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TOXIC EXPOSURE IN AMERICA:

ESTIMATING FETAL AND INFANT HEALTH OUTCOMES

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ABSTRACT: We examine the effect of exposure to toxic releases that are tracked by the Toxic Release Inventory (TRI) on county-level infant and fetal mortality rates in the United States between 1989-2002. We find significant adverse effects of TRI concentrations on infant mortality rates, but not on fetal mortality rates. In particular, we estimate that the average county-level decrease in aggregate TRI concentrations saved in excess of 25,000 infant lives from 1989-2002. Using the low end of the range for the value of a life that is typically used by the EPA of $1.8M, the savings in lives would be valued at approximately $45B. We also find that the effect of toxic exposure on health outcomes varies across pollution media: air pollution has a larger impact on health outcomes than either water or land. And, within air pollution, releases of carcinogens are particularly problematic for infant health outcomes. We do not, however, find any significant effect on health outcomes from exposure to two criteria air pollutants – PM_{10} and ozone.

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I. 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 these 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 300-600 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 pounds of carcinogens, 188 million pounds of developmental or reproductive toxins, 1 billion pounds of suspected neurological toxicants, and 1.7 billion pounds of suspected respiratory toxicants were released into the nation's air, water, and land by the manufacturing sector alone.[1]

Toxic substances in the U.S. face cradle-to-grave regulation: 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.

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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.

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 consensus on the effects of air pollution on health may be explained by serious problems in identification in cross-sectional studies due to omitted variable bias. Furthermore, 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 inaccurate if pollution concentration levels have changed dramatically over time, as is true of toxic pollutants (Needham et al. (2005)).

In this study, we focus on the health effects from 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 have shown that mobility rates for pregnant women are low, so that fetal exposure reasonably can be approximated by pollution concentrations at the mother's county of residence.

We construct a panel data set that makes 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 1989-2002. We include covariates to control for a number of potentially confounding effects. Our central identification strategy is based on exploiting the high level of variation in TRI concentrations both between counties and within counties over time.

Our preliminary findings suggest that there are significant health consequences to infants of exposure to toxic releases, although we do not find similar outcomes for fetal health. In particular, we estimate that the over-all change (decrease) in annual average county-level TRI concentration (net of pollutants regulated under the Clean Air Act (CAA)) that occurred between 1989-2002 was responsible for a decline of over 25,000 infant deaths. Valued at between $1.8M - $8.7M per life, this savings would be valued at between $45B - $217.5B.

We also find that the medium by which the toxic pollutants are released into the environment matters for health outcomes: toxic air releases are significantly more harmful to infant health than either water or land releases. This may suggest something about the mechanism by which infants are exposed differentially to these substances. We find also that carcinogenic air releases have the largest effect on infant mortality of all the pollutants that we study. In contrast to other studies, we

do not, however, find any measurable health effects on infants from exposure to ambient concentrations of either particulate matter (PM_{10}), or ozone (O_3).

The rest of the paper is organized as follows. In section II 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 III; descriptive statistics are given in section IV. Section V describes our methodology and section VI discusses data issues. In Section VII, we present our preliminary results. In Section VIII we provide describe tests for robustness that we conduct on the data, and in Section IX, we discuss policy implications and provide concluding remarks.

II. BACKGROUND

It is generally believed that both fetuses and infants are particularly vulnerable to exposure to toxic pollutants, although the biological mechanisms through which they occur are not yet well understood. The National Research Council described four ways by which these two groups were uniquely vulnerable to environmental toxins (Landrigan et al. (2004)). First, 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. Second, because the central nervous system is not fully developed until at least 6 months post birth (Choi (2006)), the blood-brain barrier may be breached by certain environmental toxins, in a manner that is less likely later in life. Third, 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. And fourth, because children have longer life-spans, exposure to environmental toxins at an earlier age, or even in utero, may lead to a higher probability of developing a chronic disease that might not occur if exposure were to occur later in life.

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 exposures. 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 only be true, however, if the mobility rate of pregnant women is low. Published studies have estimated residential mobility during pregnancy to range between 12% - 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 et al. (1992), and Zender et al. (2001).) In combination, those studies would suggest that, at most, 1% of pregnant women would not have been in residence within the birth-designated county during their pregnancy. Fel et al. (2004) also report that, in their study, mobility was not correlated with exposure to chemicals or pesticides in the workplace or at home. They did find, however, that both younger (age < 25) and older (age >35) women were more mobile, as were unemployed women and those from lower income groups.

Several epidemiological studies look at health outcomes for prenatal exposure to toxic pollutants. A number of papers find a correlation between prenatal exposure and spontaneous abortion, malformation, and low birth weight (Bove et al. (1995), Carpenter (1994), Landrigan et al (1999)). Others, however, find no such correlation (Baker et al (1988), Croen et al (1997), Fielder et al (2000), Kharrazi (1997), Sonsiak (1994)). More recent work suggests that the health effects may be tied only to particular categories of toxic pollutants. For example, Meuller et al. (2007) look at the relationship between fetal deaths and maternal proximity to a hazardous waste sites, but finds statistically significant results only for proximity to waste sites associated with pesticides.

Infant health outcomes may be affected both by exposure that occurs in utero and after birth. It is well documented that infants are at particular risk for exposure to heavy metals, such as lead and methyl mercury (Landrigan et al. (2004)). Choi et al. (2004) find that there is a higher risk of developing childhood brain cancers when mothers live close to a TRI emitting facility. Making use of TRI data, Marshall et. al (1997) find that there is a slight increase in certain birth defects due to exposure to toxic releases.

