What are the Long-term Effects of Prenatal Air Pollution Exposure? Evidence from the BHPS

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Abstract

The detrimental impacts of air pollution on human health are significant. High levels of pollutants can increase mortality in the elderly, reduce worker productivity, and decrease birth weights. While a consensus is beginning to emerge on the contemporaneous effects of air pollution on health, the long-term effects are still largely unknown. Because of data difficulties and omitted variable bias, it is usually difficult to determine how air pollution from years or decades earlier will affect current economic outcomes. Combining data from a comprehensive national survey with historical pollution data from the United Kingdom allows me to isolate the impact of prenatal particulate matter exposure on adult outcomes. I find that those with higher levels of exposure are more likely to be disabled and earn lower wages. There is also some evidence that the second trimester of gestation is when the fetus is the most vulnerable and there is a lower threshold for exposure to influence health.

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1 Introduction

The total effect of air pollution on human health remains a central question in environmental economics. The benefits of pollution are easily calculable; when a factory produces the number of workers employed or goods produced can be accurately measured and tabulated. Costs are more opaque. Beyond observable damages to the local environment, air pollution can cause short and long term negative impacts on people that vary from increased childhood asthma rate (Currie and Walker 2011) to increased mortality (Chay and Greenstone 2003). Unfortunately, the effects of pollution are notoriously difficult to isolate (Greven et. al 2011). Locations with higher levels of air pollution have lower housing prices (Chay and Greenstone 2005), leading to populations that may differ in pollution avoidance or other behaviors when compared to those that live in areas with higher air quality.[1] Through Tiebout sorting (Banzhaf and Walsh 2008), those with a greater distaste for pollution may vote with their feet and emigrate, biasing estimates. Also, pollution needs to be isolated from other determinants of health at both the individual and location level. A plethora of factors may affect the health of a population as well as air quality. The long-term effects of air pollution on health are especially difficult to quantify. Economists have yet to ascertain, for example, if prenatal air pollution exposure affects adult outcomes decades later. The British Household Panal Survey (BHPS), a longitudinal representative sample of the United Kingdom, combined with detailed pollution data can be used circumvents many of these issues.

The effects of in utero shocks on adults and even children are difficult to determine largely because of deficiencies in many data sets. As stated by Almond and Currie (2011), "...many prominent data sets, such as the Current Population Survey, do not include information on where someone was born or precise date of birth. As a result, many interesting and policy-relevant experiments linked to a certain time and place may never be analyzed." The BHPS is unique in that each respondent's date and location of birth are included. England is divided into over 300 local authority districts, many of which are less than 100 square kilometers.[2] The district of birth is asked of every respondent in the BHPS which can then be matched to detailed pollution data. This paper also uses a respondent's current district of residence, which is part of the confidential component of the BHPS. Using narrow location of birth as a proxy for general socioeconomic status and using current district to control for current air pollution exposure allows me to isolate the effect of prenatal exposure.

An advantage to analyzing the effects of air pollution on health in the UK is the presence of the National Health Service (NHS), which is a single payer healthcare system that covers all British citizens. Founded in 1948, by the beginning of the sample period the program had established the first nationally run insurance scheme in the world. In other countries there would be concerns that citizens who were exposed to worse air pollution also had worse health insurance. With universal health insurance, however, it is reasonable to assume that all survey respondents had access to roughly the same level of healthcare.

The BHPS data has been combined with air pollution data provided by the UK's Department of Food, Environment, and Rural Affairs (DEFRA). As part of the CAA of 1956, pollution monitors were established across England beginning in 1961.[3] By the 1970s, hundreds of monitors from around the country were taking daily readings of sulfur dioxide and black smoke, an early version of particulate matter. The monitoring network was used for decades before being slowly replaced by more comprehensive and automated systems in the 1990s. The monitoring network initially was focused in urban areas, which is where the majority of

[1] Air pollution may also have a larger effect on those in lower socioeconomic classes (Forastiere et al. 2007)

[2] The BHPS eventually covered the entire United Kingdom, but the first waves focused on England so only English respondents will be analyzed.

[3] The network would later be expanded to include Wales, Scotland, and Northern Ireland.

BHPS respondents are located.

After linking BHPS and air pollution data the results suggest that prenatal exposure does impact adult outcomes. Especially with regards to workers who file for disability or are not employed due to a long term sickness, there is a statistically significant and economically meaningful relationship to prenatal exposure. I find that a standard deviation increase in average black smoke exposure over the course of a pregnancy is associated with a two percentage point increase in the probability that a worker is disabled or long term sick. For measures of health and educational achievement, the second trimester appears to be particularly important. Even when controlling for first and third trimester exposure, second trimester pollution exposure is associated with worse health and lower educational attainment, and these results are statistically significant. There is also evidence that a threshold must be reached before pollution can affect health, perhaps because of the human body's fetal protections. This is some of the first evidence that prenatal air pollution exposure negatively impacts adult outcomes. The results are generally more attenuated then contemporaneous effects of exposure on adult health, but given the probable permanence of prenatal exposure the magnitudes are still economically significant.

The remainder of the paper is organized as follows. Section 2 discusses the epidemiology of air pollution and previous economic research. Section 3 describes the data from the BHPS and air pollution network. Section 4 details the empirical approach. Section 5 delineates the results. Section 6 concludes.

2 Epidemiology Background

The effects of air pollution on health has been a major focus of epidemiology and environmental economics over the last few decades. Epidemiologists have usually focused on comparing people who have lived in areas with differing amounts of air pollution, similar to this paper, while economists have sometimes used natural experiments to help reduce omitted variable bias. The effects of air pollution could be far reaching. Extreme pollution found in 1950s London or present day Beijing can cause immediate deaths (Zhang et al. 2007). Cities with consistently high levels of pollution may have much higher mortality rates compared to cleaner cities (Dockerty et al. 1993). Even everyday levels of pollution within current safety standards can increase mortality and morbidity levels in children and adults (Glinianaia et al. 2004). Beyond immediate effects, attention has also been paid to the possible effects of prenatal air pollution on birth outcomes. The following sections will focus on the various air pollution studies and the conclusions that have been drawn.

2.1 Pollutants and Biological Pathways

Many pollutants are thought to have adverse effects on human health. Historically, one of the biggest concerns has been particulate matter (PM) or total suspended particles (TSPs). Originally, most pollution monitors focused on the number of TSPs in the air of any size. As it became more apparent, however, during the 1970s and 1980s that smaller particles do more harm, TSP monitors were replaced by PM-10 readers.[4] According to the EPA, particulate matter is "a complex mixture of extremely small particles and liquid droplets. Particle pollution is made up of a number of components, including acids (such as nitrates and sulfates), organic chemicals, metals, and soil or dust particles." In recent years it has become more evident that particles of 10 micrometers are not as harmful as particles that have a diameter of 2.5 micrometers or less, and focus has shifted to PM-2.5. The counter-intuitive nature of PM is that smaller particles do

[4] PM-10 means that the monitor will record all particles that are 10 micrometers or less in diameter.

more damage (Oberdorster et al. 2005). Larger particles are more easily captured by bodily defenses, while smaller particles can make their way into the lungs. PM-2.5 is capable of passing through the lungs and directly into the blood stream.

Also of interest is ozone (O_3), nitrogen dioxide (NO_2), carbon monoxide (CO), and carbon dioxide (CO_2). The effects of these pollutants are not as clear and have not been studied as much as PM, but ozone in particular could be quite dangerous to human health. The main cause of ozone is motor vehicles, while the main cause of nitrogen dioxide is agriculture. Sulfur Dioxide (SO_2) is also a harmful pollutant, but human abatement activities have drastically reduced concentrations in the United Kingdom. Much of the CO emitted comes from cars, while the majority of CO_2 is emitted from fossil fuel use.

The biological mechanisms between air pollution and human health are not completely known.[5] Some relationships, such as between PM and lung cancer, are fairly straightforward. Particles containing dangerous chemicals that lodge in the lungs can interfere with cellular function and cause lung cancer (Pope et al. 2002). Other pathways remain largely unknown. Hernstadt and Muehlegger (2015), for example, show that neighborhoods that are downwind of highways have higher rates of violent crime compared to upwind neighborhoods. The mechanisms between vehicle created pollution and violent behavior is unclear.

The pathways between prenatal air pollution and fetal health are even less certain. The amniotic sac creates a buffer zone between the fetus and outside world, but if the mother is exposed to air pollution the pollutants might pass through (Perera et al. 1999). Smoking, for example, has widely been show to have strong effects on later infant health (Gilliland et al. 2001). Although a fetus is somewhat protected from outside pollutants, should pollutants penetrate the amniotic sac it will be vulnerable because of high rates of cell replication (Sram et al. 2005).

The dose response for air pollution exposure is thought to be linear (Ostro 1983), although this is not known with certainty, and almost all of the dose response research has only considered contemporaneous responses in adults. Schwartz and Zanobetti (2000) find that the dose response of PM on adult mortality is linear to the lowest levels recorded. These results have been replicated in further research and for other pollutants (Dominici et al. 2002; Jerrett et al. 2013). There is also some evidence that the dose response function of pollutants on low birth weight are also linear (Bobak 2000). To my knowledge, it has not been determined whether the dose response function of prenatal exposure on adult outcomes is concave, linear, or convex.

2.2 Contemporaneous Effects on Adults

Studies in both epidemiology and economics have largely focused on the immediate effects of air pollution on adult health. A growing body of research shows that increases in airborne pollutants not only increase mortality and morbidity, but have lesser effects as well. Janke et al. (2009) finds that a 10 $\mu g/m^3$ increase in PM-10 results in a 2.7 percent increase in adult mortality.[6] Heutel and Ruhm (2013) shows that air pollution could account for 30 percent of the already observed link between economic growth and mortality. The epidemiological literature generally finds that a 10 $\mu g/m^3$ increase in PM-10 increases adult mortality by one percent, and the effect is linear (Pope et al. 1995).

The costs of pollution on mortality and morbidity are large. Moretti and Niedell (2011) use traffic from the Port of Los Angeles as a source of exogenous variation of air pollution and calculate that the cost of

[5] Some research has been done by exposing human cells directly to pollutants in labs, but this may not be a good comparison for cells currently in human bodies. See Maier et al. (2008)

[6] $\mu g/m^3$, or micrograms per cubic meter, is the standard density measure of pollutants.

increased hospitalizations from ozone alone costs Los Angeles $44 million annually. Even localized pollution that comes from a single location like an airport can result in increased morbidity or mortality (Schlenker and Walker 2015). These costs are either implicitly or explicitly recognized by consumers; the marginal willingness to pay to avoid one additional unit of PM-10 has been estimated at approximately $100 (Bajari et al. 2012). A similar study in the United Kingdom finds that the marginal willingness to pay to avoid a one unit increase of carbon monoxide is £135 (Giovanis and Ozdamar 2014).

