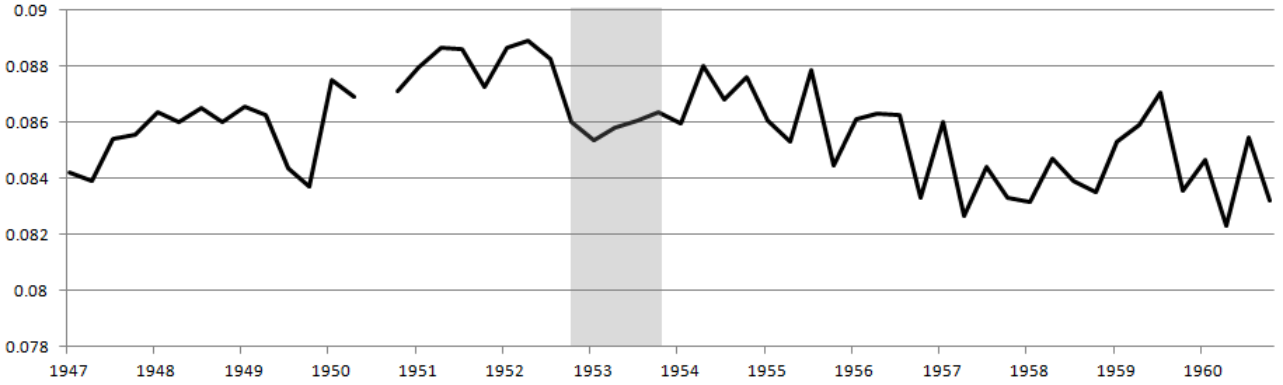
Notes: The (affected) 1952 cohort would enter the labour market between 1969 and 1971 when average levels of unemployment were 2.8% on average. The (unaffected) 1953 cohort would enter the labour market between 1970 and 1972 when average levels of unemployment were 3.1% on average. Source: Office of National Statistics

Figure 10: Births in London (left axis), England & Wales (right axis)



Notes: Data from the Registrar General

Figure 11: Ratio of Births in London, and England & Wales



Notes: Data from the Registrar General

Table 1: Summary Statistics for Long-Term Outcomes

	All Cohorts		**1953 Cohort**	
	London	England & Wales	London	England & Wales
Fixed at conception				
Percentage male birth	0.49	0.50	0.50	0.50
Percentage non-white	0.04	0.01	0.03	0.01
Potential outcomes from exposure				
Holds degree in 1981	0.11	0.09	0.10	0.09
Unemployed in 1971	0.02	0.03	0.04	0.04
Unemployed in 1981	0.05	0.07	0.05	0.06
Unemployed in 1991	0.04	0.04	0.01	0.02
Unemployed in 2001	0.02	0.03	0.03	0.02
Unemployed in 2011	0.04	0.03	0.03	0.04
Hours worked in 1971	40.5	41.9	39.7	42.1
Hours worked in 1991	35.9	35.5	35.5	35.5
Hours worked in 2001	38.1	38.3	38.1	38.6
Permanently Sick in 1971	0.00	0.00	0.00	0.00
Permanently Sick in 1981	0.00	0.00	0.00	0.00
Permanently Sick in 1991	0.02	0.02	0.01	0.02
Permanently Sick in 2001	0.04	0.06	0.03	0.06
Permanently Sick in 2011	0.06	0.08	0.09	0.10
Died before 2011	0.03	0.03	0.02	0.04
Cancer registration before 2011	0.09	0.10	0.09	0.10

Notes : Data from the Office of National Statistics Longitudinal Study. Sample size for each census is around 45,200, of which around 6,000 were born in London. In 1953, sample sizes are approximately 4700, of which, around 660 were born in London. Post-exposure statistics based on 'all cohorts' would not be expected to correspond to those for the 1953 cohort, especially for early life events.