Because of similarities in terms both of econometric issues and issues of causality, it is useful to look also at the literature on air pollution and health. Greenstone and Chay (2003a), for example, examine the effects of total suspended particulates (TSPs) on infant mortality rates. They use the changes in TSP pollution concentrations that were generated by the 1981-82 recession as a "quasi-

experiment" to identify changes in infant mortality at the county level in the U.S. Their underlying assumption is that the recession-induced variation in county-level TSP concentrations is exogenous to infant mortality rates. They compare cross-sectional results for each year between 1978-1984 to a panel-data, fixed effects model (in first-differences) and show that the traditional cross-sectional approach can produce misleading results due to unobserved, omitted confounders. Using an approach that mitigates many of these identification problems, Greenstone and Chay find that a 1 $\mu g/m^3$ reduction in TSP concentration results in approximately 4-8 fewer infant deaths per 100,000 live births at the county level. Over the 1980-82 recession, they estimate that the reduction in TSPs led to approximately 2500 fewer infant deaths.

Currie and Neidell (2005) also look at the relationship between ambient air pollution concentrations and infant and fetal mortality. They focus on California during the 1990s and examine 3 different criteria air pollutants: carbon monoxide, particulate matter, and ozone. Unlike most other air pollution studies, Currie and Neidell allow for correlations across pollutants in their effect on infant mortality. Taking individual data that they aggregate up to the zip code-month level, they estimate an approximate linear hazard model and find a significant effect of carbon monoxide on infant mortality (although not on fetal mortality) and estimate that the significant reduction in carbon monoxide concentrations in California saved approximately 1,000 infant lives over the 1990s.

Taking our cues from both Greenstone and Chay (2003a, 2003b) and Currie and Neidell (2005), we make use of the variation in TRI releases across location (by county) and time to identify the effects of TRI releases on health outcomes. To control for potential confounding effects, we include parental characteristics, prenatal care information, and medicaid and other income transfers. We also allow for the possibility that other types of pollution exposure may affect health outcomes. In particular, we include measures for particulate matter and ozone concentration. Those two criteria air pollutants may also proxy for toxic air pollution concentrations that derive from mobile sources of pollution, as they are highly correlated with fuel combustion.

III. DATA

We combine data from various sources to construct a comprehensive set of measures at the county level for the period 1989-2002. Data on pregnancy outcomes are from the National Center

for Health Statistics (NCHS). Data on toxic emissions are from the Toxic Release Inventory, maintained by the U. S. Environmental Protection Agency (EPA). Those two data sets are supplemented by county level data on income, job composition, transfer payments from health and unemployment benefit programs, and population, all from the U.S. Bureau of Economic Analysis. Data on land and water area are taken from the U.S. Census 2000 Gazatteer Files. In this section we provide a detailed description of the primary data used in this study.

Health Outcomes Data

Our dependent variable and many important control variables are taken from infant[3] birth and death records, and fetal death records provided by NCHS. These records are constructed from a census of death and birth certificates, as required by law in all states.

The NCHS, in cooperation with the states and territories of the U.S., has promulgated a uniform instrument with which to collect information on each fetal death. (Note that our estimate of pregnancies comes from adding births and fetal deaths in a given year, as such it does not include terminated pregnancies.)

Infant Data: Birth certificates contain information about the parents of the baby, in addition to limited details about the medical history of the mother and the specific pregnancy. The variables that we use as controls include the reported age, education, marital status and race of the parents; tobacco and alcohol consumption; and the level of pre-natal care as indicated by the number of prenatal visits to a doctor.

We use death certificates to identify the cause of death as coded using the International Classification of Diseases. We remove infant deaths caused by external factors such as physical injuries from our measures, as they should not be related to the exposure of toxic releases. We refer to the retained observations as "internal" infant deaths.

It should be noted that death records generally do not contain information on county of maternal residence. We therefore compute the internal infant death rate as the ratio of infant deaths in a county to births by mothers residing in a county.

Fetal Data: Information in the fetal death files includes some of the same

[3] An infant is defined as being an individual under one year of age.

information that is available in birth certificates, such as the reported age, education, marital status and race of the parents; tobacco and alcohol consumption; and the level of prenatal care. The period of gestation is also included. Deaths of fetuses at less than 20 weeks are not well reported in the data set. Birth certificates and fetal death records also report the county of the mother's residence coded using the Federal Information Processing Standard (FIPS).

Using the individual level data described above, we compute county level statistics based on the county of residence of the mother, for infant death rates due to internal causes, and death rates for fetuses with a period of gestation of more than 20 weeks. Our control variables are also aggregated to the county level, by computing averages of measures such as maternal and paternal age, maternal years of education, and the number of prenatal visits. We also compute for each county and year the fraction of pregnant mothers in each of the following categories: white, African-American, smoking mothers, mothers that consume alcohol, and mothers that are married. The health data set, thus aggregated to the county-year level by the residence of the mother, is then merged with a data set on toxic releases.

Toxic Release Data

Data on toxic releases are taken from the Toxic Release Inventory. The TRI was introduced in 1986 under the Emergency Planning, Community Right To Know Act (EPCRA) and requires that all manufacturing plants with ten or more full time employees that either use or manufacture more than a threshold level of a listed substance report their toxic releases to a publicly maintained database. The first year of reporting was in 1987. At that time, there were approximately 300 TRI listed substances. In 1995, this list was expanded to include 286 new substances. Today (2008), the TRI covers 581 individually listed chemicals, 27 chemical categories, and 3 delimited categories containing another 58 chemicals. Reporting thresholds have remained at 10,000 lbs (annually) for most chemicals, with the exception of 4 persistent, bio-accumulative, toxic chemical (PBT) categories, and 16 PBT chemicals. (EPA website: www.epa.gov/tri/lawsandregs/pbt/pbtrule.htm) Because of changing thresholds and both the addition and deletion of reporting chemicals over time, we restrict our analysis to the stable base set of 1988 chemicals that are not affected by changing reporting thresholds.