Beyond mortality and morbidity, air pollution may have lesser effects as well. Since the 1980s, there is evidence that TSP and other pollutants reduce productivity (Crocker and Horst 1981; Hausman et al. 1984), and reduce the accumulation of human capital (Graff Zivin and Neidell 2013). In order to calculate the true cost of pollution, it is important to establish if there is a gradient of effect. If pollution exposure does not result in death or hospitalization, it could still cause lower levels of productivity or minor health issues. Even when pollution is below dangerous levels, the effects could be large for those who work outdoors (Graff Zivin and Neidell 2012; Chang et al. 2014). Along with mortality, even the closure of one large emitter can increase productivity by an economically significant amount (Hanna and Oliva 2015). These results show that even when air pollution is at relatively low levels, the economic costs could be large. The question remains, however, if there are long-term pollution effects as well as contemporaneous effects.

2.3 Contemporaneous and Long-term Effects on Children

While children may not have the same mortality rates as adults when exposed to pollutants, their developing mental faculties may be more vulnerable. Lavy et al. (2014) shows that even same day pollution can affect high school students, who perform worse on tests on days with higher levels of PM-10 and CO. Physically, asthma is one of the greatest concerns; high levels of CO increase asthma-related hospital admission drastically (Neidell, 2004). High levels of PM also increases respiratory-related issues in children (Romieu et al. 2007). Lleras-Muney (2010) uses the unique moving habits of military families and determines that ozone could increase respiratory related hospitalizations by 24 percent. The long-term effects of childhood exposure, while more difficult to analyze, help determine the true cost of pollution. Beatty and Shimshack (2015), shows that carbon monoxide can cause respiratory issues in children a year after exposure. CO is also particularly dangerous to infants (Currie and Neidell 2005; Currie et al. 2009).

2.4 Prenatal Air Pollution

Both epidemiologists and economists have focused on prenatal air pollution in recent years.[7] Using low birth weight or other birth related dependent variables is useful because of data availability and lack of omitted variable bias. Although there is increasing evidence that exposure to air pollutants while in utero does have adverse impacts, many of those effects appear to be small and more research is needed (Maisonet et al. 2004). For example, beyond birth indicators, pollution could increase the likelihood that a child develops autism (Roberts et al. 2013). The timing of pollution exposure is also important. As the fetus develops during gestation, it may be more vulnerable during certain times than others.

It is unclear when in a pregnancy air pollution does the most damage. Using the 2008 Beijing Olympic pollution restrictions as a natural experiment, Rich et al. (2015) finds that the biggest changes in birth weight are during the eighth month of pregnancy. Other papers have found that the fetus is most vulnerable

[7] Currie et al. (2013) focuses on the economic research, while Ruckerl et al. (2011) details the epidemiology. Here I will synthesize both fields.

during the first trimester (Ha et al. 2001; Lee et al. 2013), the first and third trimester (Ritz et al. 2007), or that the effect is consistent across trimesters (Dadvand et al. 2013). The mixed results so far illustrate the lack of consensus and the benefits of further study.

Although more difficult to determine because of omitted variable concerns, there has been some research into how prenatal pollution effects childhood or adult outcomes. Bharadwaj et al. (2014) uses within sibling variation to determine that exposure to CO while in utero reduces fourth grade test scores. TSP has a similar effect on high school students (Sanders 2012). Isen et al. (2015), finds that prenatal exposure decreases adult labor force participation and wages in the United States. Isen et al. differs from this paper in several ways. First, the scope of this paper is considerably smaller; the number of survey respondents that can be accurately matched to pollution data numbers around 1,000, compared to millions of observations that Isen et al. uses across the United States. This paper also is not able to rely on an exogenous policy shock as an instrumental variable, a key strength of Isen et al.'s identification strategy. The advantages to my approach are three-fold. First, English districts are less than a tenth of the size on average of US counties; this enables a much more accurate measurement of pollution exposure. Second, the BHPS asks a much larger battery of questions than the US Census data, allowing me to test a series of different outcome variables. Third, restricted BHPS data includes the current district of residence, not just of birth, so respondents current exposure to pollution can be accounted for.

2.5 Contribution

This paper adds to the literature on air pollution in several ways. First and foremost, along with Isen, this is one of the first papers to examine the effects of prenatal air pollution on adult outcomes. This is important because the total costs of air pollution need to be established. It is possible that prenatal air pollution adversely affects birth outcomes, as the literature shows, but it is also possible that beyond birth there are no detrimental effects. If there is a strong initial culling effect at birth with increased mortality but no long term impacts, then the established literature has calculated the majority of the cost. On the other hand, there could be a wide spectrum of effects caused by prenatal air pollution. Along with increased mortality at birth, some could suffer from decreased mental cognition or increase susceptibility to illness. This can only be determined by linking prenatal exposure to adult outcomes. If prenatal air exposure is related to an increased probability of disability or ill health, then the true costs of air pollution are much larger than originally thought.

Secondly, the timing of prenatal exposure is important to understand. As shown above, various research has concluded that any or all three trimesters could be a time of fetal vulnerability. It is also possible that different pollutants have the largest impacts at different times or that different aspects of development are most vulnerable to retardation during different trimesters. This paper does not seek to answer all these questions, instead, I will attempt to establish when during gestation particulate matter partially determines adult outcomes. Although I find that the second trimester is the most important, it does not mean that other literature is incorrect or incomplete. Rather, given that most of the trimester literature has focused on birth outcomes, as gestation progresses various aspects of development could have different periods of peak vulnerability.

3 The Data

The British Household Panel Survey is the largest of its kind in the United Kingdom. Begun in 1991, Wave 1 surveyed 10,300 people drawn from 5,500 households located in 250 areas around Great Britain. The survey was expanded in 1999 to include more responses from Scotland and Wales, and again in 2001 to include more respondents from Northern Ireland. Subjects 16 years or older take the entire survey, while those 11-15 will take an abbreviated youth survey.[8] The vast majority of the surveys are taken in person at the respondent's house, although sometimes the survey is done by telephone and occasionally by proxy.[9] The survey covers a wide range of issues, from simple demographic information to health outcomes, labor status, and how the subject feels about the current state of Great Britain. The survey has changed over the years and expanded, but many of the questions that were asked in 1991 were still being asked during the 18th and final wave of the survey in 2008.[10]

The original subject pool consisted of every member of 5,500 households. All first wave respondents are designated Original Sample Members (OSMs). All OSMs are followed for all years of the sample unless they become deceased or move away from the United Kingdom. If an OSM moves from one household in the UK to a second household, then all members of their new household are also included so long as the OSM still lives with them. These respondents are denoted Temporary Sample Members (TSM). If an OSM has a child with a TSM, the child becomes an OSM and the TSM becomes a Permanent Sample Member (PSM), and will continue to be surveyed each year regardless of their relationship status with the OSM. The BHPS makes a concerted effort to follow up with subjects every year, and the household nature of the subject pool allows survey takers to keep track of OSMs that may frequently move. This process means that the subject pool slowly evolves over time as respondents die, divorce, remarry, have children, and move. In total around 32,000 people have taken part in the BHPS, although only 5,000 participated in all 18 waves.

The BHPS has become more comprehensive over time, but the core questions asked for the purposes of this paper have been part of the survey in all 18 waves. The key variable that allows me to account for many omitted variables is "district of birth". By knowing the date and exact location of birth, I can accurately determine the amount of pollution that each person was exposed to during gestation. The adult outcome variables were not asked in every wave and the number of respondents who answered the question vary, but given the large size of the survey there are usually at least 1,000 people answering the same question in different survey waves.

England is divided up into 326 districts (see Figure 1).[11] Each district elects a district council that has local governmental powers. About 60% are known as non-metropolitan districts, and the rest are either unitary authorities, metropolitan districts, or London boroughs. There are slight changes in how much power each type of district has, but the main distinction is the urban versus rural nature of the different categories. The districts included in this paper were chosen based on size and population (see Figure 2). Smaller districts will provide more accurate air pollution exposure data because the exact place of birth is not included. Districts with higher populations will increase the sample size. In all, pollution data from 68 districts is matched with the BHPS data. This covers roughly half of the BHPS survey respondents. Several now distinct districts were one district when the BHPS began, however those districts were usually divided into one large district and one small district. Pollution monitors from the larger current district are used

[8] The youth questionaire began in 1994.

[9] Proxy respondents are given an abbreviated survey. They are not asked questions such as, "How has your general health been over the last 12 months" or other questions that may not be answered accurately.

[10] In 2008 the BHPS became the Understanding Society survey, which can be linked to the BHPS.

[11] Occasionally a district will split or two will merge, but this is rare.

and assigned to survey respondents from the older joint district.

Tables 1 and 2 show basic statistics for the districts of birth included in the sample. About half of the districts make up the inner and outer boroughs of London; the other half are from other metropolitan areas that had large numbers of BHPS respondents. Urban areas are used because of more extensive and earlier pollution monitor coverage and they are generally smaller. The medium size of all English districts is 212 km^2, while the medium size for districts in the sample is only 72 km^2 (see Table 3). Most of the districts have a relatively small number of observations, which is indicative of the nationally representative nature of the BHPS; only eight districts have three or more percent of all of the observations and the median district only contains 1.11 percent of all observations. These districts, because of their urban nature, have high populations and are dense; the median district has about 266,000 people total and over 4,000 people per km^2.

Concerns about air quality go back centuries in the United Kingdom. During the Middle Ages King Edward I banned coal burning, indicating that even without instrumentation the adverse impacts of particulate matter were noticeable. In the 17th century an adviser to King Charles II suggested planting large gardens throughout the city to deal with pollution. As the United Kingdom became more urban during the 20th century, pollution problems increased. London became famous for its "pea-soupers", dense fogs that were tinged yellow-green by coal smoke. The city's topography makes it prone to large temperature inversions, where emitted particulate matter become trapped under a layer of hot air near ground level.

Events came to a head in 1952, when atmospheric conditions and temperatures combined to trap airborne pollutants over the city from December 5th to December 9th. For five days lights had to be kept on during the daytime, cars were abandoned on curbs, and the fog seeped into houses and other buildings. As seen in Figure 3, visibility declined dramatically. In the following days the smog's death toll was put at 4,000.[12] The country was finally galvanized to action, and several years later the world's first Clean Air Act (CAA) was signed into law (Brimblecombe 2006). Although air pollution would continue to be a problem for years to come, severe pollution events like the Great Smog of 1952 would become increasingly rare, and a network of pollution monitors were set up across the country.

In 1961 the Government of the United Kingdom established the word's first coordinated national air pollution network, called the National Survey. The National Survey established monitors throughout Great Britain and monitored levels of Sulfur Dioxide and Black Smoke (BS). Black smoke can be thought of as a measure of particulate matter. Mainly generated by fuel combustion when coal was the main source of heat for British families and then diesel fuel from cars, monitors would record how much filters visibly darkened each day and convert that to a pollution density.[13] Over the decades the National Survey network expanded until hundreds of stations were active throughout the UK. Beginning in the 1990s the National Survey was replaced by new automated networks that could take readings of many different pollutants. Air pollution data is provided by the Department of Environment, Food, and Rural Affairs (DEFRA).

