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Toxic exposure in America: Estimating fetal and infant health outcomes from 14 years of TRI reporting

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ABSTRACT

We examine the effect of exposure to a set of toxic pollutants that are tracked by the Toxic Release Inventory (TRI) from manufacturing facilities on county-level infant and fetal mortality rates in the United States between 1989 and 2002. Unlike previous studies, we control for toxic pollution from both mobile sources and non-TRI reporting facilities. We find significant adverse effects of toxic air pollution concentrations on infant mortality rates. Within toxic air pollutants we find that releases of carcinogens are particularly problematic for infant health outcomes. We estimate that the average county-level decreases in various categories of TRI concentrations saved in excess of 13,800 infant lives from 1989 to 2002. Using the low end of the range for the value of a statistical life that is typically used by the EPA of $1.8M, the savings in lives would be valued at approximately $25B.

1. Introduction

Over 75,000 different chemical substances, used or manufactured in the United States, are currently registered with the EPA under the Toxic Substances Control Act (TSCA). The majority of those substances are relatively new, having been developed since World War II, and for many, little is known about their effects on health. Since 1988, the Toxic Release Inventory (TRI) has tracked environmental releases by manufacturing plants in the U.S. of 300 to 600 of these substances, all of which are either known to be, or suspected of being, hazardous to human health. It is estimated that, in 2000, more than 100 million lbs of carcinogens, 188 million lbs of developmental or reproductive toxins, 1 billion lbs of suspected neurological toxins, and 1.7 billion lbs of suspected respiratory toxins were released into the nation's air, water, and land by the manufacturing sector alone.[1]

Toxic substances face cradle-to-grave regulation in the U.S.: Their storage, handling, transportation, and disposal are all strictly regulated. Yet, for most of these substances, there is no formal regulation of their *releases* into the environment. In part, this may be due to a belief that at low levels of perceived exposure there are no significant health effects.[2] And, to a large extent, there was little public concern over toxic releases until the discovery in 1978 of toxic wastes buried beneath a neighborhood in Love Canal, N.Y., and then of a strong correlation between residential proximity to Love Canal and significantly elevated rates of cancer, neurological disorders, birth defects, and still births.

Love Canal spurred a number of epidemiological studies into the health effects of toxic exposure. The bulk of that research consists of cross-sectional studies, usually on adults, and provides mixed results on the relationship between toxic pollution exposure and health outcomes. That is similar to what has been observed in the literature on (non-toxic) air pollution and health. As pointed out by Greenstone and Chay (2003a), the lack of a consensus on the effects of air pollution on health may be explained by identification problems due to omitted variable bias that often arise in cross-sectional studies. A second problem is that studies of adult health outcomes may be flawed by the inability to measure accurately life-time exposure to pollutants. Even abstracting from mobility issues, using current levels of pollution to proxy for life-time exposure will be

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[1] See U.S. PIRG Report, executive summary (January 22, 2003).

[2] No comprehensive data set exists for ambient toxic pollutants. Data on ambient toxic concentrations for only a small number of toxic pollutants have been recorded for a select number of states in 1996, and only periodically since that time.

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inaccurate if pollution concentration levels have changed dramatically over time, as is true of toxic pollutants (Needham et al., 2005).

A third problem is the absence of data on toxic pollution concentrations. At best, toxic releases are available at the facility level in the manufacturing sector for facilities that are required to report to the TRI. No data exist, however, for TRI non-reporters within the manufacturing sector or toxic polluters not required to report to the TRI. Because the contributions of pollution from these sources are unobserved and change over time, they cannot be accounted for in cross-sectional studies. Studies thus far, have not controlled for these time-varying omitted variables, potentially leading to estimation bias.

In this study, we investigate the health effects of toxic pollution exposure on two particularly vulnerable groups: fetuses surviving at least 20 weeks in utero and infants under one year of age. By doing so, we mostly avoid the problems associated with trying to proxy for life-time exposure levels. Empirical studies show that mobility rates for pregnant women are low, so that fetal exposure can reasonably be approximated by pollution concentrations in the mother's county of residence.