TRI data are reported at the facility level. Separate reports are filed for each TRI substance

for which the facility meets the reporting requirements. Data are broken down by medium (air, water, underground, etc.), and information is provided as to whether the substance is known to be carcinogenic. Using TRI-provided information on chemical CAS number, we further classify TRI chemicals as a developmental or reproductive toxin if it is listed as such in the State of California Safe Drinking Water and Toxic Enforcement Act. The TRI data set also provides information on whether a given chemical is simultaneously regulated under the Clean Air Act.

Using this, we construct, for each county-year observation, the total pounds of TRI releases *net* of any Clean Air Act releases by air, water, and "land" (where land is the residual category = aggregate releases - air releases - water releases); broken down into by carcinogenic, and developmental and/or reproductive emissions. (We exclude CAA chemicals from our measures of TRI concentrations to avoid any possibility of "double counting" because we include measures of criteria air pollution concentrations in our models of health outcomes.) Using geographic data from the Census 2000 Gazatteer Files, we construct a crude measure of "concentration" by dividing total pounds of releases by land-area.

Criteria Air Pollution Data

When examining the relationship between TRI releases and health, it is important to control for the effect that other pollutants may have on health outcomes. We therefore supplement the TRI pollution data with data on concentrations of criteria air pollution, as provided by EPA's National Air Data Group. Those data were extracted from recordings taken from pollution monitors located in various counties across the nation. The data set provides means, variances, medians, and higher percentiles of concentrations observed by monitoring stations in a given day of a year. Of these values, we make use of the daily average concentration and the 95^{th} percentile concentration. In some counties, there are multiple monitoring stations. In those cases, we use the simple average across all monitoring stations for the daily average concentration and for the 95^{th} percentile concentration. Most counties, however, do not have any monitoring stations that measure all categories of criteria air pollution concentrations. We choose to concentrate on particulate matter (PM_{10}) and ozone (O_3) because these pollutants had the least number of missing county level observations, and because a number of studies have shown a potential link between their ambient concentration levels and adverse health outcomes for both infants and the unborn. An additional

benefit of including PM_{10} and O_3 in our study is that they are highly correlated with mobile source emissions of pollution. This is important, as we cannot directly control for mobile sources of pollution, and they are known to be major contributors to airborne toxic releases.

Other Data Sources

Several county level controls are also used in our study. Data on per capita income, Medicaid transfers, food stamp participation, and other government supplemental income transfers are taken from the Bureau of Economic Analysis (BEA). The fraction of the labor force employed in the manufacturing sector as well as county-level unemployment rates also come from the BEA.

IV. BIRTHS, DEATHS, AND TOXIC RELEASES: 1989-2002

The TRI-internal infant death and fetal death data set, linked with county-level demographics data, consists of 42,370 county-year observations. Between 1989-2002, there were over 56 million live births in the United States, with just under 420,000 internal infant deaths and 889,000 fetal deaths recorded. More than 55.8 billion lbs of toxic pollutants were released into the environment from the manufacturing sector, 4.2 billion lbs of which were carcinogens (2.9 billion lbs in the form of air releases), and 3.9 billion lbs of which were developmental or reproductive toxins (27.7 million lbs in the form of air releases).

Of the 42,370 county-year observations for which we have TRI, birth and infant/fetal death information, and county-level demographic information, only 11%, or 4,528 county-years, also have air monitoring stations that collect PM_{10} and ozone concentrations. The restricted sample that includes observations on (non-toxic) ambient air pollution concentrations captures 32.5 million live births, or 58% of the total, over the sample period, and is the basis for the regression analysis.[4] Select summary statistics for this more restricted data set (the "regression" sample) are presented in Tables 1-3, and described below. It consists of an unbalanced panel with between 243-350 counties in the United States. Those counties range in population from as small as 2,300 to as large as 9,700,000.

In real terms, average per capita income level is increasing during our sample period, as are

[4] Further discussion of how these observations were chosen, and the robustness of findings based on the restricted sample, is found in Section VI.

medicaid transfers (as well as other income transfers). Not surprisingly, the percentage of jobs in the manufacturing sector steadily declined. That may be important for our study, as TRI releases come predominantly from manufacturing, and workers in that sector may experience additional exposure to toxic chemicals in their workplace, which may in turn affect infant and fetal health outcomes.

With respect to parental characteristics that may affect health outcomes, we note that the average maternal age at birth increased slightly over time. If due to a reduction in teenage pregnancies, which are known to be associated with poorer health outcomes for both the fetuses and infants (reference), this might lead to lower infant and fetal mortality rates. If, on the other hand, it is due to women bearing children later in life, it might be detrimental to fetal and infant mortality. Maternal behavioral characteristics, however, clearly point to potential improvements in fetal and infant health. The consumption of tobacco during pregnancy fell dramatically over the 14 years covered by our study, from a high of 16.44% to a low of 8.61%. The consumption of alcohol during pregnancy likewise fell between 1990-1999, but then rose dramatically. One possible explanation for that reversal is an increasing number of studies began to suggest that there were positive (or no) health effects, for both mother and fetus, of a very small amount of alcohol consumption during pregnancy.[5]

Nationwide, mean county-level infant deaths from internal causes declined almost monotonically between 1989-2002 from 948.9 to 660.9 deaths per 100,000 live births, or by nearly 30%. A smaller decline (9%) was observed for fetal deaths (post 20 weeks gestation). In our regression sample, we observe a similar decline for infant deaths from internal causes (approximately 28%), but a much larger decline in fetal deaths (33%) than the national trend. We note also that internal infant mortality rates vary significantly across TRI concentrations (net of Clean Air Act chemicals) by quartile, being significantly higher for the dirtiest TRI counties. The same pattern holds true for fetal mortality rates. (See Figures1and 2.)