After matching air pollution data with the BHPS, the final data set has 23,964 completed survey questionnaires from 3,442 different respondents.

[12] It has since been increased to 12,000 (Bell et al. 2004).

[13] Although there are some black smoke monitors still in use, focus has shifted to particulate matter 10 and 2.5. Also, particulate matter is no longer distinguished by how much it darkens a filter.

4 Empirical Approach

The greatest concern in determining the effects of air pollution on human outcomes is socioeconomic motivated endogeneity concerns. Those that live in areas with low air quality are going to differ from those that live in areas with high air quality along multiple dimensions. To address this issue, I leverage detailed location data included in the BHPS in order to examine within area changes of pollution. In order to isolate the effect of prenatal air pollution on adult outcomes the following specification is used:

$$y_{idtcm} = \alpha + \beta Pollution_{dt} + X_i^{'}\Gamma + \theta_d + \Xi_c + \eta_m + \epsilon_{idtcm}$$

Where outcome y_{idtc} is one of several physical or cognitive outcomes for person i born in district d, at time t, currently lives in district c, and was interviewed in month-year m.[14] The variable of interest is β, which could be positive or negative depending on the outcome variable. X_i is a vector of individual characteristics such as year of birth. θ_d is a vector of district of birth fixed effects and Ξ_c is a vector of current district of residence fixed effects. For all specifications standard errors are clustered by survey respondent because most respondents are interviewed multiple times and their responses could be correlated across survey wave.

What allows me to reliably identify the effect of prenatal exposure on the outcome variables is the inclusion of the district of birth and current district. Places with low air pollution have cheaper home prices and poorer residents (Chay and Greenstone 2005). Therefore the correlation between pollution and these outcome variables is expected to be negative, but this could be because of the lower socioeconomic status of neighborhoods with high pollution, not the pollution itself. By including district of birth fixed effects, any time invariant neighborhood conditions are accounted for. Instead, all pollution effects are determined for those who were born within a given district.

It is also possible that those born in districts with worse air pollution will later move to districts that also have low air quality. If this is the case, any correlation between prenatal exposure in adult outcome could be misleading. It would be impossible to say whether the current air quality or previous air quality was causing health issues. To address this issue, I have gained access to the restricted BHPS data that includes current district of residence as well. A vector of current district of residence fixed effects and interview month fixed effects allows me to account for those living in areas with poor air quality.

Districts could still change overtime as areas gentrify or decay. Some of the other control variables help account for this. Year of birth fixed effects account for nationwide economic conditions during the year of birth. Current district of residence will also help; those of higher or lower socioeconomic status will tend to cluster in similar districts. The greatest threat to the identification strategy is if those who are born in district-years with high pollution then live in districts with higher pollution relative to those born in the same district and the same year but at times of lower pollution. While possible, this seems unlikely given that consumers would not have up to date information on changing pollution levels in various districts.

4.1 Measures of Air Pollution Exposure

The lack of consensus of just how air pollution impacts human health and uncertainty about the dose response curve suggests that several different measures of air pollution exposure should be analyzed. One key dimension is average exposure versus maximum exposure. As seen during the Beijing Olympics and in

[14] The BHPS surveys are taken every year beginning in September. About 90 percent of the interviews take place by November 30th, but some are not completed until May of the following year. Because some variables, such as number of visits to the doctor, are given from a set start date, it is important to control for what month a survey was given to each respondent.

the aftermath of the Chernobyl meltdown (Zhang et al. 2007; Almond et al. 2009), even a brief pollution event can cause large effects. High levels of fine particulate matter may cause damage over a matter of hours. On the other hand, long term exposure at above average but not extreme levels of pollution may be damaging as well. If the dose response curve is linear without a lower threshold, then the average pollution at any level could be damaging. Different combinations of maximums and averages are used to help establish if exposure in various forms affect adult outcomes.

I use three different measures of air pollution across pregnancy. The first, denoted "Avg. BS Preg.", is an average of black smoke exposure across all days and all monitors in any given district. An average pollution reading is then assigned to each person based on their district and month of birth. The second, "MAM BS Preg." is a combination of maximums and averages.[15] First, the highest pollution reading in a district on a given day is recorded. Then those highest readings are averaged across a month. Finally, each person in the sample is assigned the maximum of those monthly averages. The third measure over an entire pregnancy, "MAA BS Preg.", finds the average reading of all monitors over a given day, averages those readings over a month, and then uses the maximum month from up to nine months prior to birth. For trimester exposure, the equivalent of a total average and a maximum average month of average daily readings are used. Although the trimesters are correlated with one another, all measures are below the .70 cutoff recommended by Tabachnick et al. (2001) to avoid multicollinearity concerns.

Table 4 shows the air pollution summary statistics. Most notable is the right-tailed dispersion of the data. As seen in Figure 4, most nine month periods have a low amount of black smoke in the air, but in some districts pollution will spike to high levels. At the other end of the spectrum, for much of England during the sample period air quality is quite good. Although direct comparisons between black smoke and PM-10 or PM-2.5 are not possible, 10 percent of the district-pregnancy observations have under 20 µg/m^3 of black smoke. For comparison, 35 or less µg/m^3 of PM-2.5 and 50 or less µg/m^3 of PM-10 are considered safe.

4.2 Outcome Variables

The expansive nature of the BHPS allows me to test several different measures of adult outcomes. These variables have been chosen to examine possible cognitive and physical impacts of prenatal pollution exposure. The first is whether a respondent's job status is given as long-term sick or disabled.[16] This is the most "severe" of the dependent variables in that the costs of having people who wish to work but are physically unable is high for society. As an alternative labor outcomes, wages and unemployment are also examined.

The third outcome studied is an indicator variable for whether or not self-reported health is poor. If a respondent self-reports their health over the last twelve months to be poor or very poor then the indicator is assigned to be one, while if an overall health of excellent, good, or fair was given the indicator variable is assigned to be zero. An alternative health variable is whether a respondent went to the doctor frequently in the last year.[17] 34 percent of respondents said that they went to the doctor three or more times in the last year, and those respondents are assigned to the high number of doctor visits group. Number of visits to the doctor is less subjective than overall health status, but the BHPS does not contain any additional

[15] The first "M" or "A" indicates maximum or average month over the course of a pregnancy. The second letter indicates maximum or average day in a month. The third letter indicates the maximum or average monitor reading on any given day.

[16] The options respondents can choose from are self employed, employed, unemployed, retired, family care, full time student, long term sick/disabled, on maternity leave, government training scheme, or something else.

[17] The exact question is how many times a respondent went to their general practitioner since September 1st of the previous year.

information about why a respondent made an appointment.

Finally educational achievement is used as a dependent variable. Respondents were asked to give their highest level of educational achievement: higher degree (post-grad), first degree (bachelors), HND or HNC (trade degree), A-level (qualification to move to higher education), O-level (similar to high school qualification), or CSE (alternative to the O-level). Those that received a bachelors or higher are assigned to the "higher ed" group in the corresponding indicator variable.[18]

Table 5 shows summary statistics for the different outcome variables. Wages, the only variable that is not binary, has a similar data distribution to the different measures of BS; it is skewed right. It is important to note, however, that the wage at last pay period could be misleading because respondents had different pay period lengths. This concern will be addressed with extra controls that are explained in the following section. Only about 1 percent of the survey responses said they were disabled, while five percent said they were suffering from poor or very poor health. 32 percent visited their general practitioner three or more times in the past year, while 27 percent have at least the English equivalent of a bachelors degree.

5 Results

For all specifications with a binary dependent variable Probit models were used. Probit models are preferred to linear probability models because for many of the specifications there are a large number of observations that are predicted to be outside of the zero-one probability range when linearity is assumed.[19] This is especially the case with the probability of an observation being reported as less than zero percent. This is evidence that there is a lower threshold for air pollution to impact prenatal health. While the literature thus far has shown a linear dose response curve, the results are based on adult mortality. Most of the adults who die during high air pollution days are already frail so it is sensible that there would be a steady increase in deaths as those weakest die first when air pollution increases. Given the natural protections that a mother's body will create for a fetus, however, it could be that a low level of air pollution will not cause harm. Eventually a threshold is reached, and then air pollution will retard physical or mental development. This subject is an avenue for further study. One of the disadvantages of using a Probit model is that each indicator group must include both possible values of the binary outcome variable. For example, any district of birth or other variable that is part of the indicator vectors without at least one respondent that is both disabled and not disabled will not be included. In a linear probability model these observations will not contribute to the coefficients but will affect the standard errors.

In each specification the pollution measures have been transformed to have a standard normal distribution. This is done to ease interpretation of the coefficients. Unless otherwise noted each dependent variable is regressed on the measure of air pollution and vectors of indicator variables for the district of birth, district of current residence, interview month-year, month of birth, year of birth, and race of respondent. All standard errors are clustered at the respondent level.

[18]See Appendix A for details of the original BHPS variables that disabled, poor health, doctor visits, and higher education were derived from.

[19]Linear probability model results are available upon request by the author.

5.1 Disability

Table 6 displays the results where disability status is the dependent variable. Panel A includes the full sample, while Panel B is restricted to any districts that are less than 200 km^2.[20] Larger districts may not have as accurate of pollution readings, and without more specific location data on place of birth there is no way to determine how far monitors are from residences. The coefficient in Column 1 Panel A indicates that a standard deviation increase in average black smoke across an entire pregnancy corresponds to a 1.44 percentage point increase in the probability that an individual is disabled or long-term sick. This result is statistically significant at the 10 percent level. Using the max-average-max and max-average-average measures of black smoke results in similar magnitudes in Columns 2 and 3. Columns 4 and 5 use each trimester as the variables of interest. The only statistically significant result is average black smoke exposure during the second trimester.

In Panel B the results are quantitatively similar to Panel A. Columns 2 and 3 indicate that a standard deviation increase in exposure leads to a two percentage point increase in probability of disability or long-term sickness and the results are statistically significant at the five percent level. This increased magnitude and statistical significance could indicate that the larger districts contain classical measurement error, which would attenuate the true effect. In Column 4 the second trimester of total average exposure is once again statistically significant at the 10 percent level.

While these magnitudes are not large, a few considerations should be kept in mind. First, as already mentioned, the distribution of pollution data is skewed to the right. For example, the top one percent of respondents were exposed to over 1000 µg/m^3 of black smoke using the max-average-max definition of pollution. This is more than five times the average exposure, and corresponds to a 10.5 percentage point increase in probability of being disabled or long-term sick compared to a person exposed to the average amount of black smoke. Also, aggregating over the entire United Kingdom which has a labor force of approximately 33 million, a standard deviation increase in black smoke would increase the number of those either disabled or long-term sick by 693,000.

