GROWING UP IN A STEEL TOWN: EARLY CHILDHOOD EXPOSURE TO POLLUTION AND LATER-LIFE MORTALITY

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Abstract. A vast literature evaluates health consequences of exposure to pollutants. An important prevailing theory is that exposure to high levels of pollution *in utero* and in childhood can result in a lifetime of health problems, leading to increased mortality rates in later life. I evaluate this claim using a unique proprietary dataset—Medicare records matched to Social Security records which identify birthplace (and thus to likely residence at young ages). My focus is on a sample of over 200,000 individuals born in small towns in Pennsylvania, 1916–1927, who later migrated out of Pennsylvania. Many of these individuals were born in "steel towns"—towns with steel mills and rolling mills, which were scattered throughout Pennsylvania in the early twentieth century—and thus likely had early-life exposure to high levels of air pollution. I find that these individuals have significantly higher rates of mortality post-age 65 than those born in comparable towns that did not have steel production facilities. I find, moreover, that old-age mortality is particularly high for individuals born in steel towns that had multiple mills or in low-elevation steel towns (which were likely in valleys, which typically had higher concentrations of particulate matter).

1. Introduction

Scientists have conducted thousands of studies evaluating the health impacts of air pollution. This concentrated research effort is not surprising; evidence suggests that pollution from a variety of sources (e.g., trucks and automobiles, energy generation, and industrial processes used in steel production) can have important detrimental effects for human morbidity and mortality. Careful studies that document the effects of pollution are crucial for the formulation of appropriate policy responses.

An important theory is that exposure to pollutants is damaging for fetal, infant, and childhood physical development—potentially leading to lifelong health deficits and early mortality. For example, in her well-known book, *When Smoke Ran Like Water,* Davis (2002)

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presents forceful arguments for the proposition that childhood exposure to steel production in towns like Donora, Pennsylvania, had profound negative consequences for the later-life health of individuals who grew up in these towns.[1]

It is a daunting prospect to test this "long reach" hypothesis systematically. Ideally a researcher would like the following design: A very large number of individuals would be assigned, prior to birth, to different locations; "treatment" groups would be exposed to varying levels of potentially dangerous pollutants, and the "control" group would be assigned to comparable locations with low levels of pollution. After childhood these individuals would then be relocated to comparable places with the same level of pollution. Finally, several decades later mortality rates would be compared for the treatment and control groups.

Of course, there exists no experiment that uses the design described in the previous paragraph, and it appears that there is no research that attempts such an analysis using a historical "natural experiment" that parallels such a design. In this paper I undertake the first such large-scale study, focusing on early childhood exposure to pollution from steel mills in small Pennsylvania towns in the early twentieth century.[2]

The key to this study lies in the history of the Pennsylvania steel industry. In the early twentieth century, a large share of U.S. steel production was in Pennsylvania, and much of this production was in steel mills and rolling mills located in many small towns throughout the Commonwealth. (There were also, of course, hundreds of similarly-sized towns that had no steel mills.) By the 1930s, there was a slump in steel production, which would likely have been followed by plant closures, as a consequence of falling demand (DiFrancesco, *et al.*, 2010). In turn many of the mills in small towns, non-central to Pittsburgh, would have closed in favor of more efficient plants, and in turn many people moved away from these mill towns, as I show below. More generally there was a large migration of individuals out of

[1]Donora was the location of the infamous "killing fog of 1948." From October 26 to October 31 an air inversion trapped particulate matter from local metal production facilities in Donora, leading to a dramatic spike in mortality (Davis, 2002).

[2]While there appears to be no studies linking early-life pollution exposure to older-age mortality, there is research in economics the examines early-life conditions to later health outcomes. One example is Almond's (2006) examination of *in utero* exposure to the 1918 influenza pandemic for later-life outcomes. A second example is Maccini and Yang's (2009) study linking rainfall in the year of birth to later-life outcomes for individuals born in Indonesia.

the small towns—both mill and non-mill towns—to locations outside of Pennsylvania and to larger cities in Pennsylvania. During the years leading up to World War II there was a vast increase in U.S. steel production, but most of this production was concentrated in large integrated facilities in such locations as Pittsburgh and other centralized steel communities, like Clairton and Donora.[3]

My focus is on a large sample of individuals who were born in small towns in Pennsylvania in 1916 through 1927, but who in later life lived outside of Pennsylvania. The idea is to see if individuals born in mill towns, who were likely exposed to high levels of pollutants as children, had generally higher levels of mortality in older age than individuals who were born in non-mill towns. The point of the comparison relies on the fact that the sample of interest relocated outside of Pennsylvania to presumably similar locations, and were not systematically exposed to different levels of pollution in later life.

The analysis relies on unique data, the Duke Medicare/SSA Data Set. The starting point of these data is the administrative files of the Medicare Part B program, which include date of birth (used for Medicare eligibility determination) and date of death (used for the purpose of terminating benefits) for people aged 65 and older in the United States. These data do not include place of birth, but they do include Social Security numbers, which were matched to the Numerical Identification Files of the Social Security Administration, which have "town or county of birth" for most individuals. As described below, the data are reasonably complete for the cohorts I study, 1916–1927. The data include only individuals aged 65 and older, as 65 is the age of eligibility for Part B benefits.