Although TRI releases have fallen significantly when compared to their originally reported

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[5] See, for example, the meta-analysis done by Fade, Vivian B, Graubard, Barry; "Alcohol Consumption during Pregnancy and Infant Birth-Weight," Annals of Epidemiology. 4,4 (July 1994): 279-284.

levels in 1988 (over 40%, nationwide), the decline in releases has been far from monotonic. In our regression sample, between 1990-1999, aggregate TRI concentrations rose by over 140%. After1999, they fell by more than 65%. Similarly, TRI air concentrations increased by 22% between 1990-1999 and fell by 53% between 1999-2002. The pattern for carcinogenic air concentrations is likewise, volatile. Carcinogenic air concentrations more than doubled between 1996-1997, and then increased by more than 16-fold between 1998-1999. It then fell by more than 85% between 1999-2002. (Similar patterns exist for the unrestricted sample of TRI reporters.) A quick glance at Table 3 shows that the variation in TRI releases, in aggregate form, as well as by select categories, is high both within and between counties.

In contrast to TRI concentrations, ambient air concentrations for ozone and particulate matter are quite stable throughout our sample. Average county-level ozone concentrations (ppm) rose from 0.0248 to 0.0283, whereas PM_{10} concentrations ($\mu g/m^3$) fell from 33.28 to 25.24. The variance in concentrations is very small, across time, across county, and even within county.

V. METHODOLOGY

To estimate the effects of toxic pollution on health outcomes (infant and fetal mortality), we adopt the widely used approach of assuming that the effects of the covariates on health is linear and additive.[6] The "true" model can thus be described by:

$$(1) \qquad Y_{it} = \beta X_{it} + \theta Z_{it} + \Pi W_{it} + \epsilon_{it}$$

$$(2) \qquad \epsilon_{it} = \lambda_{it} + \alpha_i + \gamma_t + u_{it}$$

where, i indexes county and t indexes year. X_{it} is our independent variable of interest, the concentrations of TRI releases; Z_{it} are a set of covariates that capture aggregate parental characteristics; W_{it} are controls for county-level income distribution and non-toxic air pollution concentrations, and u_{it} is an orthogonal error term.

To estimate β consistently, we require $E[X_{it} \cdot \epsilon_{it}] = 0$. By omitting county-fixed or time-fixed factors such as α_i or γ_t that are correlated with both TRI concentrations and infant (fetal) health

[6] See, for example, Greenstone and Chay (2003a).

statistics, we may introduce bias into the estimate of β. Unobservables that vary by county and year, like λ_{it}, may also bias the estimates if they are correlated with X_{it}. Ideally, one would like to introduce county-time interaction effects to absorb all potential sources of bias. That is not, however, feasible, due to a constraint on our degrees of freedom.

To efficiently estimate (1), we can use a first-difference model to remove the county-level, unobserved fixed effects. To do so, we take the difference between county level observations at period t and t-1 to obtain:

$$(3) \qquad \Delta Y_{it} = \beta \Delta X_{it} + \theta \Delta Z_{it} + \Pi \Delta W_{it} + \Delta \epsilon_{it}$$

$$(4) \qquad \Delta \epsilon_{it} = \Delta \lambda_{it} + \Delta \gamma_{t} + \Delta u_{it}$$

For consistent estimation of (3) after including time-fixed effects to control for $\Delta\gamma_{t,}$ we need to assume that $E[\Delta X_{it} \cdot \Delta \lambda_{it}] = 0$. This would imply that the change in pollution concentration undertaken by manufacturing plants in a particular county is not correlated with changes in other (uncontrolled) factors that are correlated with levels of pollution in that county. To control for any potential sources of bias that might violate $E[\Delta X_{it} \cdot \Delta \lambda_{it}] = 0$, we include state-time (in lieu of county-time) variables in our first-difference model. Our ideal equation for estimation using generalized least squares, weighted by the number of live births in a county (or number of unterminated pregnancies, in the case of fetal deaths), is then:

$$(5) \qquad \Delta Y_{it} = \beta \Delta X_{it} + \theta \Delta Z_{it} + \Pi \Delta W_{it} + \xi_{st} + \nu_{it}$$

where *s* indexes the state of county *i*.

For consistent estimation of (5), we must assume that $E[\Delta X_{it} \cdot \nu_{it}] = 0$. Intuitively, we are saying that the distribution of toxic pollution from the manufacturing sector across counties within a given state is exogenous to variations in county characteristics that may affect infant (fetal) mortality rates and that are not captured in ΔZ_{it} or ΔW_{it}. Since we control for state-time interaction effects, we need only assume that the location choice of different types of manufacturing industries (heavy polluters or otherwise) within a state is random with respect to other factors that might affect pre-natal or peri-natal health. Our assumption will also be reasonable as long as the variation in $\Delta\lambda_{it}$

within a state is low for each year in our sample. Our maintained assumption is that, by controlling for state-time interaction effects we have eliminated most sources of potential bias from our model.

An examination of the correlation between the TRI release statistics and covariates, Z_{it} and W_{it} indicate that the correlation between the levels of TRI pollution and most parental and county characteristics is low, as well as with the criteria air pollution concentrations (see Table 2). Only for Medicaid benefits do we observe a correlation greater than 15% with pollution concentrations. (For the sample of large counties > 250,000 in population, post 1996, we also find high correlations between pollution measures and demographic characteristics like racial composition and percentage of children born in wedlock. This, in and of itself, may be important for issues relating to environmental justice and public policy.) In any event, the correlation measures for those variables that we *can* explicitly control for suggests that bias due to λ_{it} should not be large. It may also be argued that if enough factors that potentially affect pre-natal and peri-natal health are controlled for, we do not need to worry about bias from $\Delta\lambda_{it}$ because our existing controls will absorb the effect of such confounding variables. A Hausman test for exogeneity may be used to test that hypothesis directly.