Table 7 separates the data by those that were born in London (Panel A) and those that were born in the rest of the England (Panel B). Districts in London are useful because they are much smaller than the average district and generally have some of the most comprehensive pollution readings.[21] As seen in Columns 1, 2, and 3, a standard deviation increase in black smoke increases the probability that a worker says he/she is disabled or long term sick by 10.6-11.5 percentage points, and those results are all statistically significant at the one percent level. When examining the impact by trimester, the second trimester has the largest effects. While the entire pregnancy results are highly statistically significant and depend on 1,055 surveys, they only depend on about 93 individuals and should be used with caution. When only the rest of England is used the results are similar to those from the entire sample.

5.2 Unemployment and Wages

For two alternative measures of employment I use unemployment and wages. Table 8 displays the results when the outcome variable is whether the respondent is unemployed at the time of the survey. In both the full sample and small district sample the results are generally not statistically significant and the the results

[20]The large districts omitted are Doncaster (568 km^2), Sheffield (368 km^2), Birmingham (268 km^2), Bradford (366 km^2), Leeds (552 km^2), Wakefield (339 km^2), Broadland and Norwich (591 km^2), Oxford; Vale White Horse; and West Oxford (1338 km^2), Babergh; Ipswich (633 km^2) and Thamesdown (230 km^2).

[21]The average size of a London Borough is 50 km^2, or about 85% of the size of Manhattan Island.

have small magnitudes. It is unknown why there does not appear to be a relationship between prenatal air pollution and unemployment. Perhaps those that do not work because of bad health would file for disability or leave the work force entirely instead of staying unemployed.

There are additional omitted variable concerns about wages, but the detailed nature of the BHPS makes it possible to circumvent most of them. The question regarding wages in the BHPS asks, "The last time you were paid, what was your gross pay - that is including any overtime, bonuses, commission, tips or tax refund, but before any deductions for tax, national insurance or pension contributions, union dues and so on?". If the most recent paycheck amount is atypical compared to the usual amount, the results would be biased. Given that a large number of the surveys are given during November and December, those employed in retail could have higher wages than usual during the Christmas shopping season. Fortunately, a later question asks, "Your take home pay last time was ________. Is this the amount you usually receive (before any statutory sick pay or statutory maternity pay)?" Only those that responded yes are included in the sample. A second concern is the differing lengths of pay periods.[22] Those that are paid monthly could have a higher pay listed than those paid biweekly even if their hourly wage is lower. Pay period is also asked during the survey, and the length of pay period is used as an additional control. Finally, the BHPS divides each job into 19 different job types and this is used as an additional control.[23]

The results from the specifications where wages is the dependent variable are shown in Table 9. Once again Panel A contains the full sample and Panel B restricts the sample to small districts. Columns 1, 2, and 3 show large decreases in wages for those exposed to high levels of prenatal air pollution; one standard deviation increase in black smoke corresponds to a possible loss of £223 per pay period. This result does not appear to depend on when during a pregnancy the air pollution was highest. The results attenuate dramatically and lose statistical significance when the large districts are omitted from the sample. It is unclear why this is the case as smaller district should produce more precise results.

5.3 Poor Health and Doctor Visits

Table 10 displays results from using overall health as the dependent variable. Each respondent was asked, "Please think back over the last 12 months about how your health has been. Compared to people of your own age, would you say that your health has on the whole been ..." and respondents that answered poor or very poor are assigned to have poor health. The results of the three air pollution variables that cover an entire pregnancy are the expected sign but are not statistically significant. When dividing black smoke exposure by trimester, the second trimester has the correct sign and is statistically significant. The coefficients indicate that a standard deviation increase in black smoke during the second trimester corresponds to a two percentage point increase in the probability that a respondent suffered from poor or very poor health over the last 12 months.

Table 11 displays the results when a high number of doctor visits over the last year is the dependent variable. The results are generally the expected sign but not statistically significant. Interestingly, once again the second trimester seems to be the most significant. In Panel A Columns 4 and 5, a standard deviation increase in black smoke during the second trimester implies a three percentage point increase in the probability of a high number of doctor visits. These coefficients maintain a similar magnitude and statistical significance when only small districts are used.

[22] 51 percent of respondents gave their average pay period as a month, 29 percent one week, eight percent four weeks, and two percent two weeks. Eight percent also gave their annual pay instead of a pay period.

[23] See Appendix C for a list of the job types and their frequency in the data.

5.4 Higher Education

As a final test a respondent's educational achievement is regressed on pollution exposure. This variable is slightly different from the rest in that the result does not vary within respondent; the highest educational level achieved at any time during the survey period is given as their educational achievement. Results are shown in Table 12. Mirroring previous results, the entire pregnancy variables are not statistically significant, but all are the expected sign. Also as seen previously, the second trimester has negative results that are statistically significant. The coefficient in Panel A Column 4 indicates that a standard deviation increase in prenatal black smoke exposure during the second trimester corresponds to a six percentage point decrease in the probability that the respondent attains a college degree. This is, however, balanced out by the positive and statistically significant coefficient during the third trimester.

6 Discussion

This paper provides some of the first evidence linking prenatal air pollution exposure to adult outcomes. By using detailed geographic information provided by the BHPS combined with the world's first nationwide pollution monitor network, I am able to leverage large changes in particulate matter across England and link that to labor market, health, and educational outcomes decades later. Furthermore, having access to district of birth and district of current residence allows me to have a measure of general socioeconomic status and current exposure to determine the effect of prenatal exposure.

I find that prenatal particulate matter exposure does have impacts on adults. A standard deviation increase in black smoke exposure results in a 1.5 percentage point increase in the probability of being disabled or long term sick. Given the skewness of the data, that makes some individuals in high pollution areas much more likely to have long term issues from prenatal exposure. Those with more exposure also have lower wages when compared to those who gestated during lower pollution periods. A standard deviation increase in exposure is associated with up to £223 less per pay period.

The remaining outcome variables are not significantly associated with the full pregnancy pollution measures. However, there is a consistent negative association with pollution exposure during the second trimester. A standard deviation increase in average black smoke exposure during the second trimester results in a six percentage point decrease in the probability of having a college degree, a three percentage point increase in the probability of visiting the doctor three or more times, and a two percentage point increase in the probability of being in poor health. All of these results are statistically significant at the one percent level.[24]

This provides some evidence that the second trimester may be the most important time of development. Previous research has indicated that the first trimester is when the fetus is most vulnerable, but these results are not necessarily mutually exclusive. Different phases of development could be the most vulnerable at different times. Previous research has focused on birth outcomes, which are going to be largely influenced by the health of the mother. Perhaps the mechanisms that determine birth weight and preterm birth are susceptible during the first trimester, but cognitive development will be most impacted during the second trimester. There is also evidence that there is a lower threshold for prenatal exposure affecting adult outcomes. This is in contrast to the literature on adult mortality and morbidity. Once again, the results here do not contradict with previous research. The protective measures that shield a fetus from contamination could establish a threshold that adults in the outside world do not have. This provides good news for

[24]Occasionally the first or third trimester results exhibit the perverse sign, but not consistently across outcome variables.

abatement programs; once a certain (still unknown) level is reached, prenatal exposure will no longer be a large issue.

Neither of these are firm conclusions; economics and epidemiology would greatly benefit from further study. There are still concerns about omitted variable bias and the results here are not always consistent. Data difficulties and privacy concerns will continue to make it difficult for researchers to definitely establish the amount of exposure alone, and linking this information to future outcomes is difficult. This is an important early step, however, in determining the impacts that prenatal air pollution exposure has on adult outcomes.

References

[1] Almond, D., and Currie, J. Killing me softly: The fetal origins hypothesis. *The Journal of Economic Perspectives: A Journal of The American Economic Association 25*, 3 (2011), 153.

[2] Almond, D., Edlund, L., and Palme, M. Chernobyl's subclinical legacy: Prenatal exposure to radioactive fallout and school outcomes in Sweden. *The Quarterly Journal of Economics 124*, 4 (2009), 1729–1772.

[3] Bajari, P., Fruehwirth, J. C., Kim, K. I., and Timmins, C. A rational expectations approach to hedonic price regressions with time-varying unobserved product attributes: The price of pollution. *The American Economic Review 102*, 5 (2012), 1898–1926.

[4] Banzhaf, H. S., and Walsh, R. P. Do people vote with their feet? an empirical test of Tiebout's mechanism. *The American Economic Review* (2008), 843–863.

[5] Beatty, T. K., and Shimshack, J. P. Air pollution and children's respiratory health: A cohort analysis. *Journal of Environmental Economics and Management 67*, 1 (2014), 39–57.

[6] Bell, M. L., and Davis, D. L. Reassessment of the lethal London fog of 1952: Novel indicators of acute and chronic consequences of acute exposure to air pollution. *Environmental Health Perspectives 109*, Suppl 3 (2001), 389.

[7] Bharadwaj, P., Gibson, M., Zivin, J. S. G., and Neilson. Gray matters: fetal pollution exposure and human capital formation. *NBER Working Paper* (2014).

[8] Bobak, M. Outdoor air pollution, low birth weight, and prematurity. *Environmental Health Perspectives 108*, 2 (2000), 173.

[9] Brimblecombe, P. The Clean Air Act after 50 years. *Weather 61*, 11 (2006), 311–314.

[10] Chang, T., Zivin, J. S. G., Gross, T., and Neidell, M. J. Particulate pollution and the productivity of pear packers. *NBER Working Paper* (2014).

[11] Chay, K. Y., and Greenstone, M. The impact of air pollution on infant mortality: evidence from geographic variation in pollution shocks induced by a recession. *Quarterly Journal of Economics 118*, 3 (2003), 1121.

[12] Chay, K. Y., and Greenstone, M. Does air quality matter? evidence from the housing market. *Journal of Political Economy 113*, 2 (2005).

[13] Crocker, T. D., and Horst, R. L. Hours of work, labor productivity, and environmental conditions: A case study. *The Review of Economics and Statistics* (1981), 361–368.

[14] Currie, J., and Neidell, M. Air pollution and infant health: What can we learn from California's recent experience? *Quaterly Journal of Economics*, 3 (2005), 1003–1030.

[15] Currie, J., Neidell, M., and Schmieder, J. F. Air pollution and infant health: Lessons from New Jersey. *Journal of Health Economics 28*, 3 (2009), 688–703.

[16] Currie, J., and Walker, R. Traffic congestion and infant health: Evidence from E-ZPass. *American Economic Journal: Applied Economics 3*, 1 (2011), 65–90.

[17] Currie, J., Zivin, J. S. G., Mullins, J., and Neidell, M. J. What do we know about short and long term effects of early life exposure to pollution? *NBER Working Paper* (2013).