Table 2: Summary Statistics for Environmental and Pollution Data

	mean	std.dev	min	max
Quarterly minimum temperature	0.4	1.4	-3.6	4.3
Quarterly Maximum temperature	13.6	5.6	2.9	24.4
Quarterly Days of Frost	3.9	5.2	0.0	22.9
Quarterly Inches of Rain	54.5	29.0	2.3	145.0
Quarterly average black smoke	186.6	110.1	40.3	730.3

Notes: Temperatures measured in degrees Celsius. Weather data from the Met Office. Pollution data measured in microgrammes per meter cubed. Data from the Fuel Research Board.

Table 3: Summary Statistics for Short-Term Outcomes

	mean	std.dev	min	max
Births per 1000 people	4.6	4.4	0.2	34.3
Minimum temprature	0.4	1.4	-3.6	4.3
Quarterly average black smoke	185.9	53.7	100.6	350.7

Notes: Births data from the Registrar General for England and Wales. Temperatures measured in degrees Celsius. Weather data from the Met Office. Pollution data measured in microgrammes per meter cubed. Data from the Fuel Research Board.

Table 4: Regression Coefficients – Births Following the Smog

	(1) DD(1)	(2) DD(2)	(3) DD(3)	(4) DD(4)
Births following Smog	-0.09**	-0.09**	-0.09*	-0.09*
	(0.042)	(0.042)	(0.052)	(0.052)
Observations	789	789	789	789
Borough FE	no	yes	no	yes
Year FE	no	no	yes	yes

Notes: Dependent variable is births per one thousand people. Coefficients can be interpreted as the change in births per one thousand people. Robust standard errors are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. Data on births from the Registrar General for England and Wales, data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 5: Regression Coefficients – Infant Mortality Following the Smog

	(1) DD(1)	(2) DD(2)	(3) DD(3)	(4) DD(4)
Infant deaths in quarter of the Smog	0.01	0.01	0.01*	0.01*
	(0.006)	(0.006)	(0.007)	(0.006)
Infant deaths after the Smog	0.01	0.01	0.01**	0.01**
	(0.007)	(0.007)	(0.006)	(0.006)
Observations	789	789	789	789
Borough FE	no	yes	no	yes
Year FE	no	no	yes	yes

Notes: Dependent variable is deaths among infants less than one year old, per one thousand people. Coefficients can be interpreted as the change in infant deaths per one thousand people. Robust standard errors are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. Data on births from the Registrar General for England and Wales, data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 6: Regression Coefficients – Respondent is Male

	(1)	(2)	(3)	(4)
In Utero	-0.04		-0.03	
	(0.024)		(0.023)	
Infant	-0.06**		-0.05**	
	(0.024)		(0.023)	
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

Notes: Reported coefficients are differences-in-differences estimates of the change in the percentage of males in the sample, caused by exposure to the Great London Smog. Specifications (2) and (4) are omitted because the dependent variable contains individual information. Robust standard errors are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 7: Regression Coefficients – Respondent is Not White

	(1)	(2)	(3)	(4)
In Utero	-0.01*		-0.00	
	(0.006)		(0.005)	
Infant	-0.03***		-0.02**	
	(0.007)		(0.006)	
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

Notes: Reported coefficients are differences-in-differences estimates of the change in the percentage of people that are not white in the sample, caused by exposure to the Great London Smog. Specifications (2) and (4) are omitted because the dependent variable contains individual information. Robust standard errors are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 8: Regression Coefficients – Degree-Level Qualification

	(1)	(2)	(3)	(4)
In Utero	-0.02	-0.02	-0.03**	-0.03**
	(0.012)	(0.012)	(0.014)	(0.013)
Infant	-0.00	-0.00	-0.02	-0.02
	(0.019)	(0.018)	(0.020)	(0.021)
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

Notes: Reported coefficients are differences-in-differences estimates of the change in the percentage of people with a degree-level qualification, caused by exposure to the Great London Smog. Robust standard errors are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 9: Regression Coefficients – Unemployment