VI: DATA ISSUES

Although our full data set consists of approximately 42,370 county-year observations, we have relatively few observations for county-level criteria air pollution concentrations, reducing the total number of county-year observations available for our regression analysis to 4,198. When estimating the model described by equation (5), we also eliminate a small number of outliers, so that the estimating data set consists of 4,154 observations. We define outliers as being observations for which the change in total TRI concentrations (net of CAA chemicals) are either in the top or bottom 0.5% of the over-all distribution (based on the 42,370 county-year observations). For the regression data set, this amounts to dropping the top 23 and bottom 21 observations for changes in aggregate TRI concentrations. This restricted data set captures approximately 54% of all live births in the U.S. over our sample period.

We also find that a large number of counties report no TRI releases in a given year. This may be because there was no manufacturing activity in that county, or none of the facilities met the

reporting criteria. In the case where there was no manufacturing activity, then the correct measure for TRI releases in that county would be zero. If, however, the facilities simply did not meet the reporting requirements – due to the size of the facility, or by falling below the reporting threshold, a "zero" level of TRI release would under-estimate the true value. For our purposes, we report "zero" levels of releases for all non-reporting counties. This will tend to under-estimate the effects of TRI releases on health outcomes.

All results and estimates of elasticities, and numbers of lives lost or saved are based on the restricted regression sample which excludes outliers.

VII. Preliminary Results

Table 4 summarizes the effects of TRI concentrations on infant mortality and fetal mortality (post 20 weeks) rates per 100,000 live births or 100,000 unterminated pregnancies from estimating the first-difference model described in (5), above. All models include year dummies and year-state interactions. The infant mortality regressions are weighted by total number of live births in each county and year, whereas the fetal mortality regressions are weighted by the total number of unterminated pregnancies. Robust standard errors that allow for correlation across states are reported. Controls for parental characteristics are included in each specification as well as measures for real per capita income and medicaid transfers. We also include air pollution concentrations for PM_{10} and ozone that are recorded at air monitoring stations across the country. Hausman tests were used to test the exogeneity assumption required for (5) to yield consistent estimators, and in each specification described below, the null hypothesis of exogeneity could not be rejected at a 5% level of significance.

Aggregate TRI Releases

The estimated coefficient on aggregate TRI concentrations in the infant mortality regression is 0.008 and is statistically significant at the 5% level. This yields an implied elasticity of infant mortality with respect to TRI-concentration measured at the mean of approximately 0.007.[7] Using

[7] The mean, annual, county-level concentration of aggregate TRI pollution ($\overline{X}$) is 675.7 lbs/sq. mile. The mean, annual, county-level number of internal deaths ($\overline{Y}$) is 775.6. Using these numbers, we can

the annual, county-level average change in aggregate TRI concentration of -1.2%, or approximately 8.1 lbs/sq.mile/year, we estimate that over the 14 year sample period, the decrease in TRI concentration led to a savings of 25,237 infant lives. Using values of life numbers commonly used by EPA ($1.8M - $8.7M per life) suggests an aggregate value for these lives of $45.4B - $219.6B (see Table 7). We do not find any relationship between aggregate TRI concentrations and fetal death rates, post 20-weeks of gestation.

Somewhat surprisingly, we do not find any effects of non-toxic air pollution concentrations on health outcomes for infants. Those findings are robust to using either the mean aggregate concentration level or the 95th percentile measure for ozone and PM_{10}. One possible explanation may be that average county-level concentration levels are quite low during our sample period so that the health effects are small. Also, there is little variation across counties or over time in ambient ozone or PM_{10} concentrations in our data, which could explain the large standard errors on these two coefficients.

We do, however, observe that ambient concentrations of mean county-level particulate matter have a statistically significant, negative effect on fetal mortality rates. That is, when particulate matter concentrations increase, fetal death rates observed in the sample fall. Although this may be counter-intuitive, what it may reflect is a "harvesting" effect. Our data are limited to fetal deaths after 20 weeks gestation, and the fall in deaths among that group may be due to an increase in pre-20 week fetal deaths. Unfortunately, we cannot reliably test this hypothesis, because of the poor data quality on pre-20 week fetal death data.

Consistent with findings in other pollution-health studies, we find that per capita income and medicaid payments do not affect infant or fetal health outcomes. Maternal behavior, however, has a measurable effect on fetal mortality rates. There is a strong, positive correlation between tobacco and (despite some then contemporaneous studies suggesting the contrary) alcohol use and fetal mortality rates: increased use of either significantly increases the rate of fetal mortality post-20 weeks gestation.

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calculate the elasticity as: $\epsilon = \hat{\beta}\left(\frac{\bar{X}}{\bar{Y}}\right)$ or, 0.008 (675.7/775.6) = 0.007. See Table 7 for all values used in the elasticity calculation and the confidence interval on lives lost or saved.

TRI by Air and Water and Land

One question of interest is whether different pollution media have differential effects on health. For example, infants undergo direct exposure to air pollution and their small and less developed lung capacity may adversely affect their ability to deal with airborne toxins. They may thus be more susceptible to air than water pollution. Fetuses, on the other hand, are exposed to both air and water pollution only through maternal exposure. The mechanisms through which maternal exposure lead to fetal exposure almost surely differ across pollution media.

In Table 5, we report estimates for TRI concentrations partitioned by air, water, and "land," where land is simply the residual level of release once air and water releases are accounted for. We find that neither TRI water nor land releases have a measurable effect on either infant or fetal mortality rates. TRI air releases, however, have a large, significant effect on infant mortality rates. The coefficient on TRI air concentration is 0.023, implying an elasticity of approximately 0.005. For an annual average change in TRI county-level air concentration of -4.86% for 14 years, this would imply a savings of 73,776 infant lives, or $132.8B -$641.85B (see Table 7).