[18] Dadvand, P., Parker, J., Bell, M. L., Bonzini, M., Brauer, M., Darrow, L. A., Gehring, U., Glinianaia, S. V., Gouveia, N., Ha, E.-h., et al. Maternal exposure to particulate air pollution and term birth weight: a multi-country evaluation of effect and heterogeneity. *Environmental Health Perspectives* (2013).

[19] Dockery, D. W., Pope, C. A., Xu, X., Spengler, J. D., Ware, J. H., Fay, M. E., Ferris Jr, B. G., and Speizer, F. E. An association between air pollution and mortality in six US cities. *New England journal of medicine 329*, 24 (1993), 1753–1759.

[20] Dominici, F., Daniels, M., Zeger, S. L., and Samet, J. M. Air pollution and mortality: estimating regional and national dose-response relationships. *Journal of the American Statistical Association 97*, 457 (2002), 100–111.

[21] Forastiere, F., Stafoggia, M., Tasco, C., Picciotto, S., Agabiti, N., Cesaroni, G., and Perucci, C. A. Socioeconomic status, particulate air pollution, and daily mortality: differential exposure or differential susceptibility. *American Journal of Industrial Medicine 50*, 3 (2007), 208–216.

[22] Gilliland, F. D., Li, Y.-F., and Peters, J. M. Effects of maternal smoking during pregnancy and environmental tobacco smoke on asthma and wheezing in children. *American Journal of Respiratory and Critical Care Medicine 163*, 2 (2001), 429–436.

[23] Giovanis, E., Ozdamar, O., et al. The effects of air pollution on health status in Great Britain. *Working Paper* (2014).

[24] Glinianaia, S. V., Rankin, J., Bell, R., Pless-Mulloli, T., and Howel, D. Particulate air pollution and fetal health: a systematic review of the epidemiologic evidence. *Epidemiology 15*, 1 (2004), 36–45.

[25] Graff Zivin, J., and Neidell, M. The impact of pollution on worker productivity. *The American Economic Review 102*, 7 (2012), 3652–3673.

[26] Graff Zivin, J., and Neidell, M. Environment, health, and human capital. *Journal of Economic Literature 51*, 3 (2013), 689–730.

[27] Greven, S., Dominici, F., and Zeger, S. An approach to the estimation of chronic air pollution effects using spatio-temporal information. *Journal of the American Statistical Association 106*, 494 (2011), 396–406.

[28] Ha, E.-H., Hong, Y.-C., Lee, B.-E., Woo, B.-H., Schwartz, J., and Christiani, D. C. Is air pollution a risk factor for low birth weight in Seoul? *Epidemiology 12*, 6 (2001), 643–648.

[29] Hanna, R., and Oliva, P. The effect of pollution on labor supply: Evidence from a natural experiment in Mexico City. *Journal of Public Economics 122* (2015), 68–79.

[30] Hausman, J. A., Ostro, B. D., and Wise, D. A. Air pollution and lost work. *NBER Working Paper* (1984).

[31] Herrnstadt, E., and Muehlegger, E. Air pollution and criminal activity: Evidence from Chicago microdata. *NBER Working Paper* (2015).

[32] Heutel, G., and Ruhm, C. J. Air pollution and procyclical mortality. *NBER Working Paper* (2013).

[33] Isen, A., Rossin-Slater, M., and Walker, W. R. Every breath you take-every dollar you'll make: The long-term consequences of the Clean Air Act of 1970. *NBER Working Paper* (2014).

[34] Janke, K., Propper, C., and Henderson, J. Do current levels of air pollution kill? the impact of air pollution on population mortality in England. *Health Economics 18*, 9 (2009), 1031–1055.

[35] Jerrett, M., Burnett, R. T., Beckerman, B. S., Turner, M. C., Krewski, D., Thurston, G., Martin, R. V., van Donkelaar, A., Hughes, E., Shi, Y., et al. Spatial analysis of air pollution and mortality in California. *American Journal of Respiratory and Critical Care Medicine 188*, 5 (2013), 593–599.

[36] Lavy, V., Ebenstein, A., and Roth, S. The impact of short term exposure to ambient air pollution on cognitive performance and human capital formation. *NBER Working Paper* (2014).

[37] Lee, P.-C., Roberts, J. M., Catov, J. M., Talbott, E. O., and Ritz, B. First trimester exposure to ambient air pollution, pregnancy complications and adverse birth outcomes in Allegheny County, PA. *Maternal and Child Health Journal 17*, 3 (2013), 545–555.

[38] Lleras-Muney, A. The needs of the army using compulsory relocation in the military to estimate the effect of air pollutants on children's health. *Journal of Human Resources 45*, 3 (2010), 549–590.

[39] Maier, K. L., Alessandrini, F., Beck-Speier, I., Josef Hofer, T. P., Diabate, S., Bitterle, E., Stoger, T., Jakob, T., Behrendt, H., Horsch, M., et al. Health effects of ambient particulate matter - biological mechanisms and inflammatory responses to in vitro and in vivo particle exposures. *Inhalation toxicology 20*, 3 (2008), 319–337.

[40] Maisonet, M., Correa, A., Misra, D., and Jaakkola, J. J. A review of the literature on the effects of ambient air pollution on fetal growth. *Environmental Research 95*, 1 (2004), 106–115.

[41] Moretti, E., and Neidell, M. Pollution, health, and avoidance behavior evidence from the ports of Los Angeles. *Journal of Human Resources 46*, 1 (2011), 154–175.

[42] Neidell, M. J. Air pollution, health, and socio-economic status: the effect of outdoor air quality on childhood asthma. *Journal of Health Economics 23*, 6 (2004), 1209–1236.

[43] Oberdorster, G., Oberdorster, E., and Oberdorster, J. Nanotoxicology: an emerging discipline evolving from studies of ultrafine particles. *Environmental Health Perspectives* (2005), 823–839.

[44] Ostro, B. D. The effects of air pollution on work loss and morbidity. *Journal of Environmental Economics and Management 10*, 4 (1983), 371–382.

[45] Perera, F. P., Jedrychowski, W., Rauh, V., and Whyatt, R. M. Molecular epidemiologic research on the effects of environmental pollutants on the fetus. *Environmental Health Perspectives 107*, Suppl 3 (1999), 451.

[46] Pope, C. A., Dockery, D. W., and Schwartz, J. Review of epidemiological evidence of health effects of particulate air pollution. *Inhalation toxicology 7*, 1 (1995), 1–18.

[47] Pope III, C. A., Burnett, R. T., Thun, M. J., Calle, E. E., Krewski, D., Ito, K., and Thurston, G. D. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *The Journal of the American Medical Association 287*, 9 (2002), 1132–1141.

[48] Rich, D. Q., Liu, K., Zhang, J., Thurston, S. W., Stevens, T. P., Pan, Y., Kane, C., Weinberger, B., Ohman-Strickland, P., Woodruff, T. J., et al. Differences in birth weight associated with the 2008 Beijing Olympics air pollution reduction: Results from a natural experiment. *Environmental Health Perspectives 123*, 9 (2015), 880.

[49] Ritz, B., Wilhelm, M., Hoggatt, K. J., and Ghosh, J. K. C. Ambient air pollution and preterm birth in the environment and pregnancy outcomes study at the University of California, Los Angeles. *American Journal of Epidemiology 166*, 9 (2007), 1045–1052.

[50] Roberts, A. L., Lyall, K., Hart, J. E., Laden, F., Just, A. C., Bobb, J. F., Koenen, K. C., Ascherio, A., and Weisskopf, M. G. Perinatal air pollutant exposures and autism spectrum disorder in the children of nurses' health study II participants. *Children's Health* (2013).

[51] Romieu, I., Meneses, F., Ruiz, S., Sienra, J. J., Huerta, J., White, M. C., and Etzel, R. A. Effects of air pollution on the respiratory health of asthmatic children living in Mexico City. *American Journal of Respiratory and Critical Care Medicine 154*, 2 (1996), 300–307.

[52] Ruckerl, R., Schneider, A., Breitner, S., Cyrys, J., and Peters, A. Health effects of particulate air pollution: a review of epidemiological evidence. *Inhalation toxicology 23*, 10 (2011), 555–592.

[53] Sanders, N. J. What doesn't kill you makes you weaker prenatal pollution exposure and educational outcomes. *Journal of Human Resources 47*, 3 (2012), 826–850.

[54] Schlenker, W., and Walker, W. R. Airports, air pollution, and contemporaneous health. *The Review of Economic Studies* (2015), rdv043.

[55] Schwartz, J., and Zanobetti, A. Using meta-smoothing to estimate dose-response trends across multiple studies, with application to air pollution and daily death. *Epidemiology 11*, 6 (2000), 666–672.

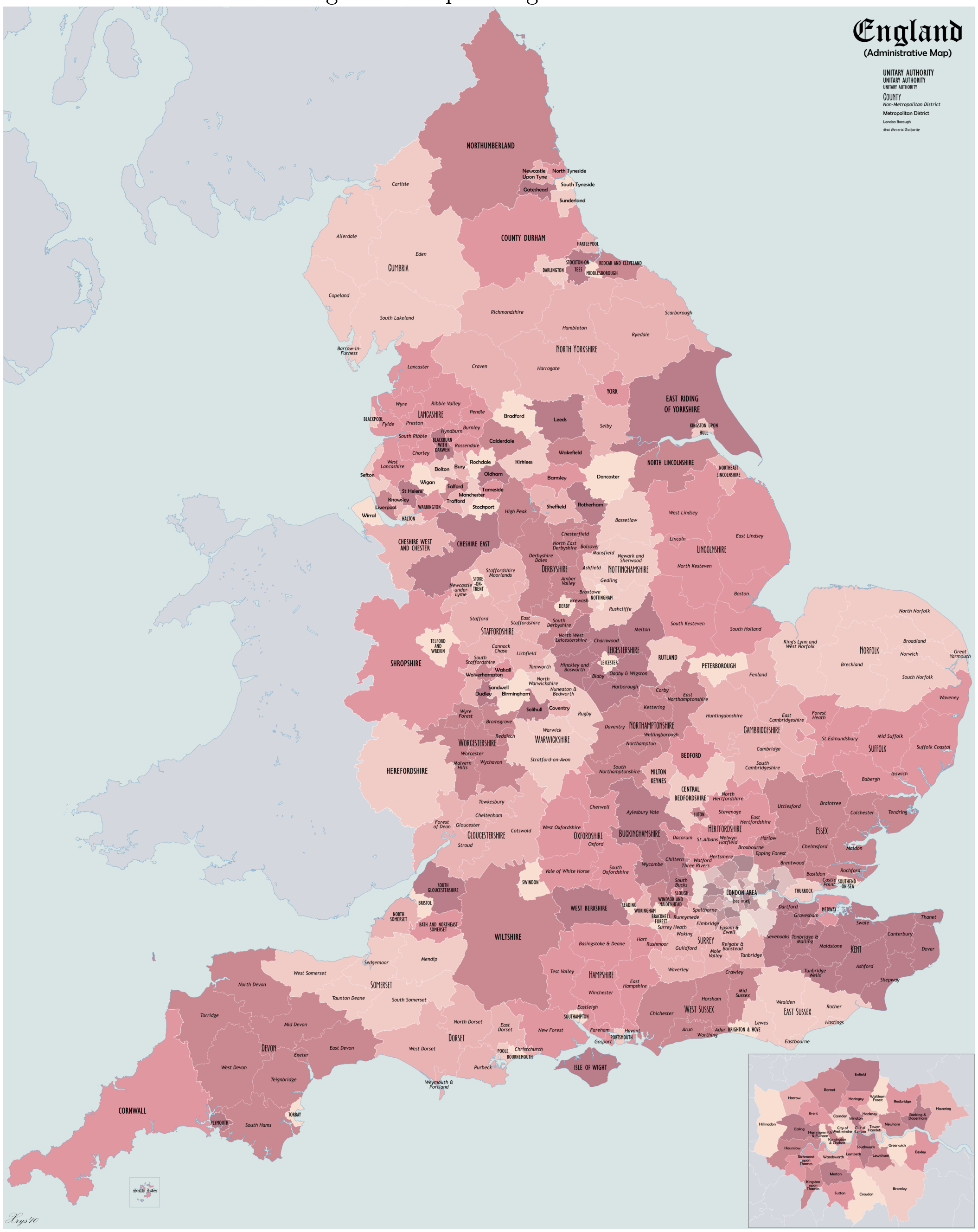
[56] Sram, R. J., Binková, B., Dejmek, J., and Bobak, M. Ambient air pollution and pregnancy outcomes: a review of the literature. *Environmental Health Perspectives* (2005), 375–382.