	(1)	(2)	(3)	(4)
In Utero 1971	0.00	0.00	0.00	0.00
	(0.009)	(0.010)	(0.011)	(0.011)
Infant 1971	-0.01	-0.01	-0.01	-0.01
	(0.007)	(0.007)	(0.008)	(0.008)
In Utero 1981	0.01	0.01	0.00	0.01
	(0.019)	(0.019)	(0.020)	(0.020)
Infant 1981	0.01	0.01	-0.00	0.00
	(0.013)	(0.014)	(0.013)	(0.014)
In Utero 1991	-0.01	-0.01	-0.01	-0.01
	(0.010)	(0.009)	(0.009)	(0.009)
Infant 1991	-0.03***	-0.02**	-0.02***	-0.02**
	(0.009)	(0.009)	(0.007)	(0.007)
In Utero 2001	-0.00	0.00	0.00	0.00
	(0.006)	(0.006)	(0.006)	(0.006)
Infant 2001	-0.00	0.00	0.00	0.00
	(0.007)	(0.007)	(0.007)	(0.007)
In Utero 2011	-0.01	-0.01	-0.01	-0.01
	(0.007)	(0.007)	(0.008)	(0.007)
Infant 2011	-0.01	-0.01	-0.02	-0.02
	(0.008)	(0.009)	(0.012)	(0.012)
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

Notes: Reported coefficients are differences-in-differences estimates of the change in the percentage of people who are unemployed, caused by exposure to the Great London Smog. Robust standard errors are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 10: Regression Coefficients – Hours Worked

	(1)	(2)	(3)	(4)
In Utero 1971	-8.17**	-7.85**	-8.03**	-7.78**
	(3.442)	(3.422)	(3.302)	(3.338)
Infant 1971	-4.95**	-4.19*	-4.33**	-3.78*
	(2.314)	(2.313)	(1.912)	(1.995)
In Utero 1991	-1.79***	-1.27**	-1.68**	-1.24*
	(0.666)	(0.601)	(0.662)	(0.652)
Infant 1991	-1.03	-0.09	-0.85	-0.05
	(0.650)	(0.658)	(0.786)	(0.773)
In Utero 2001	-1.31	-0.82	-1.17	-0.76
	(0.842)	(0.724)	(0.810)	(0.714)
Infant 2001	-0.22	0.65	0.01	0.76
	(0.958)	(0.942)	(0.891)	(0.837)
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

Notes: Reported coefficients are differences-in-differences estimates of the change in hours worked, caused by exposure to the Great London Smog. Robust standard errors are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 11: Regression Coefficients – Permanently out of Work due to Health

	(1)	(2)	(3)	(4)
In Utero 1971	-0.00***	-0.00***	-0.00*	-0.00*
	(0.001)	(0.001)	(0.001)	(0.001)
Infant 1971	-0.00	-0.00	-0.00	-0.00
	(0.002)	(0.002)	(0.002)	(0.002)
In Utero 1981	-0.00**	-0.01**	-0.00**	-0.00**
	(0.002)	(0.002)	(0.002)	(0.002)
Infant 1981	0.00	-0.00	0.00	-0.00
	(0.002)	(0.003)	(0.003)	(0.003)
In Utero 1991	-0.00	-0.00	-0.00	0.00
	(0.006)	(0.006)	(0.005)	(0.005)
Infant 1991	0.00	0.00	0.00	0.00
	(0.005)	(0.006)	(0.006)	(0.006)
In Utero 2001	-0.02*	-0.02	-0.02*	-0.02*
	(0.009)	(0.009)	(0.010)	(0.010)
Infant 2001	0.00	0.01	0.00	0.00
	(0.009)	(0.010)	(0.010)	(0.011)
In Utero 2011	-0.01	-0.01	-0.01	-0.01
	(0.013)	(0.013)	(0.013)	(0.013)
Infant 2011	-0.01	-0.01	-0.02	-0.01
	(0.010)	(0.011)	(0.012)	(0.012)
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