In the partitioned regressions we again find no statistically significant effects of non-toxic air pollution concentrations, per capita income levels, or income transfers on infant mortality rates, but we do still observe the (potential harvesting) effect of particulate matter on fetal mortality (post 20 weeks gestation). The effects of maternal behavior (tobacco and alcohol use) on fetal mortality rates are also similar to those found in the regressions using aggregate TRI concentrations.

TRI Carcinogens, Developmental, and Reproductive Toxins

Exposures to carcinogens and to developmental or reproductive toxins, are thought to be particularly hazardous to human health. Here, we look to see whether toxic releases that are either known or are suspected carcinogens, or developmental/reproductive toxins have a measurable affect infant and fetal mortality rates.

Because of our earlier finding that different pollution media may have different health effects, we now parse aggregate TRI releases (net of CAA pollutants) by both media (air, water, and land) and type (carcinogenic, developmental/reproductive, "other"), including a separate variable for each of the 9 different categories. We summarize our results in Table 6. What we find is that carcinogenic air concentrations have a large, significant adverse effect on infant mortality. With a coefficient

estimate of 0.33, or an implied elasticity of 0.0005, this suggests that given an average, annual, county-level *increase* in carcinogenic air concentrations of 0.8%, 1264 infant lives were lost between 1989-2002, with a value of between $2.28B - $11B. (See Table 7.)

We also find, however, that developmental/reproductive water concentrations have a large, *beneficial* effect on infant mortality. With a coefficient estimate of -0.66, and an implied elasticity of -0.0002, we estimate that the decrease in developmental/reproductive water concentrations of 6.9% (average, county-level increase), this led to a *loss* of 3666 lives between 1989-2002. One possible explanation for this surprising result on TRI developmental/reproductive water concentrations is that there may be a harvesting effect. Although we do not see any effect of developmental/reproductive water concentrations on fetal deaths post 20 weeks gestation (we observe a negative, non-significant effect), with respect to fetal deaths pre-20 weeks gestation, we do find a negative, significant effect (with the understanding that the data are not very accurate). A conceivable explanation may be that higher exposure to developmental or reproductive toxins leads to an increase in terminated pregnancies of potentially less-viable fetuses, which in turn may lead to a decline in fetal and infant death rates. Data on pregnancy terminations, unfortunately, is difficult to obtain. Further investigation on the validity of this result is still necessary.

It is important to note, however, that although this particular specification does not actually fail to reject the Hausman test for exogeneity, the p-value on the test (8%) is actually close to rejecting exogeneity at conventional levels, and it is the developmental/reproductive water concentration variable that appears to drive this result.[8] That raises concerns for the possibility that at least some of the coefficient estimators are biased and inconsistent in this model. And as just noted, we have reason to be concerned about the developmental/reproductive water concentration variable. This is not an issue in the aggregated models of TRI concentrations (presented in Tables 4 and 5), as this variable makes up a small proportion of over-all TRI releases (under 0.04%, on average, in any county-year). For our less-aggregated model, however, it is important to ensure that the values of the other estimated coefficients are robust. To do so, we re-estimate that specification,

[8] In the regression used to conduct the Hausman test, which included both levels and first-differences of the independent variables of interest, no levels variables were found to be individually statistically significant except for that on the developmental or reproductive water concentrations variable, which had a t-statistic greater than 2.

excluding the TRI developmental/water concentration variable. Reassuringly, we find that all coefficient estimates remain robust. Furthermore, in this restricted model, the p-value on the Hausman test rises to p = 0.86 (consistent with the p-values found in the aggregated TRI models), indicating that the coefficients have been consistently estimated.

We find a similar, negative effect with respect to developmental/reproductive air concentrations on fetal mortality. The coefficient estimate is -0.22, yielding an implied elasticity between fetal mortality and TRI developmental/reproductive air concentrations of -0.0004. With an average, annual, county-level decline of 2.62%, this would lead to an increase, over 14 years, of approximately 2865 additional fetal deaths.

VIII. Additional Checks for Robustness

Because of the complicated nature of our data set, it is important to ensure that our regression results are not driven by spurious correlation, outliers, or sample selection. Here, we discuss some of the tests for robustness that we conducted.

The most significant loss of data was due to the small number of county-year observations for which we have PM_{10} and ozone concentration data. Although we believe that it is critical to include these measures because (1) they may affect infant and fetal mortality rates and (2) they proxy for toxic releases from non-manufacturing sources (e.g. mobile sources of pollution), we re-estimated all regressions excluding those variables. In doing so, the total number of county-year observations that may be included in the regressions increases to 38,277, once the top and bottom 0.5% of the change in aggregate TRI county-year concentration observations are trimmed as outliers. (The number of observations reported here is more than 1% smaller than the full TRI-infant/fetal health data set due to the loss of county-year observations for which we do not have data on per capita income or other income transfers.) Not surprisingly, we find that using the larger sample (excluding PM_{10} and ozone concentrations) the coefficient estimates (standard errors) on the variables of interest are somewhat smaller (bigger) than for the restricted models reported in Tables 4-6, but of the same sign, magnitude, and general significance level. This shows both that the omitted variable bias is small (recall from Table 3 that the correlations between the criteria air pollution concentrations and TRI concentrations are less than 10%), but exists; and also that the

sample of counties for which we have air monitoring data does not introduce any significant sample selection bias.

We also explore the sensitivity of our results to our having assigned a "zero" concentration levels to counties that do not report TRI releases. That procedure tends to underestimate the health effects of TRI concentrations. To verify that this is the case and to obtain an upper bound on the health effects, we re-estimate all models excluding any county-year that has "zero" aggregate TRI concentration. The estimated coefficients remain stable (and, as anticipated, they are somewhat larger), and the level of statistical significance does not change dramatically. Hence, we conclude that little bias is introduced by that procedure.