The elements of the data set are place of birth, zip code of residence in older age or death (among those who are deceased), gender, and race. I merged these records to additional data. First, I used Pennsylvania historical records to determine the town-level location of steel mills and rolling mills in the early twentieth century. Second, I matched the older-age residence zip code to the median income in the zip code to form a crude proxy of lifetime prosperity for individuals in my sample. I thereby assembled complete records for more than 770,000 white individuals born in Pennsylvania, 1916–1927. Of this sample, more

[3]By 1940, the Pittsburgh area had 42% of U.S. steel production capacity (Warren, 1973).

than 200,000 were born in small towns—mill towns and non-mill towns—*and* subsequently migrated out of Pennsylvania. Below I show that these groups are highly comparable along observable dimensions, so my research focuses almost exclusively on this latter group.

I find that among individuals in my sample, those who were born in steel towns have significantly higher mortality rates than those born in non-steel towns. In particular, I look at mortality at ages 65 to 75. The overall mortality rate for these ages is approximately 20 percent. Individuals born in mill towns have mortality that is 1.3 percentage points higher than comparable individuals born in non-mill towns. Thus mortality is approximately 6.5% higher for those born in mill towns. People born in mill towns with two or more steel production facilities have particularly high mortality rates, 1.8 percentage points higher than in non-mill towns (i.e., 9% higher than in non-mill towns). Finally, I find that among individuals born in steel towns, mortality rates are higher for those born in low-elevation steel towns, i.e., towns that were likely in valleys, which would have typically had higher concentrations of particulate matter.

Of course, a major concern with my empirical work is the potential for omitted variable bias, as carefully discussed in Chay, Dobkin, and Greenstone (2003). An example is the possibility that localities with metal production tended to be more prosperous than other locations, which could have led to increased labor-force participation, higher wages, and possibly better health care and education. These are factors which would typically improve lifetime well-being and in turn, health outcomes. I discuss this issue when interpreting my findings.

The paper proceeds in three additional sections:

In Section 2 I provide a brief critical review of existing work on the health impacts of TSPs, and fine and coarse PM, a particularly dangerous subset of "traditional" TSPs.[4] The primary point that emerges from this review is the absence of papers that evaluate the consequence of childhood pollutant exposure to later-life mortality.

[4]Traditionally TSPs are defined as airborne particles or aerosols that are less than 100 micrometers. In 1987, as a new health standard, the U.S. EPA replaced the TSP with an indicator for only coarse particulate matter, PM_{10}—that is, 10 micrometers or less. Again, in 1997, a stricter standard for particles less than 2.5 micrometers was established, holding fine particulate matter, $PM_{2.5}$, as the dangerous particle of interest (Fierro, 2000).

In Section 3 I provide a historical overview of the steel industry. I discuss the process by which steel was manufactured and the potentially harmful chemicals that were released during production.

In Section 4 I turn to statistical evidence on the association between childhood exposure to steel pollutants and later-life mortality.

Section 5 concludes.

2. Literature

The approaches to studying health impacts of pollution are varied—reflecting differences in expertise and methodology across disciplines. Much of the relevant work falls within the following three overlapping strands of literature:

First, there are many studies in the broad area of public health and environmental sciences that establish associations between exposure to pollutants and morbidity or mortality. A prominent example is the pioneering Harvard Six Cities study (Dockery, *et al.,* 1993), which shows a strong and robust association between city-level air pollution (especially particulate matter or PM) and mortality (especially cancer and cardiopulmonary disease).

Second, there are many studies in medical and epidemiological journals that seek to establish pathophysiological links between pollution and morbidity and mortality. Such work might entail experimental studies involving animals. Alternatively, these studies look at physiological impacts of real-world pollution on the health of surrounding animal populations or vegetation, while others might look at epidemiological evidence regarding pollution and specific hypothesized physiological deficits among humans. An example is important research demonstrating an association between exposure to fine PM and deficits in lung function development among children (Gauderman, *et al.*, 2004).

Third, economists have made a distinctive contribution by focusing on potential threats to the validity of causal inferences drawn from statistical associations between pollution concentrations and health outcomes. Economists often implement novel identification strategies that aim to plausibly establish causality. An example is an important analysis by Chay and Greenstone (2003a). These authors motivate their work by arguing that pollution is not

randomly assigned to individuals, hence the need to find quasi-random variation for estimating the impact of pollution on health. They proceed by using such variation—generated by the 1981-1982 recession—and find that a reduction in location-specific total suspended particulates (TSPs) generates a substantial decline in the infant mortality rate.

Many papers on pollution exposure suggest that pollutant threats are especially important for fetal and infant exposure (e.g., Chay and Greenstone, 2003), and for child-age physical development (e.g., Gauderman, *et al.*, 2004). It seems reasonable to believe, therefore, that exposure to pollution at young ages could lead to lifelong health deficits and early mortality; in general there are well founded concerns that poor early-life conditions—*in utero* and in developmental stages from infancy through late childhood—can have a "long reach," affecting health in important ways in later life.[5]

2.1. **Approaches and Challenges for Studying Health Effects of Particulate Air Pollution.** As noted above, a vast literature is concerned with the health consequences of TSP emissions, such as those released into the environment as the result of industrial processes, traffic, or other sources. Pope and Dockery (2006) provide an extensive and enlightening review of the extant literature on the health consequences of air pollution. In the Pope-Dockery taxonomy, most studies fall into three categories:

First, there are many studies that focus on short-term pollution exposure and mortality. These studies include analyses of severe pollution episodes such as the famous October 1948 "killing fog" in Donora, Pennsylvania (see Davis, 2002, and references therein). While these events are dramatic, systematic analysis is difficult. It is especially difficult to evaluate the extent to which excess mortality is the consequence of harvesting, whereby most mortality is among individuals who would have died soon in any case. For my purposes—the study of childhood pollutant exposure to later-life health—such events are unlikely to be useful unless they are truly dramatic.[6]

[5]This idea is linked closely to the work of Barker (1995), and plays an important role in work by Fogel (2004). A recent relevant study on the "Barker hypothesis" is Almond (2006). Among the other papers that study the childhood origins of health are Case, *et al.* (2002), Case, *et al.* (2005), and Hayward and Gorman (2004).