[57] Tabachnick, B. G., Fidell, L. S., and Osterlind, S. J. *Using Multivariate Statistics*. Allyn and Bacon Boston, 2001.

[58] Zhang, M., Song, Y., and Cai, X. A health-based assessment of particulate air pollution in urban areas of Beijing in 2000–2004. *Science of the Total Environment 376*, 1 (2007), 100–108.

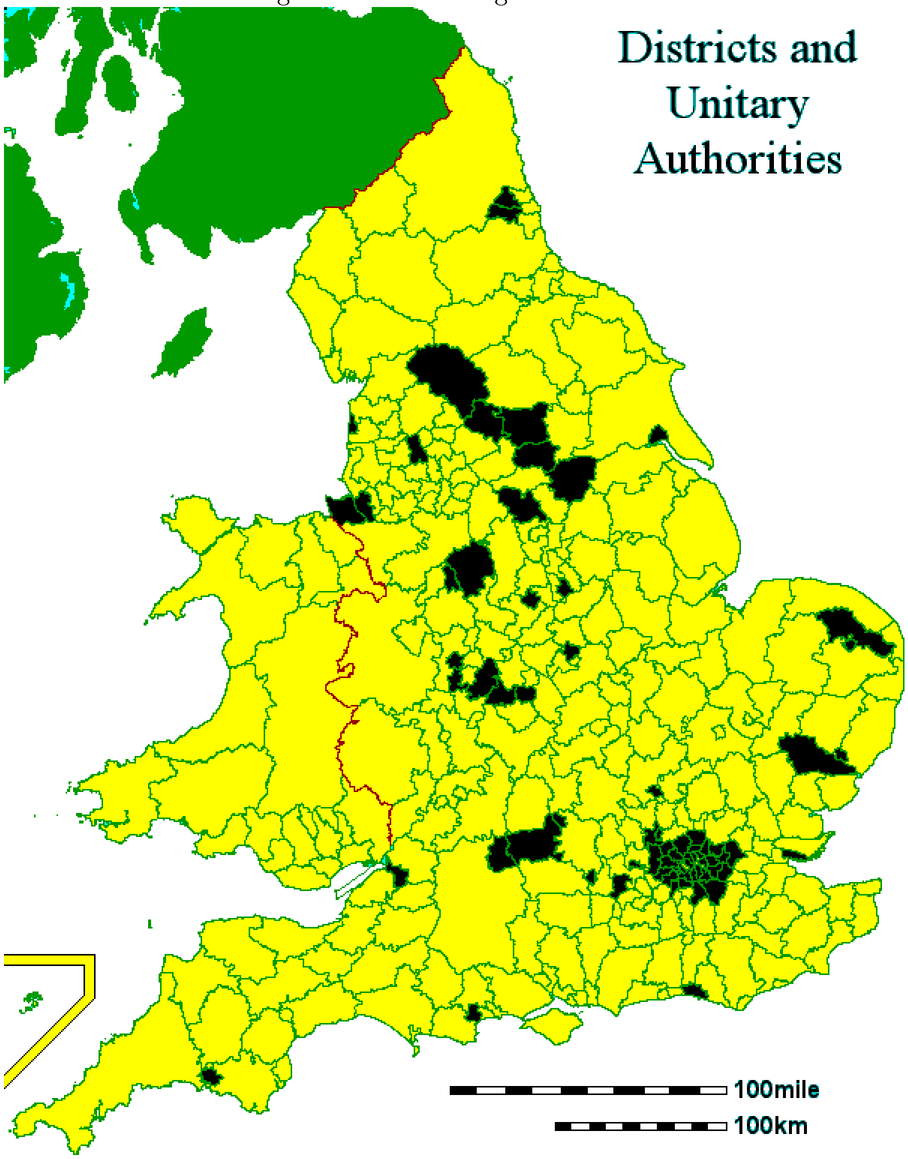
Figures and Tables

Figure 1: Map of English Districts



The above map was created by Wikipedia user XrysD and can be found at https://commons.wikimedia.org/wiki/File:England_Administrative_2010.png.

Figure 2: Chosen English Districts



Black districts are those with matching air pollution data. Map used by permission of the author. © Keith Edkins 2011

Figure 3: Great Smog of 1952

Photo by NT Stobbs

Figure 4: Black Smoke Histograms

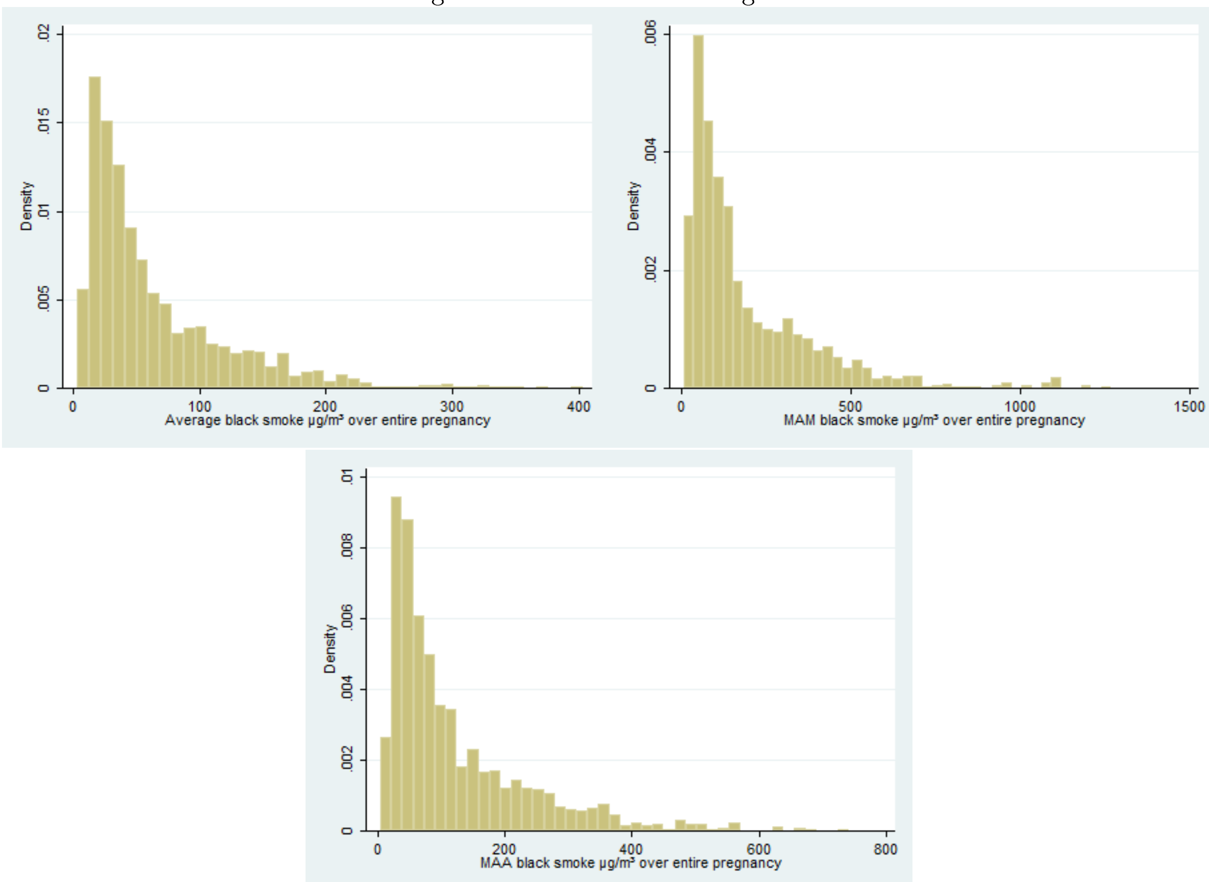


Table 1: District Characteristics

District	Observations	Percent of Obs.	km^2	London	Population	People/km^2
Wesminster	215	0.90	21.49	1	233292	10856
Camden	117	0.49	21.79	1	234846	10778
Hackney	141	0.59	19.05	1	263150	13814
Hammersmith & Fulham	94	0.39	16.4	1	178465	10882
Haringey	46	0.19	29.6	1	267541	9039
Islington	179	0.75	14.86	1	221030	14874
Lambeth	272	1.14	26.81	1	318216	11869
Lewisham	326	1.36	35.15	1	291933	8305
Newham	136	0.57	36.2	1	324322	8959
Southwark	76	0.33	28.86	1	302538	10483
Tower Hamlets	130	0.54	19.78	1	284015	14359
Wandsworth	1798	0.74	34.26	1	312145	9111
Barking and Dagenham	84	0.35	36.11	1	198284	5491
Barnet	2297	0.95	86.75	1	374915	4322
Bexley	127	0.53	60.58	1	239865	3959
Brent	122	0.51	43.23	1	320762	7420
Bromley	427	1.78	150.13	1	321278	2140
Croydon	229	0.96	86.5	1	376040	4347
Ealing	142	0.59	55.54	1	342118	6160
Enfield	343	1.43	80.83	1	324574	4016
Greenwich	237	0.99	47.33	1	268678	5677
Harrow	80	0.33	50.46	1	246011	4875
Havering	277	1.16	112.35	1	245974	2189
Hillingdon	108	0.45	115.7	1	292690	2530
Hounslow	142	0.59	55.98	1	265568	4744
Kingston Upon Thames	201	0.84	37.26	1	169958	4561
Merton	297	1.24	37.62	1	203515	5410
Redbridge	119	0.50	56.42	1	293055	5194
Richmond Upon Thames	51	0.21	57.41	1	193585	3372
Sutton	163	0.68	43.85	1	198134	4518
Waltham Forest	125	0.52	38.81	1	268020	6906