Notes: Reported coefficients are differences-in-differences estimates of the change in the probability of being permanently out of work due to illness, caused by exposure to the Great London Smog. Robust standard errors are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 12: Regression Coefficients – Cancer Diagnosis

	(1)	(2)	(3)	(4)
In Utero	0.00	-0.00	0.00	0.00
	(0.014)	(0.014)	(0.014)	(0.014)
Infant	0.03	0.02	0.03*	0.03*
	(0.017)	(0.017)	(0.016)	(0.016)
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

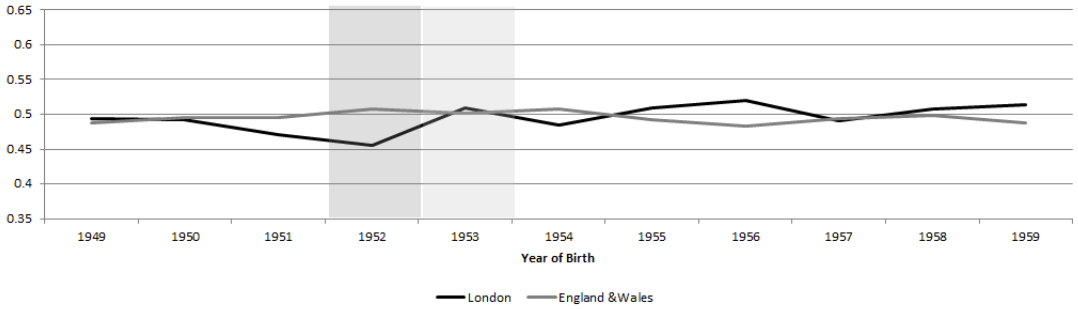
Notes: Reported coefficients are differences-in-differences estimates of the change in the probability of developing a cancer, caused by exposure to the Great London Smog. Robust standard errors are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Table 13: Regression Coefficients – Died before Age Sixty

	(1)	(2)	(3)	(4)
In Utero	-0.02***	-0.02***	-0.02***	-0.02***
	(0.006)	(0.006)	(0.006)	(0.006)
Infant	-0.02**	-0.02**	-0.02*	-0.02*
	(0.009)	(0.010)	(0.010)	(0.011)
Observations	459	459	459	459
Areas	51	51	51	51
Weather	yes	yes	yes	yes
Individual	no	yes	no	yes
Trends	no	no	yes	yes

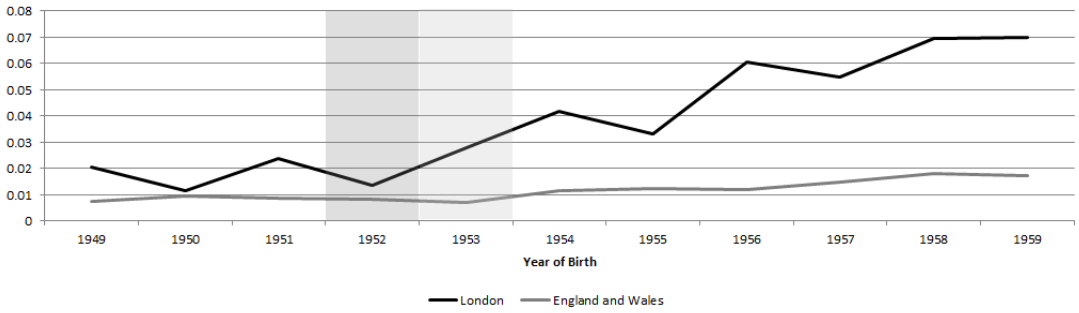
Notes: Reported coefficients are differences-in-differences estimates of the change in the probability of dying before 2011 (or the age of around sixty, for those affected by the smog), caused by exposure to the Great London Smog. Robust standard errors are in parentheses, *** p<0.01, ** p<0.05, * p<0.1. All specifications include controls for levels and squares of black-smog particulate matter exposure and temperature. Fixed effects are included for year of birth and area of birth. Some specifications also include information on gender and ethnicity in each cell, and London, England and Wales time trends. Source: Office of National Statistics Longitudinal Study. Data on weather from the MET office, data on black smoke pollution from the Fuel Research Board.