[6]Thus, it will surely be worthwhile to conduct long-term health studies for individuals who were exposed to radioactive iodine in the aftermath of the 1986 Chernobyl nuclear reactor explosion. Similarly, perhaps

Second, there are many prominent studies that focus on long-term particulate exposure. These studies include the famous Harvard Six Cities study (Dockery, *et al.,* 1993) and the American Cancer Society (ACS) prospective cohort studies (Pope, *et al.,* 1995). There have been many follow-up papers and re-analyses of the data collected for these studies. This research stream is particularly notable because the follow-up time is impressive (over 16 years in some cases). Still, it is worth noting that the time frame is no where near long enough to investigate any association between early-childhood pollutant exposure and older-age health.

Third, there are studies for which the "time scale" of exposure varies. In some instances such variation might be reasonably thought of as quasi-random. An example is a 13-month shutdown of a steel mill in Utah Valley, which occurred because of a labor dispute (Pope, *et al.,* 1992). Again, though, none of the studies reviewed by Pope and Dockery (2006) evaluate such variation to study long-run effects.

Beyond those three categories, the Pope-Dockery taxonomy highlights studies that document associations between particular physiological responses and pollution. Among the important studies of this type are papers by Gauderman and colleagues that study the association between particulate exposure and lung development among children ages 10–18 (Gauderman, *et al.,* 2004, and Gauderman, *et al.,* 2007). Those papers show that exposure to air pollution is associated with deficits in lung development. No long-term follow-up studies are available, i.e., research about effects of childhood exposure to fine PM on older-age mortality. But in light of the work by Gauderman and co-authors, such research would seem warranted.

Finally, it is worth noting that most of the studies mentioned here do not account for the fact that pollution exposure is not randomly assigned. As mentioned above, this is a major concern, as emphasized by Chay and Greenstone (2003a). Also, as mentioned above, Chay and Greenstone make an important contribution by showing that the impact of particulate matter on infants is to increase mortality, by using quasi-random variation in air quality generated by the 1981–1982 recession. Other papers in the economics literature have

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exposure to volcanic magmatic gases could be studied for the purposes of determining the long-term impacts of exposure to such elements as aluminum and rubidium (Durand, *et al.,* 2004).

provided evidence regarding the contemporaneous impact of pollution on child and infant health, including Chay and Greenstone (2003b) and Currie and Walker (2011).[7]

In sum, then, there appears to be a substantial gap in the literature on health impacts of fine particulate air pollution, such as coarse particulate matter, PM_{10}, and fine particulate matter, $PM_{2.5}$. The literature does document that pollution harms infant and child health, and stunts important dimensions of development, such as lung development during childhood. However, it appears that there is no research that asks if the damage has a "long reach," i.e., affects mortality at older ages. Ideally, a study of these issues would not only look for a statistical association between childhood pollutant exposure and old-age mortality, but would do so in a way that allows for plausible identification of causality.

3. The Steel Industry

To set up the empirical analysis below, it helps to briefly consider the history of iron and steel production, culminating in the early twentieth century integration of mills (both in terms of technology and business), e.g., with the founding of the United States Steel Corporation in 1901 (Warren, 1973). An assessment of the technologies used in these plants leads to a better understanding of the chemicals released into the atmosphere.

3.1. **Technologies and Production Process.**

The process of making steel, at the most basic level, involves inputs of energy and iron ore. By the early 20th century, the basic process was standard: iron ore was transformed into pig iron, and then refined into steel. Some mills existed separately for the purpose of melting and reshaping, such as rolling mills, which produce plates or quality flat and long products (Beer, *et al.,* 1998).

In their study of energy-efficient steel making, Beer, *et al.* (1998) produce a thorough survey of the history of the steel and iron industry. They note that the basic process of producing usable iron is to free slag from the ore to produce wrought iron. By 1300 A.D. Stuckovens were introduced to Germany, which at 3-5 m high, akin in shape and design to the modern blast furnace. The new technology was to use energy to produce iron products. Initially charcoal was the source fuel; coke was introduced to the iron market in 1718, and

[7]This last paper is also a good source for referencing additional efforts along these lines.

by 1790 it made up 90% of pig iron production. By 1900 several additional landmark iron-making technology were introduced: steam engines, hot blast, and closed top furnaces were introduced (Beer, *et al.,* 1998).

As Beer, *et al.,* describe, there are three basic processes of steel production: the refining process, the melting process, and the process of both refining and melting. The refining process removes impurities from pig iron, such as carbon, silicon and manganese.[8] By contrast, melting processes are for the purpose of melting and casting steel into the desired shape. Several processes had emerged along these lines by 1900—perhaps the most famous of which was was the Bessemer process, patented by Bessemer in 1855. In this process, the metal is melted by the heat of the oxidation of the carbon and the other impurities in the refining process. Cold air is blown through a refractory acid-lined vessel in the Bessemer converter. No additional fuel was required other than the initial 1 ton coal per 1 ton steel consumed. The popular Bessemer was used in early twentieth century Pennsylvania plants.[9] In addition, there was widespread use of the open heath furnace, which was being developed in France (Beer, *et al.,* 1998).