Table 2: District Characteristics Continued

District	Observations	Percent of Obs.	km^2	London	Population	People/km^2
Manchester	1094	4.57	115.65	0	520215	4498
Liverpool	881	3.68	111.84	0	473073	4230
Wirral	637	2.66	157.05	0	320914	2043
Doncaster	623	2.60	567.99	0	304185	536
Sheffield	1087	4.54	367.95	0	563749	1532
Gateshead	331	1.38	142.36	0	200505	1408
Newcastle upon Tyne	780	3.25	113.45	0	126052	1111
Birmingham	1334	5.57	267.79	0	1101360	4113
Coventry	339	1.41	98.65	0	337428	3420
Dudley	397	1.66	97.97	0	315799	3223
Sandwell	259	1.08	85.56	0	316719	3702
Wolverhampton	338	1.41	69.43	0	252987	3644
Bradford	644	2.69	366.42	0	528155	1441
Leeds	538	2.25	551.72	0	766399	1389
Wakefield	820	3.42	338.61	0	331379	979
Bristol	590	2.46	109.61	0	442474	4037
Luton	310	1.29	43.35	0	210962	4866
Bracknell Forest; Slough	137	0.57	141.92	0	262575	1850
Reading	402	1.68	40.4	0	160825	3981
Derby	510	2.13	78.03	0	252463	3235
Plymouth	532	2.22	79.78	0	261546	3278
Bournemouth	99	0.41	46.18	0	191390	4144
Brighton	82	0.34	82.67	0	281076	3400
Southend-on-Sea	62	0.26	41.76	0	177931	4261
Portsmouth	572	2.39	40.36	0	209085	5181
Southampton	418	1.74	49.84	0	245290	4922
Three Rivers; Watford	72	0.30	110.24	0	185928	1687
Kingston upon Hull	213	0.89	71.45	0	257710	3607
Blackburn	392	1.64	137.02	0	146743	1071
Blackpool	356	1.49	34.85	0	140501	4032
Leicester	211	0.88	73.34	0	337653	4604
Broadland; Norwich	580	2.42	591.02	0	263433	446
Nottingham	946	3.95	74.61	0	314268	4212
Oxford; Vale White Horse; W. Oxford	351	1.46	1337.64	0	282849	211
Stoke-on-Trent	659	2.75	93.45	0	251027	2686
Babergh; Ipswich	222	0.93	633.19	0	223811	353
Thamesdown	735	3.07	230.09	0	215799	938

Table 3: District Summary Statistics

Variable	Obs	Mean	Std. Dev.	Min	Max	P50
Observations	68	352.41	285.78	46	1334	265.5
Percent of obs.	68	1.47	1.19	.19	5.57	1.11
Area	68	134.56	204	14.86	1337.64	72.39
London	68	.46	.5	0	1	0
Population	68	296306	144402	126052	1101360	266555
Pop. Density	68	4785.75	3454.32	211	14874	4128.5

Table 4: Pollution Summary Statistics

Variable	Obs	Mean	Std. Dev.	Min	Max	P10	P50	P90	P95
Avg. BS Preg.	23964	64.5	56.7	3.7	403.4	15.6	43.4	145.4	181.9
MAM BS Preg.	23964	195.6	196.2	5.4	1267.6	36	124.2	450.9	566.1
MAA BS Preg.	23964	123.2	113	5.4	741.5	27.9	79.1	279.5	357.3
Avg. BS 1st Tri.	23026	64.1	68.2	2.1	584.3	13.5	39.2	153	211
Avg. BS 2nd Tri.	23244	64.1	67.7	2.8	584.3	13.1	39.5	157	208.6
Avg. BS 3rd Tri.	23496	59.9	60.5	1.9	584.3	12.9	38.4	138.7	185.4
MAA BS 1st Tri.	23026	84.4	91.6	2.9	741.5	16.3	51	194.2	276.8
MAA BS 2nd Tri.	23244	84.1	90.7	3.7	729.6	16.3	51.7	207	271.4
MAA BS 3rd Tri.	23496	79	81.5	3.4	660.2	15.9	50	181.4	252.3

Table 5: Outcome Variable Summary Statistics

Variable	Obs	Mean	Std. Dev.	Min	Max	P10	P50	P90	P95
Wage	14512	2825.29	7240.75	1	134000	94	898.5	3700	17500
Disabled	14511	.01	.08	0	1	0	0	0	0
Poorhealth	13544	.05	.22	0	1	0	0	0	0
Doctor	14465	.32	.47	0	1	0	0	1	1
Higher Ed.	14475	.27	.44	0	1	0	0	1	1

Table 6: Disability

VARIABLES	(1) Disabled	(2) Disabled	(3) Disabled	(4) Disabled	(5) Disabled
Panel A: Full Sample					
Avg. BS Preg.	0.0144*				
	(0.00806)				
MAM BS Preg.		0.0122*			
		(0.00701)			
MAA BS Preg.			0.0171**		
			(0.00776)		
Avg. BS 1st Tri.				-0.00394	
				(0.00632)	
Avg. BS 2nd Tri.				0.0160**	
				(0.00717)	
Avg. BS 3rd Tri.				0.00346	
				(0.00814)	
MAA BS 1st Tri.					0.00550
					(0.00661)
MAA BS 2nd Tri.					0.00814
					(0.00733)
MAA BS 3rd Tri.					0.0124
					(0.00817)
Observations	11,068	11,068	11,068	10,233	10,233
Panel B: Smaller Districts Only					
Avg. BS Preg.	0.0167*				
	(0.00960)				
MAM BS Preg.		0.0209**			
		(0.00906)			
MAA BS Preg.			0.0241**		
			(0.00978)		
Avg. BS 1st Tri.				0.000553	
				(0.00849)	
Avg. BS 2nd Tri.				0.0172*	
				(0.00937)	
Avg. BS 3rd Tri.				0.00731	
				(0.00981)	
MAA BS 1st Tri.					0.0150
					(0.00916)
MAA BS 2nd Tri.					0.00422
					(0.0101)
MAA BS 3rd Tri.					0.0182*
					(0.0105)
Observations	6,908	6,908	6,908	6,368	6,368

*** p<\$0.01, ** p<\$0.05, * p<\$0.1. The dependent variable is whether the respondent was either disabled or long-term sick at the time the survey was given. Standard errors are clustered at the respondent level. Controls for each column are district of birth, district of current residence, interview month-year, month of birth, year of birth, and race of respondent.

Table 7: Disability - London and Rest of England

VARIABLES	(1) Disabled	(2) Disabled	(3) Disabled	(4) Disabled	(5) Disabled
Panel A: London Only					
Avg. BS Preg.	0.117***				
	(0.0451)				
MAM BS Preg.		0.107***			
		(0.0380)			
MAA BS Preg.			0.106***		
			(0.0383)		
Avg. BS 1st Tri.				-0.251***	
				(0.0692)	
Avg. BS 2nd Tri.				0.336***	
				(0.0666)	
Avg. BS 3rd Tri.				0.139***	
				(0.0433)	
MAA BS 1st Tri.					-0.146
					(0.106)
MAA BS 2nd Tri.					0.229***
					(0.0829)
MAA BS 3rd Tri.					0.184***
					(0.0561)
Observations	1,055	1,055	1,055	772	772
Panel B: Rest of England					
Avg. BS Preg.	0.0185**				
	(0.00921)				
MAM BS Preg.		0.0133*			
		(0.00771)			
MAA BS Preg.			0.0194**		
			(0.00877)		
Avg. BS 1st Tri.				-0.00556	
				(0.00679)	
Avg. BS 2nd Tri.				0.0183**	
				(0.00777)	
Avg. BS 3rd Tri.				0.00649	
				(0.00856)	
MAA BS 1st Tri.					0.00345
					(0.00681)
MAA BS 2nd Tri.					0.0106
					(0.00766)
MAA BS 3rd Tri.					0.0137
					(0.00861)
Observations	8,867	8,867	8,867	8,542	8,542

*** p<\$0.01, ** p<\$0.05, * p<\$0.1. The dependent variable is whether the respondent was either disabled or long-term sick at the time the survey was given. Standard errors are clustered at the respondent level. Controls for each column are district of birth, district of current residence, interview month-year, month of birth, year of birth, and race of respondent. Unlike the previous table year of birth is now continuous instead of a vector of indicator variables to prevent too many observations from being disregarded.

Table 8: Unemployment

VARIABLES	(1) Unemployed	(2) Unemployed	(3) Unemployed	(4) Unemployed	(5) Unemployed
Panel A: Full Sample					
Avg. BS Preg.	0.00798				
	(0.00631)				
MAM BS Preg.		0.00356			
		(0.00527)			
MAA BS Preg.			0.00517		
			(0.00624)		
Avg. BS 1st Tri.				-0.00506	
				(0.00517)	
Avg. BS 2nd Tri.				0.00943*	
				(0.00562)	
Avg. BS 3rd Tri.				-0.00113	
				(0.00610)	
MAA BS 1st Tri.					-0.00251
					(0.00496)
MAA BS 2nd Tri.					0.00703
					(0.00588)
MAA BS 3rd Tri.					0.00207
					(0.00617)
Observations	17,677	17,677	17,677	16,744	16,744
Panel A: Small Districts Only					
Avg. BS Preg.	0.0140*				
	(0.00733)				
MAM BS Preg.		0.0103			
		(0.00716)			
MAA BS Preg.			0.0107		
			(0.00776)		
Avg. BS 1st Tri.				0.00187	
				(0.00587)	
Avg. BS 2nd Tri.				0.00947	
				(0.00631)	
Avg. BS 3rd Tri.				8.78e-05	
				(0.00831)	
MAA BS 1st Tri.					0.00517
					(0.00600)
MAA BS 2nd Tri.					0.00649
					(0.00653)
MAA BS 3rd Tri.					0.00337
					(0.00889)
Observations	12,298	12,298	12,298	11,566	11,566

*** p<$0.01, ** p<$0.05, * p<$0.1. The dependent variable is whether the respondent was unemployed at the time the survey was given. Standard errors are clustered at the respondent level. Controls for each column are district of birth, district of current residence, interview month-year, month of birth, year of birth, and race of respondent.