Figure 12: Respondent is Male



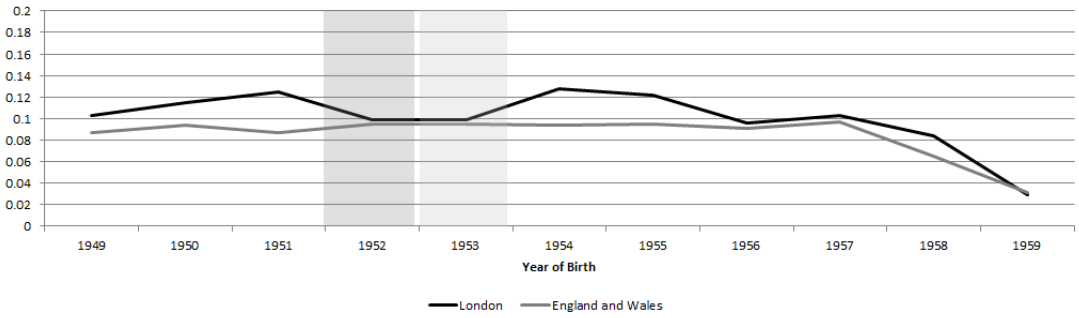
Notes: Those affected by the Great London Smog as Infants highlighted in Dark Gray. Those affected *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study

Figure 13: Respondent is Non-White



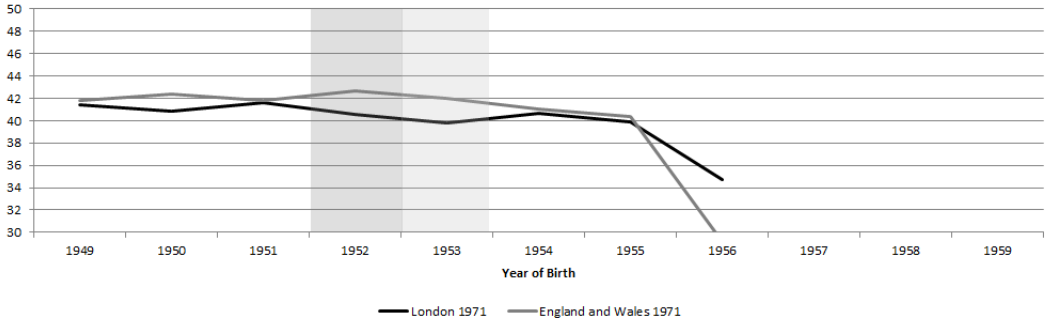
Notes: Those affected by the Great London Smog as Infants highlighted in Dark Gray. Those affected *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study

Figure 14: Respondent Has a Degree



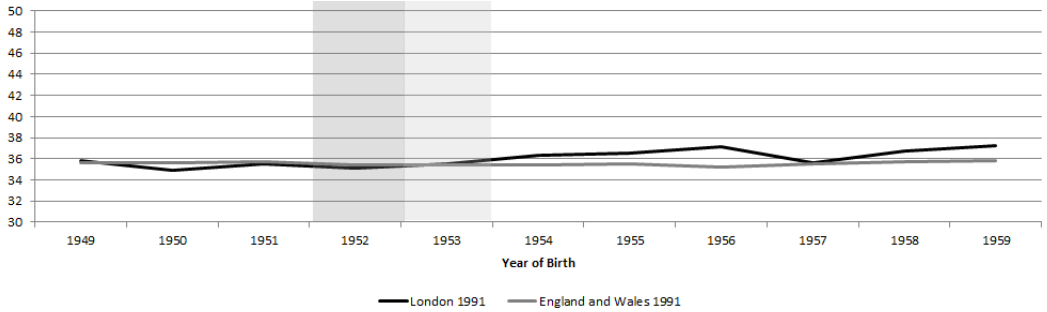
Notes: Those affected by the Great London Smog as Infants highlighted in Dark Gray. Those affected *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study