Warren (1973) notes that at the end of the 1800s, the steel industry consisted of a large number of relatively small firms that would buy raw materials on the open market. Pig iron was purchased by mills that were only capable of refining from the secondary product. Iron ore and coke, much of which came from Connellsville (Warren, 2001), was procured by pig iron furnaces. In 1903, for efficiency reasons, steel companies began to build by-product coke ovens in the mills for integrated production of pig iron and steel, beginning in the Pennsylvania region in the Pittsburgh area. Warren (1973) suggests that this change in coking was particularly beneficial to Pittsburgh as they could send coking coal down the Monongahela. However, the establishment of linkages between sites in the Commonwealth was slow and it wasn't until 1918 when great headway was made toward integration—when U.S. Steel put up the world's largest by-product coke oven on a site north of Clairton Works,

[8]These industries also include those that free the pig iron from slag to produce wrought iron, instead of allowing the pig iron to move on to steel production.

[9]Note here that the"Bessemer Process" could also include the adapted Thomas process or the basic Bessemer Process, which used only basic refractory lining in the Bessemer converter.

and a supply of coal was barged daily down the Monongahela to Pittsburgh (and also gas to Edgar Thomas, Homestead, Duquesne, and Clairton Works).

An important development was unified large-scale production. In 1901, the United States Steel Corporation, including Carnegie, Federal and National Steel, American Steel and Wire, and National Tube, among others, began business. They immediately made efforts to acquire raw materials and within five years, many acquisitions were made by U.S. Steel Corporation around the Pittsburgh area. Between 1901 and 1927, over 60 plants were shut down, early abandonment giving an indication of the major changes to come.[10]

The small firms came to notice their weak position. Gentlemen's agreements informally guided the small mill market as pools formed, provided penalties for overproduction and sometimes paying firms to withdraw from trade. Warren (1973) observes:

> The very uncertain trade conditions of the last twenty years of the nineteenth century threatened their position, and provided much of the motive behind the backward integration of the period and the proliferation of pools and great consolidations.

Many firms survived until 1931 when primary production for steel and iron sharply declined and continued to stay low until 1939 (DiFrancesco, *et al.*, 2010). Warren (1973) suggests that the decline in demand, along with other industrial changes tended to weed out poorly located or inefficient mills and furnaces. Over time there has been a substantial decline in production at non-central, non-integrated mills located in the small towns of Pennsylvania.

3.2. **Exposure to Pollution from Steel Production.** The dangers from exposure to steel and iron production have been widely studied. Coal burning in iron and steel production leads to the release of CO_2, and the process of iron smelting releases CO (Climate Leaders, 2003). Among other symptoms, exposure to CO_2 can lead to asthma and increased rates of cancer (Jacobson, 2008). Exposure to CO can lead to wide-ranging clinical symptoms from

[10]Some plant closures were in the Pittsburgh area, where an overwhelming number of plants along the confluence of the rivers kept the industry tight.

cardiovascular to respiratory and neurobehavioral effects at even low concentrations; unconsciousness and death can result after prolonged or acute exposure to high concentrations (Fierro, *et al.*, 2001.)

Fierro (2000) notes that additional metallic and gaseous emissions are produced through smelting and steel mills, emitting the most common size of particulate matter, PM_{10} and $PM_{2.5}$. Coarse particulate matter is released in the form of solids as a product of coal burning; fine particulate matter, gaseous particles, are derived from coal combustion and additional chemical reactions from metal processing in these plants, traveling far distances of 100s to 1000s of kilometers and lasting in the atmosphere for days to weeks, embedding themselves deep in the upper respiratory tract of residents and possibly presenting harm to organs. PM_{10} has been associated with increased hospital admissions for COPD, asthma, and lower respiratory tract infections, including bronchitis and pneumonia, in elderly patients (Fierro, 2000). Fierro (2000) also points to the strong association between $PM_{2.5}$ and cardiac disease.

Researchers are obtaining a more thorough understanding of how specific compounds in emissions from these integrated steel mills and other coal-burning facilities, such as separate blast iron furnaces and steel mills, effect health through compiled medical studies. A recent report on the toxicological properties of coal emissions showed that coal-burning power plants, for example, produce hazardous air pollutants which cause irritation and tissue damage to the eyes, skin, and breathing passages at high levels of exposure (Billing, 2011). Billing additionally suggests that exposure to these pollutants can cause latent diseases that can develop over many years and may be a contributing factor to such fatal conditions as heart disease and brain impairments. In a 1989 report prepared by the Radian Corporation for the EPA, that specifically focuses on the health hazards of steel mills, it is noted that substances of concern to public health not only include coke oven emissions from coal-burning, but also heavy metal emissions (e.g. copper, cadmium, and chromium). Chromium, for example, has been shown to cause damage to nasal passages and, in long run studies, has been linked to lung cancer (Radian Corporation, 1989).

One of the most important studies that provides direct evidence about this issue is the Utah Valley "natural experiment" (Pope, 1989). The study shows that during the closure of

the steel mill, children's hospital admissions were substantially lower than when the mill was operating. This was particularly true for bronchitis and asthma. Similar findings pertained for adults, though the relationship was not as strong. Pope (1989) also gives links to other literature on pollution from steel. I do not discuss that literature further here, other than to note, again, the literature does not focus of the long-reaching effects of exposure to this sort of pollution.

4. Childhood Exposure to Pollution from Steel Production and Older-Age Mortality

As discussed above, the question addressed by this paper is: "Do individuals who likely had relatively high levels of early childhood exposure to high levels of coarse and fine PM, due to proximity to steel production, have higher levels of mortality at older ages (post age 65) than individuals who likely had lower levels of exposure?"