We construct a panel in which we make use of facility level annual toxic release data that we aggregate to the county-year level and link to files of all births and deaths in the U.S. between 1989 and 2002. We include a large set of covariates to control for potentially confounding effects. In particular, we explicitly include proxy variables to control for toxic pollution from TRI non-reporting manufacturing sources and for mobile sources of pollution – two potentially important variables which have systematically been omitted from other studies. One proxy variable for TRI non-reporting facilities is constructed using county-level census data for all operating manufacturing facilities and is based on the ratio of TRI reporting facilities to all (operating) manufacturing facilities by 2-digit SIC code. The second proxy variable accounts for the potential level of TRI releases from non-reporting facilities by constructing an index which takes into account the distribution of non-reporting facilities by industries within each county and weights it by the national average of TRI releases within the 2-digit SIC code. The proxy for mobile source pollution is constructed from releases of criteria air pollutants that are known to be correlated with fuel combustion. Our central identification strategy is based on using what we believe are plausibly exogenous changes in toxic pollution concentrations within state-years with county fixed effects to estimate the causal effect of toxic pollution exposure on infant and fetal health outcomes.

Our findings show that there are significant health consequences to infants from exposure to toxic releases. We do not, however, find similar outcomes for fetal health, although this may be due to "harvesting" that occurs during the first 20 weeks of gestation so that fetuses that would normally survive at least 20 weeks in utero survive less than 20 weeks due to toxic pollution exposure. We cannot test this hypothesis directly, however, due to poor data quality for fetal deaths that occur during the first 20 weeks of gestation.

We do find that toxic air releases are significantly more harmful to infant health than other forms of releases (e.g. water or land) and that carcinogenic air releases have the largest effect on infant mortality. We estimate that the average county-level decline in toxic air concentrations of 9.5% per year in the manufacturing sector alone led to a total decline in infant mortality of approximately 4% in 14 years. The over all reductions by TRI reporters in the manufacturing sector in various categories of TRI concentrations (by chemical category and by media) during our sample led to a savings of over 13,800 infant lives. Using value of a statistical life measures used by the EPA of between $1.8M and $8.7M, we estimate that the value of the saved lives ranges between $25B and $121B. Our findings, however, may significantly under estimate the actual effects of toxic releases on infant mortality, as they do not include the adverse health consequences of releases by TRI non-reporters for which we find evidence. Moreover, these estimates almost surely underestimate the true savings to society associated with the reduction in toxic releases as we do not include health benefits accruing to older children and adults as we do not learn about longer term health benefits from decreased long-term exposure. Finally, in contrast to other studies, we do not find any correlation between measurable health effects on infants or fetuses from exposure to ambient concentrations of criteria air pollutants, specifically, particulate matter (PM_{10}) (our proxy for mobile source pollution), or from ozone (O_3).

The rest of the paper is organized as follows. In Section 2 we provide a brief summary of the literature, focusing in particular on epidemiological studies that relate fetal and infant health outcomes to toxic pollution exposure. We discuss data sources that are used in our study in Section 3; descriptive statistics are given in Section 4. Section 5 describes our methodology, and Section 6 discusses data issues. In Section 7, we present our results. In Section 8 we describe tests for robustness, and in Section 9 we discuss policy implications and provide concluding remarks.

2. Background

It is generally believed that both fetuses and infants are particularly vulnerable to exposure to toxic pollutants, although the biological mechanisms through which that occurs are not yet well understood. The National Research Council described four ways in which these two groups may be especially vulnerable to environmental toxins (Landrigan et al., 2004). Children have disproportionately heavy exposures to many environmental agents because of their size. Relative to their body weight, they consume significantly more food and water than adults. Toxins that are present in the food system or in the water supply may therefore be more harmful to them than to adults. And, because the central nervous system is not fully developed until at least 6 months post birth (Choi et al., 2006), the blood–brain barrier may be breached by some environmental toxins in a manner that is less likely later in life. It is also believed that developmental processes are more easily disrupted during periods of rapid growth and development before and after birth, making exposure to environmental toxins during these stages particularly harmful.

Before addressing the question of fetal or infant health outcomes from exposure to environmental toxins, it is important to address directly the question of how to measure toxic exposure. Fetal exposure is a direct consequence of maternal exposure. Most studies assume that the relevant level of exposure may be captured by the mother's place of residence at the time of delivery. That will be true, however, only if the mobility rate of pregnant women is low. Published studies have estimated residential mobility during pregnancy to range between 12 and 32%, with one study estimating that, of those that moved, only 5% changed municipality and 4% changed county during pregnancy (see Fel et al., 2004; Khoury et al., 1988; Shaw and Malcoe, 1992; Zender et al., 2001). In combination, those studies would suggest that, at most, 1.2% of pregnant women would not have been in residence within their child's birth-designated county during pregnancy and at most 1.6% would not have been in residence within their child's birth-designated municipality.

The finding of low mobility rates amongst pregnant women is important as it bounds the potential confounding effects stemming from pregnant women moving to counties (or municipalities) with different pollution characteristics based on a Tiebout-type sorting mechanism (see Banzhaf and Walsh, 2008). This could lead to