There are also concerns with the accuracy of TRI reported releases in the early years of reporting, as well as with the quality of the infant birth and death files for small counties. As a check on these potential problems, we make use of the linked birth-death records for infants that exist for the years 1996-2001. The linked birth-death files exclude all births and deaths that cannot be linked due to low data quality. On average in a given year, over 95% of all infant death records are linked with the corresponding birth certificate. The public use linked files contain information on infant births and deaths for all counties with populations greater than 250,000. This data set consists of a balanced panel of 199 counties, accounting for approximately 58% of all live births in the country (8.12 million births of 14 million, nationwide, from 1996-2001). Using this much smaller and more restricted data set, our basic regression results remain robust.

Finally, as previously noted, we exclude a small number of outliers from our regression analysis. Outliers are defined as being in the top or bottom 0.5% (approximately) of the distribution for annual average county-level changes in TRI concentrations. In total, approximately 1% of our county-year observations were excluded, of which over 57.55% reported no TRI releases. Robustness checks on the stability of our results over different outlier criteria were conducted and all results are robust over all specifications.

IX. Conclusion

Although the transportation, storage, handling, and disposal of toxic substances are regulated, in general, their releases into the environment are not. Yet, the potential health effects from

exposure to these toxins could be devastating. Here, we attempt to look at the health effects from exposure to a large body of these chemical substances on two of the most vulnerable groups in society – infants and the unborn. The primary question of concern is whether at the current levels of toxic releases and their corresponding levels of toxic concentrations there are measurable adverse health consequences. A preliminary analysis of the data suggest that there are potentially large, statistically significant effects on infant mortality rates with increases in toxic concentrations. We find that infants are more sensitive to airborne concentrations of toxins than either land or water-born concentrations, over-all, and that they are particularly vulnerable to carcinogens. Between 1989-2002, we estimate that while the decline in average annual county-level TRI concentrations saved over 25,000 infant lives, an estimated gain of $45B-$217.5B.

From a policy perspective, our findings suggests that if government programs were to be developed to encourage reductions in toxic releases, the biggest health benefits for infants would come from policies aimed at reducing toxic air releases, in general, and carcinogens, in particular. Our research also highlights the need for monitoring of toxic pollution concentrations across pollution media. Currently, no comprehensive tracking exists. Our results are based on very crude measures of concentration and exposure and more precise measures could help to refine our results.

Further study also is needed also to determine whether there are specific chemicals that are driving the results that we obtain in this paper, or, whether it is the general mix of chemicals that are released into the environment that is doing the harm.

The lack of general regulatory over-sight on toxic emissions is almost surely because of the belief that low levels of toxic pollution concentrations are not harmful to human health. Our results, however, strongly suggest that the effects of exposure, even at the current levels of concentrations, are far from benign, at least for infants under 1 year of age, and that there may also be implications for fetal health as well.

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TABLE 1: DESCRIPTIVE STATISTICS*

	1989	1990	1991	1992	1993	1994	1995
Number of Counties in Full Sample	3138	3137	3137	3136	3139	3140	3140
Total unterminated pregnancies	4106988	4227266	4178607	4140357	4075704	4023016	3966182
Total live births	4045693	4162917	4115342	4069428	4004523	3956925	3903012
Infant deaths (internal) per 100,000 live births	948.89	886.81	856.55	819.23	794.73	765.16	725.31
Fetal deaths per 100,000 unterminated pregnancies	1492.46	1522.24	1514.02	1713.11	1746.47	1642.82	1592.72
Number of Counties in Regression Sample	259	259	287	299	321	342	337
Total unterminated pregnancies	2276913	2358116	2368082	2349333	2362106	2397621	2333222
Total live births	2249141	2328440	2338932	2316678	2328710	2363847	2301647
Infant deaths (internal) per 100,000 live births	980.60	909.49	874.93	830.54	803.41	781.10	734.17
Fetal deaths per 100,000 unterminated pregnancies	1219.72	1258.46	1230.95	1389.97	1413.82	1408.65	1353.28
Mean County-Level Characteristics							
Per Capita Income (2000)	25847.69	25804	25215	25595	25227	25279	25503
Medicaid Transfers in $000,000 (2000)	201406	227543	242947	276804	281851	287576	299217
% of Jobs in Manufacturing Sector	16.50%	16.17%	15.61%	15.39%	15.25%	14.97%	14.77%
Land Area (sq. miles)	1326.13	1326.13	1242.47	1262.29	1234.48	1231.31	1199.52
Water Area (sq. miles)	108.91	108.91	102.67	102.23	104.85	105.51	99.85
Population	500857	506700	472549	462372	446888	436574	443134
Mean Parental and Demographic Characteristics (Weighted by Unterminated Pregnancies)							
Years of Mother's Education	12.44	12.38	12.41	12.45	12.51	12.53	12.60
Mother's Age	26.58	26.66	26.71	26.84	26.94	27.02	27.13
Father's Age	29.92	29.89	29.93	30.03	30.13	30.19	30.25
% of White Mothers	74.90%	75.13%	75.93%	75.85%	75.56%	75.41%	76.05%
% of Black Mothers	19.82%	19.47%	18.89%	18.82%	18.99%	18.90%	18.20%
% Mother's Consumption of Alcohol	4.58%	3.71%	3.83%	2.82%	3.85%	3.48%	3.00%
% Mother's Consumption of Tobacco	17.44%	16.44%	15.83%	15.39%	14.14%	13.45%	12.27%
Number of Prenatal Visits	10.72	10.78	10.92	11.06	11.17	11.28	11.40
Percentage Married	69.77%	68.82%	67.65%	67.08%	66.28%	64.85%	65.74%
Mean Infant Health Endowment (Weighted by Live Births)							
Birth Weight (gms)	3325.92	3328.83	3325.50	3328.66	3320.80	3319.98	3318.76
Gestation Period (weeks)	39.09	39.06	39.03	39.03	38.95	38.93	38.91