4.1. **Data.** The key to providing evidence about this research question is a unique dataset, which matches complete Medicare Part B records with Social Security records via the Numerical Identification Files (NUMIDENT) of the Social Security Administration. These records cover a relatively high fraction of the population for cohorts born in 1916 and after (with match rates typically 0.85 or higher for cohorts post 1915).[11] For most of the sample, the data include information on date and location of birth (e.g., the town and county in which people were born), and then the location by zip code at age 65 or date of death (for those who are deceased). The data also include gender and race. The records only extend through 2002, so one can look at rates of mortality for people aged 65-75 for cohorts born 1916 through 1927.[12]

I merge the Duke SSA/Medicare data to historical records that provide locations of pollution sources. Data on steel production in Pennsylvania comes from reports from the Secretary of Internal Affairs of the Commonwealth of Pennsylvania (1903). Unfortunately, it appears that Pennsylvania did not publish such records in the Annual Report of Industrial Statistics for years after 1903. Fortunately, in the early 20th century there was little expansion of steel

[11]The data were used by Black, *et al.,* 2012. Data construction is described in more detail in that paper.

[12]In addition, one could in principle study the incidence of disability for anyone enrolled prior to age 65, though the data have not yet been used for that purpose.

production in small cities, which is the focus of my study. Having said that, I do attempt to take care with this issue by collecting the location of steel production at the end of my study period using an independent historical source (American Iron and Steel Institute, 1930). One way of defining steel towns is to include only those towns identified as steel-producing towns in both sources. Importantly, when I do so my key results are virtually unchanged.[13] I also match the older-age residence zip code to the median income in the zip code to form a crude proxy of lifetime prosperity for individuals in my sample.

Table 1 provides a set of summary statistics for my sample: white individuals, with complete data records, for the birth cohorts 1916–1927 born in Pennsylvania. Among those who are excluded from the sample are individuals for whom it was not possible to match birth place to a "populated place" as given by the U.S. Geological Survey. Typically this happened if a county and city had the same name. The most important example is Philadelphia. Thus my starting sample excludes those individuals. In total the sample is quite large—over 770,000.

For reasons I discuss shorty, I will be splitting the sample according to the size of birthplaces, so Table 1 summarizes key variables according to the size of the city or town in which individuals were born. First are "Large Cities," corresponding roughly to the top quartile of the sample.[14] These are birthplaces with at least 9616 individuals in the sample. There are only 13 birthplaces for these individuals (larger cities like Pittsburgh, Johnstown, Scranton, etc.). The "Mid-Sized Cities" are birthplaces with 2535–9615 individuals in the sample (roughly the second quartile). "Towns" are birthplaces with 2534 or fewer individuals (i.e., the median and below).

The first row on Table 1 shows that in large cities, a very high fraction of people are born in places with a steel mill. Indeed, in the early twentieth century there were steel mills

[13]As I note below, in some specifications I also use locations of zinc smelters. To identify towns with zinc smelters, I use an open-file report from the U.S. Department of the Interior and the U.S. Geological Survey (2010), in which the authors recently studied historical zinc smelting sites in Appalachian states. There are four plants operating in Pennsylvania in the relevant period: Donora (1915-1957), Langeloth (1914-1947), Florence (1891-1945), and Palmerton (1898-1986). I also use data on the location of pig iron production from the Secretary of Internal Affairs (1903).

[14]I set the cut-off as close as possible to the top quartile; an exact top quartile is not possible given that many people were born in any given birthplace at the top end of the distribution.

in all but two of the 13 cities represented in this group (Wilkes-Barre and Hazelton). The second row shows that survival to age 75, conditional on being alive at 65, is approximately 80 percent and does not vary much across the size of one's birthplace. Finally, the lifetime income proxy—median zip code level income in the older-age residence—is somewhat higher for those born large cities than in smaller cities.

4.2. **Research Design.** The goal is to see if childhood exposure to high-polluting steel production is associated with older-age mortality. This entails conducting multivariate analysis in which I compare individuals born in steel mill towns to individuals born in towns without steel mills.

To highlight the challenges that lie ahead, consider the following regression model of survival:

$$S_i = \beta_0 + \beta_1 M_i + \sum_t \beta_t Z_i^t + \gamma X_i + \epsilon_i \tag{1}$$

where S_i is an outcome "survival" variable equal to 1 if individual i survives to age 75 and 0 if she or he dies (conditional on survival to age 65); M_i is 1 if the individual was born in a mill town and 0 otherwise, and is meant to capture effects of early-childhood exposure to pollutants from steel production; Z_i^t is an indicator variable equal to 1 if the individual belongs to the specified birth cohort $\times$ gender cell (e.g., men born in 1927); and X_i is a vector of all other relevant factors that affect old-age survival.

Unfortunately, while the data include reasonably good measures for S_i, M_i, and Z_i^t, there are virtually no data for X_i. So the only hope for credible analysis is to construct samples for which any X_i variables can reasonably be subsumed in the error term.

As an example of the problems this creates consider the following: Suppose that steel mills bring prosperity to a community, so that mill towns tend to have higher incomes and better schools. There is a very large literature that establishes a positive relationship between higher income levels and higher educational levels and survival.[15] Since I have an individual

[15]Grossman (1972) provides theoretical ideas on the topic. Empirical work shows strong associations between income and mortality, and education and mortality, at the country level, and individual level within countries. See, for example, Preston (2007), Preston and Elo (1995), Elo and Preston (1996), Cutler, *et al.* (2006), and Cutler and Lleras-Muney (2006, 2010).

level income measure that is a rather crude proxy and I have no data on education, this presents a problem. Simply omitting these variables from (1) likely will lead to bias. In this case, then, it would be important to restrict the sample to individuals born in places in which subsequent lifetime prosperity is similar (on the basis of available evidence).