Table 9: Wages

VARIABLES	(1) Wages	(2) Wages	(3) Wages	(4) Wages	(5) Wages
Panel A: Full Sample					
Avg. BS Preg.	-160.1*				
	(91.85)				
MAM BS Preg.		-151.9*			
		(80.54)			
MAA BS Preg.			-223.4**		
			(91.74)		
Avg. BS 1st Tri.				-158.5*	
				(86.13)	
Avg. BS 2nd Tri.				41.44	
				(102.0)	
Avg. BS 3rd Tri.				-4.443	
				(99.29)	
MAA BS 1st Tri.					-128.1
					(90.83)
MAA BS 2nd Tri.					-1.176
					(107.4)
MAA BS 3rd Tri.					16.63
					(97.76)
Observations	10,463	10,463	10,463	9,930	9,930
Panel B: Small Districts Only					
Avg. BS Preg.	6.765				
	(119.1)				
MAM BS Preg.		-16.91			
		(116.1)			
MAA BS Preg.			-93.14		
			(112.9)		
Avg. BS 1st Tri.				-156.2	
				(116.1)	
Avg. BS 2nd Tri.				145.1	
				(130.2)	
Avg. BS 3rd Tri.				88.00	
				(122.7)	
MAA BS 1st Tri.					-125.1
					(121.2)
MAA BS 2nd Tri.					103.6
					(138.8)
MAA BS 3rd Tri.					112.5
					(119.2)
Observations	7,527	7,527	7,527	7,096	7,096

*** p<\$0.01, ** p<\$0.05, * p<\$0.1. The dependent variable is whether the respondent was unemployed at the time the survey was given. Standard errors are clustered at the respondent level. Controls for each column are district of birth, district of current residence, interview month-year, month of birth, year of birth, race of respondent, job type, and length of pay period.

Table 10: Poor Health

VARIABLES	(1) Poor Health	(2) Poor Health	(3) Poor Health	(4) Poor Health	(5) Poor Health
Panel A: Full Sample					
Avg. BS Preg.	0.00516				
	(0.00735)				
MAM BS Preg.		0.00638			
		(0.00626)			
MAA BS Preg.			0.00465		
			(0.00701)		
Avg. BS 1st Tri.				-0.0139**	
				(0.00541)	
Avg. BS 2nd Tri.				0.0213***	
				(0.00700)	
Avg. BS 3rd Tri.				-0.00446	
				(0.00622)	
MAA BS 1st Tri.					-0.00705
					(0.00593)
MAA BS 2nd Tri.					0.0147**
					(0.00691)
MAA BS 3rd Tri.					0.00199
					(0.00602)
Observations	17,178	17,178	17,178	16,233	16,233
Panel A: Small Districts Only					
Avg. BS Preg.	0.00127				
	(0.00977)				
MAM BS Preg.		0.00393			
		(0.00933)			
MAA BS Preg.			-0.00181		
			(0.00954)		
Avg. BS 1st Tri.				-0.0201***	
				(0.00687)	
Avg. BS 2nd Tri.				0.0264***	
				(0.00897)	
Avg. BS 3rd Tri.				-0.0178**	
				(0.00817)	
MAA BS 1st Tri.					-0.00866
					(0.00844)
MAA BS 2nd Tri.					0.0164*
					(0.00923)
MAA BS 3rd Tri.					-0.0101
					(0.00822)
Observations	11,930	11,930	11,930	11,128	11,128

*** p<\$0.01, ** p<\$0.05, * p<\$0.1. The dependent variable is whether the respondent said their health was poor or very poor over the last 12 months. Standard errors are clustered at the respondent level. Controls for each column are district of birth, district of current residence, interview month-year, month of birth, year of birth, and race of respondent.

Table 11: Doctor Visits

VARIABLES	(1) Doctor Visits	(2) Doctor Visits	(3) Doctor Visits	(4) Doctor Visits	(5) Doctor Visits
Panel A: Full Sample					
Avg. BS Preg.	0.0144				
	(0.0136)				
MAM BS Preg.		0.0127			
		(0.0118)			
MAA BS Preg.			0.0115		
			(0.0136)		
Avg. BS 1st Tri.				-0.0156	
				(0.0126)	
Avg. BS 2nd Tri.				0.0367***	
				(0.0137)	
Avg. BS 3rd Tri.				-0.0199*	
				(0.0120)	
MAA BS 1st Tri.					-0.00839
					(0.0123)
MAA BS 2nd Tri.					0.0326**
					(0.0133)
MAA BS 3rd Tri.					-0.0136
					(0.0124)
Observations	20,035	20,035	20,035	19,014	19,014
Panel A: Full Sample					
Avg. BS Preg.	0.0117				
	(0.0175)				
MAM BS Preg.		0.00896			
		(0.0171)			
MAA BS Preg.			0.00282		
			(0.0175)		
Avg. BS 1st Tri.				-0.0258	
				(0.0157)	
Avg. BS 2nd Tri.				0.0440***	
				(0.0166)	
Avg. BS 3rd Tri.				-0.0246*	
				(0.0149)	
MAA BS 1st Tri.					-0.0170
					(0.0155)
MAA BS 2nd Tri.					0.0377**
					(0.0162)
MAA BS 3rd Tri.					-0.0235
					(0.0159)
Observations	14,265	14,265	14,265	13,437	13,437

*** p<\$0.01, ** p<\$0.05, * p<\$0.1. The dependent variable is whether the respondent visited the doctor three or more times in the last year. Standard errors are clustered at the respondent level. Controls for each column are district of birth, district of current residence, interview month-year, month of birth, year of birth, and race of respondent.

Table 12: Higher Education

VARIABLES	(1) Higher Ed.	(2) Higher Ed.	(3) Higher Ed.	(4) Higher Ed.	(5) Higher Ed.
Panel A: Full Sample					
Avg. BS Preg.	-0.0196				
	(0.0172)				
MAM BS Preg.		-0.00912			
		(0.0162)			
MAA BS Preg.			-2.70e-05		
			(0.0176)		
Avg. BS 1st Tri.				0.0127	
				(0.0177)	
Avg. BS 2nd Tri.				-0.0604***	
				(0.0181)	
Avg. BS 3rd Tri.				0.0692***	
				(0.0175)	
MAA BS 1st Tri.					0.0133
					(0.0170)
MAA BS 2nd Tri.					-0.0467***
					(0.0181)
MAA BS 3rd Tri.					0.0625***
					(0.0172)
Observations	19,746	19,746	19,746	18,564	18,564
Panel A: Small Districts Only					
Avg. BS Preg.	-0.0127				
	(0.0212)				
MAM BS Preg.		-0.0123			
		(0.0216)			
MAA BS Preg.			0.000888		
			(0.0222)		
Avg. BS 1st Tri.				0.0291	
				(0.0218)	
Avg. BS 2nd Tri.				-0.0583***	
				(0.0214)	
Avg. BS 3rd Tri.				0.0669***	
				(0.0222)	
MAA BS 1st Tri.					0.0288
					(0.0214)
MAA BS 2nd Tri.					-0.0446**
					(0.0213)
MAA BS 3rd Tri.					0.0568**
					(0.0224)
Observations	13,817	13,817	13,817	12,952	12,952

*** p<$0.01, ** p<$0.05, * p<$0.1. The dependent variable is whether the respondent has a higher education degree. Standard errors are clustered at the respondent level. Controls for each column are district of birth, district of current residence, interview month-year, month of birth, year of birth, and race of respondent.

Appendices

A BHPS Variable Creation

A.1 Unemployed and Disabled/Long-term Sick

The table below shows how respondents answered the following question: "Please look at this card (2) and tell me which best describes your current situation?". A variable for unemployed was generated and if a respondent answered "unemployed" that variable was given a value of one or zero otherwise. A variable for disabled or long term sick was generated and if a respondent answered they were "long term sick/disabled" or said they were registered disabled then that variable was given a value of one or zero otherwise.

Table 13: Unemployed and Disabled/Long-term Sick

Current Labor Force Status	Observations	Percent
missing or wild	6	0.03
proxy respondent	5	0.02
don't know	1	0.00
self employed	1,305	5.45
in paid employ	14,896	62.16
unemployed	1,451	6.05
retired	2	0.01
family care	311	1.30
ft student	2,185	9.12
long term sick/disabled	3,144	13.12
on matern leave	425	1.77
govt trng scheme	105	0.44
something else	128	0.53

Registered Disabled	Observations	Percent
missing or wild	18.00	0.12
inapplicable	1440.00	9.71
-7	40.00	0.27
yes	214.00	1.44
no	13118.00	88.46

A.2 Poor Health

The table below shows how respondents answered the question, "Please think back over the last 12 months about how your health has been. Compared to people of your own age, would you say that your health has on the whole been ...". If a respondent answered "poor" or "very poor" they were assigned a value of one for the variable "poor health".

Table 14: Poor Health

Health over last 12 months	Observations	Percent
missing or wild	3	0.01
don't know	3	0.01
excellent	6,004	26.76
good	10,852	48.36
fair	4,119	18.36
poor	1,218	5.43
very poor	241	1.07

A.3 Doctor Visits

The table below shows how respondents answered the question, "Since September 1st last year, approximately how many times have you talked to, or visited a GP or family doctor about your own health? Please do not include any visits to a hospital." Any respondent who responded with three or more times was assigned to the high visiting doctor group.

Table 15: Doctor Visits

Number of visits to GP since September 1	Observations	Percent
missing or wild	9	0.04
proxy respondent	683	2.85
don't know	13	0.05
none	6,014	25.1
one or two	9,090	37.93
three to five	4,766	19.89
six to ten	1,876	7.83
more than ten	1,513	6.31

A.4 Higher Education

The table below shows the highest educational attainment of all respondents. A indicator variable named "highered" was created and any respondent whose highest education level was "higher degree" or "first degree" was given a one or zero otherwise.

Table 16: Higher Education

Highest academic qualification	Observations	Percent
missing	95	0.4
proxy respondent	699	2.92
higher degree	479	2
1st degree	2,680	11.18
hnd,hnc,teaching	1,266	5.28
a level	5,948	24.82
o level	7,804	32.57
cse	2,706	11.29
none of these	2,287	9.54

B Job Types

The table below shows the job types workers are placed into for the BHPS.

Table 17: Job Types

Socioeconomic group: present job	Observations	Percent
missing	120	0.5
not applicable	6,067	25.32
employers,large	3	0.01
managers,large	1,464	6.11
employers,small	230	0.96
managers,small	1,058	4.41
professional self-employed	106	0.44
professional employees	681	2.84
int. non-manual,workers	2,530	10.56
int. non-man,foreman	771	3.22
junior non-manual	4,425	18.47
personal service wrkrs	1,253	5.23
foreman manual	609	2.54
skilled manual wkrs	1,677	7
semi-skilled manual wkrs	1,488	6.21
unskilled manual wkrs	540	2.25
own account wkrs	737	3.08
farmers - employers,managers	20	0.08
farmers - own account	31	0.13
agricultural workers	126	0.53
members of armed forces	28	0.12