Figure 15: Hours Worked in 1971



Notes: Those affected by the Great London Smog as Infants highlighted in Dark Gray. Those affected *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study

Figure 16: Hours Worked in 1991

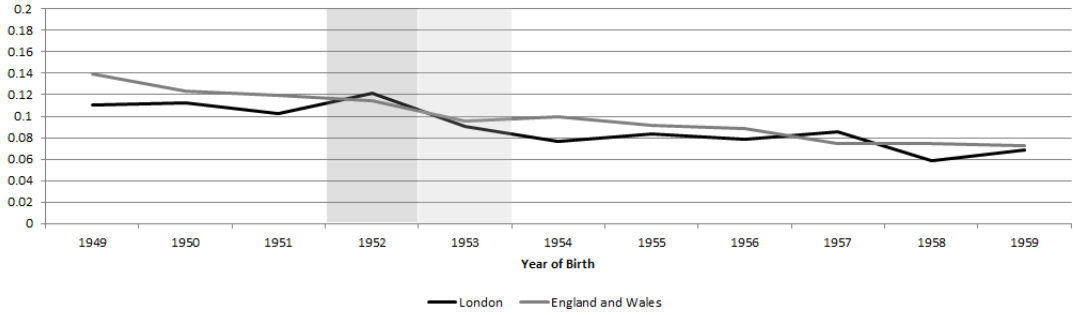


Notes: Those affected by the Great London Smog as Infants highlighted in Dark Gray. Those affected *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study

Figure 17: Hours Worked in 2001

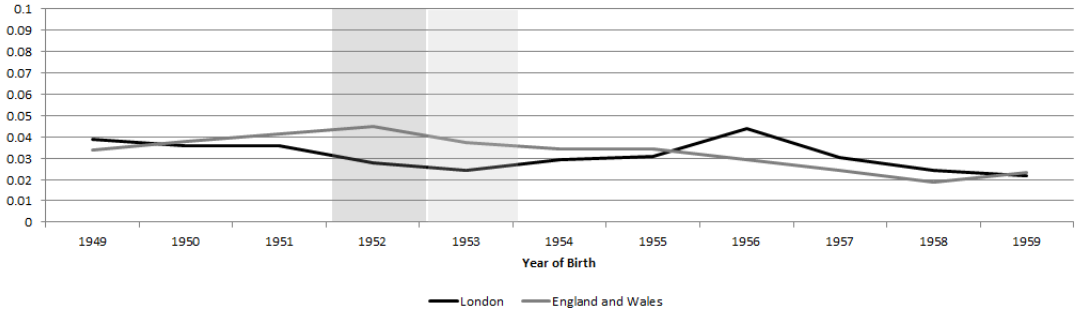
Notes: Those affected by the Great London Smog as Infants highlighted in Dark Gray. Those affected *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study

Figure 18: Respondent Has Registered Cancer



Notes: Those affected by the Great London Smog as Infants highlighted in Dark Gray. Those affected *in utero* highlighted in light gray. Data from the Office of National Statistics Longitudinal Study

Figure 19: Respondent Died Before 2011



Notes: Those affected by the Great London Smog as Infants highlighted in Dark Gray. Those affected *in utero* highlighted in light gray. The affected cohorts would have been around sixty in 2011. Data from the Office of National Statistics Longitudinal Study