As a second example, recall that I am trying to specifically isolate the effects of *childhood* exposure to pollutants on older-age mortality. So I would like to include in the X_i vector measures of residence throughout the lifetime (e.g., to "control" for cases in which people live throughout their lives in polluted places). Given this, a reasonable way to proceed is to focus on people who migrate outside of Pennsylvania, since virtually all of these people will have moved to similar locations where the level of pollution would not have systematically differed.

There is an additional problem with any analysis for people in large-cities: even in the early twentieth century, large cities like Pittsburgh were spread out over many square miles. Some people born in Pittsburgh lived in steel-producing valley communities along the Allegheny and Monongahela rivers, while others lived in the hills high above the rivers or in suburban communities. Public transportation (trolleys and inclines) allowed steel workers and their families to potentially live long distances from the mills. To a lesser degree, these same problems exist for "mid-size" cities in my sample. These were larger towns, where a substantial fraction of the population might live some distance from the mills.

In sum, then, the most promising possibility for analysis is a sample of individuals who were born in small towns *and* who have migrated out of Pennsylvania as adults. However, for this group to be a credible sample for analysis, it is crucial that these individuals be similar with respect to subsequent lifetime outcomes, at least as measured by the income proxy variable.

With this in mind, consider Table 2. Panel A shows that migration patterns out of large cities was very different for mill towns (most large cities, including Pittsburgh) and the non-mill towns (Wilkes-Barre and Hazelton). Most importantly, among those who migrated out of Pennsylvania, those coming from mill towns were substantially more prosperous than

those who were born in non-mill towns. Panel B shows that the same is true for those born in mid-size cities.

Fortunately, though, Panel C of Table 2 suggests that individuals born in small towns are a promising group for study. Among the more than 390,000 individuals born in small towns, a substantial proportion (over 22,000) were born in mill towns. A very high proportion (approximately half) of people born in small Pennsylvania towns in 1916 through 1927 had moved outside of Pennsylvania by old age. Such migration was probably "selective" in that individuals who migrated were likely people who were more ambitious and forward-looking than other individuals. Thus it would not be surprising if they ended up with higher lifetime incomes than those who remained in small towns. As it turns out, this was true for both mill towns and non-mill towns. Fortunately, the income proxy averages are virtually identical in mill and non-mill towns for both the migrating and non-migrating population.

Also, note for small towns that had steel mills as of 1903, housing was generally built up and clustered near the mills. Many of the mills were near rivers and thus were in valley towns. The exposure to TSPs would potentially have been high for children born in these towns. Finally, as mentioned previously, there is yet another good reason to focus on this group. During the 1930s steel production had likely declined in small towns, as there was consolidation in medium-sized cities (like Donora) and larger cities (like Bethlehem and Pittsburgh). A very high fraction of people born in small mill towns moved out of those towns. As it turns out, a similarly high fraction of people born in small non-mill towns also moved out of those towns. This gives us a potentially valuable comparison group for conducting our analysis. As a maintained hypothesis, virtually all of them would be moving to similar locations, i.e., locations with similar levels of adulthood pollution exposure.

4.3. **Results.** For completeness sake, I begin with a simple regression, run for the entire data set, in which the dependent variable is survival to age 75 conditional on survival to age 65. The key indicator variables is "Mill in Birthplace," which is equal to 1 if the individual was born in a steel town (and 0 otherwise). A set of sex $\times$ birth cohort indicator variables is included.

Results are reported in Table 3. The Age × Cohort Effects (not reported in the Table) are as follows: The omitted category was men in birth cohort 1916. Then for men in other cohorts, estimated effects are generally quite small, with a bit of an upward drift for later cohorts. For women estimated cohort effects are very large, typically on the order of 0.09.[16] As for our coefficient of interest, individuals born in steel towns have survival rates that are virtually the same as other individuals. In the second column of Table 3, I run the regression without the "Mill in Birthplace" variable, but include the income proxy variable. The income proxy variable is highly significant; as expected, people who live in higher-income communities also live longer. Finally the third column includes both variables. As argued above, there is very little to be learned from these regressions applied to the full sample.

I therefore turn, in Table 4, to the primary analysis. Here the sample is restricted to those individuals who were born in small towns, and who migrated out of Pennsylvania in older age. In short, these are individuals who grew up in comparable towns, some of which were mill towns and some of which were not. Because the towns were small, most of the individuals would likely have lived quite close to the mills, and would therefore have been potentially exposed to PM.

Results are striking. Among individuals in this sample, those who were born in steel towns have significantly higher mortality rates than those born in non-steel towns. Recall that the overall mortality rate for ages 65 to 75 is approximately 20 percent (see Table 1). Individuals born in mill towns have mortality that is 1.3 percentage points higher than comparable individuals born in non-mill towns. Thus mortality is approximately 6.5% higher for those born in mill towns.

We have already seen that lifetime income appears to be similar for our sample for those born in mill towns. The expectation, therefore, is that "controlling" for the income will not much affect key inference about being born in a mill town. In fact, looking at the regression in column (2) of Table 4, this turns out to be the case.

To gain insight into this outcome, consider Table 5. In this regression the dependent variable is the income proxy variable. Among individuals who were born in Towns who then

[16]In this population, as in most populations, women survive at higher rates than do men.

migrated out of Pennsylvania, those born in a mill town have virtually the same lifetime income as those born in non-mill towns.[17]

Table 6 provides additional evidence about begin born in a mill town: People born in mill towns with two or more steel production facilities have particularly high mortality rates, 1.8 percentage points higher than in non-mill towns (i.e., 9% higher mortality for those born in mill towns). This is as one would expect, if the pollution from steel production is contributing a "dose response" to the higher mortality rates in older age.

Importantly, for all of the regressions I report, there are no statistically significant differences for men and women in the impact of being born in a mill town. The last two columns of Table 6 illustrate that estimated effects are similar for men and women (though now some coefficients are statistically significant only at the 0.10 level).

The final piece of evidence concerns a potential role for elevation in shaping my findings. Among individuals born in mill towns, it seems likely the exposure to PM would have been worst for those born in low-elevation steel towns, i.e., towns that were likely in valleys, which would have typically had higher concentrations of PM. A simple way of evaluating that idea is to see if there is a correlation between survival probabilities and the elevation of the mill town (which was determined using Geographical Analysis Tools, 2012). In Table 7 I report the results of this analysis. The coefficient is indeed positive and statistically significant. Doubling elevation, e.g., from 750 feet to 1500 feet, increases the survival rate by 0.01. Thus, given the mortality rate of 0.20, the higher-elevation location in this case has an approximately 5 percent lower mortality rate.

4.4. **Additional Analyses.** The evidence reported above suggests that being born in a steel mill town is associated with higher mortality at older ages in a sample of individuals born in small towns in Pennsylvania, 1916–1927, who subsequently moved out of Pennsylvania.

Of course, this finding might not be causal if, for example, individuals born in mill towns differ substantially from those born in non-mill towns along some other dimension that might

[17]Taken at face value, the coefficient indicates that median annual income is $120 higher in old-age residences for people born in mill towns than in non-mill towns—a trivial difference. In any event, the coefficient is not statistically different than 0.

affect older-age mortality, e.g., in lifetime prosperity. However, the best available evidence is that in fact these individuals were quite similar along this dimension in older age.

Another potential problem with the analysis is that there is no way to know the age at which individuals leave their home towns. However, in general migration out from rural areas in the early to mid-twentieth century was primarily for people aged 18–30.[18] To the extent that some individuals might have moved with their families at very young ages (e.g., at age 1), I am under-estimating the impact on growing up in a mill town on older age longevity.

Another potential problem is that some of the towns I classify as "non-mill towns" might themselves have had other industrial production which produced dangerous pollution. That is, my "control" towns might not all have been low-pollution places. I did try some investigations of this possibility. In particular, I examined locations in which there was zinc production and pig iron production (but no steel production).[19] When I exclude individuals born in these towns, this makes very little difference to my regressions, presumably because they represent a very small proportion of my sample. More generally, to the extent that some non-mill towns had high levels of PM, I am underestimating the effects of childhood exposure to PM on older-age mortality.

As I mention above, I conducted an additional analysis in which towns are designated to be mill towns only if they appear in both of two historical sources, Secretary of Internal Affairs (1903) and American Iron and Steel Institute (1930). Results are virtually unchanged when I use that definition. Also, the latter data source provides an indicator of the process used in blast. I investigated to see if mortality rates were associated with process—Bessemer or non-Bessemer (mostly open hearth) methods. There were no statistically significant differences found.

Finally, for interest sake, I did estimate the key regressions from Table 4 for the sample of individuals who did not migrate out of Pennsylvania at older age. For this sample I find

[18]See, e.g., Black, *et al.,* (2012).

[19]Zinc production, in particular, is a high-pollution activity that merits further investigation. See, e.g., Batchelor, *et al.* (1926), Bleiwas and DiFrancesco (2010), Buchauer (1973), Carroll and Loneragan (1968), Chmielewski, *et al.* (1947a and 1947b), and EPA (1987). In future work I intend to examine the impacts of zinc production in more detail.

that those who were born in mills towns have lower longevity at older ages, but the effect is not statistically significant.

5. Conclusion and Directions for Future Research

My reading of the literature suggests that there is a substantial gap concerning empirical evidence about the impact of PM exposure and health. There are many studies that document that exposure to pollutants *in utero,* in infancy, and throughout childhood cause measurable contemporaneous damage to health. And there are good reasons to believe that this in turn might create health deficits later in life. However, it appears that there are no systematic studies that examine the impact of childhood exposure to pollutants on old-age mortality.

This paper provides some initial evidence on that question. Looking at a sample of over 200,000 individuals born in small towns in Pennsylvania, 1916–1927, who later migrated out of Pennsylvania, I find that individuals who were born in steel towns have significantly higher rates of mortality post-age 65 than those born in comparable towns that did not have steel production facilities. I find, moreover, that old-age mortality is particularly high for individuals born in steel towns that had multiple mills or in low-elevation steel towns (which were likely in valleys, which typically had higher concentrations of particulate matter).

There are several important directions for future work, two of which I mention here. First, I hope to study other industrial processes for which childhood exposure may have led to older-age mortality differentials. One example is zinc smelting.[20] Second, it would be extremely valuable to see if there are differences in patterns of "cause of death" for individuals who grew up in mill towns compared to those who did not. For future research, I am hopeful that for a subset of my sample, records can be matched to cause of death or other data on health problems from Medicare records.

[20]I have available records of zinc production in several States for the early twentieth century, and have begun the process of matching to the Duke SSA/Medicare data set.

TABLE 1. Sample Characteristics, Individuals Born in Populated Places in Pennsylvania, 1916–1927

	(1) Large Cities	(2) Mid-Sized Cities	(3) Towns
Percent Born in Place with a Steel Mill	87.03	36.55	5.62
Survival to Age 75 Conditional on Living to 65	80.2	79.7	80.2
Income Proxy (in 1000s)	42.8	41.6	41.7
Sample Size	191,221	193,329	391,851

Note: Author's calculations using the Duke SSA/Medicare Data Set matched to historical records on the location of steel production. "Large Cities" are birthplaces with at least 9616 individuals in the sample (roughly the top quartile), "Mid-Sized Cities" are birthplaces with 2535–9615 individuals in the sample (roughly the second quartile), and "Towns" are birthplaces with 2534 or fewer individuals (i.e., the median and below). The income proxy variable is the median annual income (in 2000) in the zip code where the individual resides post age 65. The sample includes whites only, for those for whom data are complete. The sample excludes Philadelphia (see text).

TABLE 2. Sample Characteristics for Migrants and Non-Migrants

	(1) Mill Towns	(2) Towns Without a Mill
A. Born in Large Cities		
Percent Migrating from PA	45.4	20.4
Income Proxy (in 1000s)		
Migrating from PA	44.9	40.1
Not Migrating from PA	40.2	52.9
Sample Size	166,426	24,795
B. Born in Mid-Sized Cities		
Percent Migrating from PA	50.7	45.8
Income Proxy (in 1000s)		
Migrating from PA	47.2	42.3
Not Migrating from PA	38.2	39.6
Sample Size	70,671	122,658
C. Born in Towns		
Percent Migrating from PA	52.6	47.6
Income Proxy (in 1000s)		
Migrating from PA	44.4	44.3
Not Migrating from PA	39.0	38.8
Sample Size	22,029	369,822

Note: Author's calculations using the Duke SSA/Medicare Data Set matched to historical records on the location of steel production. "Towns" are as defined in Table 1.

TABLE 3. Survival to Age 75, Full Sample of Individuals Born in Pennsylvania, 1916–1927

	(1)	(2)	(3)
Mill in Birthplace	0.0012	–	-0.0004
	(0.0020)		(0.0023)
Income Proxy	–	0.0013**	0.0013**
		(0.00005)	(0.00006)
Age × Cohort Effects Included?	yes	yes	yes
Sample Size	787,269	787,269	787,269

Note: Author's calculations using the Duke SSA/Medicare Data Set matched to historical records on the location of steel and zinc production. Dependent Variable is "Survival to Age 75" conditional on survival at age 65 (and thus being in the data). Standard errors, clustered at the town level, are in parentheses. *significant at 0.05; ** significant at 0.01.

TABLE 4. Survival to Age 75, Individuals Born in Pennsylvania Towns, 1916–1927, who Reside Outside Pennsylvania in Older Age

	(1)	(2)
Born in Mill Town	−0.013**	−0.013**
	(0.004)	(0.003)
Income Proxy	–	0.0013**
		(0.00006)
Age × Cohort Effects Included?	yes	yes
Sample Size	204,200	204,200

Note: Author's calculations using the Duke SSA/Medicare Data Set matched to historical records on the location of steel production. Dependent Variable is "Survival to Age 75" is conditional on survival at age 65 (and thus being in the data). The sample includes only those who migrate out of Pennsylvania. Standard errors, clustered at the town level, are in parentheses. *significant at 0.05; **significant at 0.01.

TABLE 5. Impact of Being Born in a Mill Town on Income, Individuals Born in Pennsylvania Towns who Reside Outside Pennsylvania in Older Age

Born in Mill Town	0.120
	(0.144)
Age × Cohort Effects Included?	yes
Sample Size	204,200

Note: Author's calculations using the Duke SSA/Medicare Data Set matched to historical records on the location of steel production. Dependent Variable is "Survival to Age 75" is conditional on survival at age 65 (and thus being in the data). The sample includes only those who migrate out of Pennsylvania. Standard errors, clustered at the town level, are in parentheses. *significant at 0.05; **significant at 0.01.

TABLE 6. Survival to Age 75, Individuals Born in Pennsylvania Towns who Reside Outside Pennsylvania in Older Age

	(1) Full Sample	(2) Full Sample	(3) Men	(4) Women
One Mill in Town	−0.010*	−0.012**	−0.015*	−0.009
	(0.005)	(0.005)	(0.007)	(0.006)
Two or More Mills in Town	−0.018**	−0.016**	−0.016	−0.017*
	(0.005)	(0.005)	(0.008)	(0.007)
Income Proxy	–	0.0013**	0.0016**	0.0011**
		(0.00006)	(0.00010)	(0.00007)
Age × Cohort Effects Included?	yes	yes	yes	yes
Sample Size	204,000	204,200	91,543	112,657

Note: Author's calculations using the Duke SSA/Medicare Data Set matched to historical records on the location of steel production. The sample includes only those who migrate out of Pennsylvania. Standard errors, clustered at the town level, are in parentheses. *significant at 0.05; **significant at 0.01.

TABLE 7. Impact of Elevation on Survival to Age 75, Individuals Born in Mill Towns

	Regression Coefficients
Log Elevation	0.0100*
	(0.0051)
Age × Cohort Effects Included?	yes
Sample Size	22,029

Note: Author's calculations using the Duke SSA/Medicare Data Set matched to historical records on the location of steel production and Geographical Analysis Tools (2012). Standard errors, clustered at the town level, are in parentheses. *significant at 0.05; **significant at 0.01.

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