Mean Fetal Health Endowment (Weighted by Unterminated Pregnancies)							
Birth Weight (gms)	1463.35	1426.67	1401.42	1413.07	1354.16	1344.59	1341.24
Gestation Period (weeks)	28.37	28.05	27.94	27.78	27.29	27.05	26.98
Mean Concentration Level for Pollution (Weighted by Live Births)							
Ozone - 8 hr (ppm)	0.0256	0.0248	0.0259	0.0243	0.0253	0.0259	0.0268
PM10 24-hr ($\mu g/m^3$)	36.6163	33.2771	33.3150	29.4102	28.8395	28.8842	27.6672
Mean Concentration Level for TRI Releases (lbs/sq. miles) (Weighted by Live Births)							
Total releases	550.416	519.983	601.2355	603.058	716.026	784.262	606.409
Air Releases	227.743	214.921	150.9805	183.683	174.073	174.365	138.964
Water Releases	29.924	21.754	14.70248	14.824	15.936	21.104	14.012
Carcinogenic Air Releases	1.1956	0.829	0.693	0.281	0.456	0.7204	0.302
Carcinogenic Water Releases	2.728	1.921	0.820	0.624	0.485	0.657	0.414
Developmental/Reproductive Air Releases	0.632	0.113	0.068	4.885	3.880	1.594	0.899
Developmental/Reproductive Water Releases	0.201	0.030	0.021	0.024	0.689	0.644	0.106
Releases of CAA Chemicals	462529.1	436302.3	541014.8	558580.8	567408.3	673722.9	380625.9

* All descriptive statistics are given for the regression sample, excluding outliers, unless otherwise noted.

TABLE I: DESCRIPTIVE STATISTICS, CONT'D

	1996	1997	1998	1999	2000	2001	2002
Number of Counties in Full Sample	3139	3140	3140	3139	3140	3141	3139
Total unterminated pregnancies	3960037	3948331	4008630	4027340	4126955	4085973	4082657
Total live births	3894874	3884329	3945192	3963465	4063823	4031531	4027376
Infant deaths (internal) per 100,000 live births	698.69	692.27	689.80	670.27	660.46	648.76	660.90
Fetal deaths per 100,000 unterminated pregnancies	1645.62	1620.99	1582.54	1586.03	1529.75	1332.41	1354.05
Number of Counties in Regression Sample	346	350	325	267	243	261	258
Total unterminated pregnancies	2323260	2254203	2187773	1985336	1705570	1755231	1716840
Total live births	2290368	2226631	2162954	1963250	1688017	1739053	1702742
Infant deaths (internal) per 100,000 live births	708.88	702.23	688.55	690.03	679.85	678.01	709.62
Fetal deaths per 100,000 unterminated pregnancies	1415.77	1223.14	1134.44	1112.46	1029.16	921.70	821.16
Mean County-Level Characteristics							
Per Capita Income (2000)	25751	26193	27489	27712	28182	28376	27983
Medicaid Transfers in $000,000 (2000)	299509	277783	287892	337331	317696	356553	377561
% of Jobs in Manufacturing Sector	14.74%	14.40%	13.67%	12.82%	12.87%	10.53%	9.68%
Land Area (sq. miles)	1228.55	1215.16	1280.13	1389.52	1160.04	1103.87	1220.36
Water Area (sq. miles)	101.75	101.69	103.13	106.54	101.79	95.98	93.10
Population	436980	428296	447787	489657	457300	454495	453593
Mean Parental and Demographic Characteristics (Weighted by Unterminated Pregnancies)							
Years of Mother's Education	12.63	12.69	12.74	12.67	12.75	12.80	12.78
Mother's Age	27.18	27.23	27.30	27.18	27.17	27.30	27.30
Father's Age	30.32	30.36	30.42	30.33	30.36	30.46	30.46
% of White Mothers	76.10%	76.23%	76.83%	75.50%	74.50%	75.46%	75.82%
% of Black Mothers	18.05%	17.82%	17.20%	18.15%	19.47%	18.93%	18.37%
% Mother's Consumption of Alcohol	2.51%	2.71%	1.84%	1.40%	7.00%	4.82%	6.69%
% Mother's Consumption of Tobacco	12.00%	11.76%	12.17%	10.87%	9.74%	9.16%	8.61%
Number of Prenatal Visits	11.44	11.54	11.55	11.58	11.53	11.49	11.48
Percentage Married	65.55%	65.63%	65.68%	64.23%	63.96%	63.92%	63.11%
Mean Infant Health Endowment (Weighted by Live Births)							
Birth Weight (gms)	3317.20	3312.46	3314.25	3307.07	3298.50	3289.02	3281.00
Gestation Period (weeks)	38.92	38.83	38.79	38.75	38.73	38.67	38.64

Mean Fetal Health Endowment (Weighted by Unterminated Pregnancies)							
Birth Weight (gms)	1345.28	1323.29	1316.90	1282.04	1275.49	1273.04	1242.93
Gestation Period (weeks)	26.95	27.21	27.24	27.47	27.40	27.42	27.45
Mean Concentration Level for Pollution (Weighted by Live Births)							
Ozone - 8 hr (ppm)	0.0264	0.0267	0.0282	0.0281	0.0267	0.0275	0.0283
PM10 24-hr ($\mu g/m^3$)	26.5492	26.7902	26.4785	27.7741	26.4085	25.6425	25.2431
Mean Concentration Level for TRI Releases (lbs/sq. miles) (Weighted by Live Births)							
Total releases	786.644	736.727	716.674	1254.847	632.316	472.289	445.048
Air Releases	137.685	144.750	200.773	259.540	119.148	124.840	120.602
Water Releases	16.947	13.097	17.346	16.483	10.753	15.636	19.651
Carcinogenic Air Releases	0.432	0.824	0.492	8.398	0.214	0.224	1.323
Carcinogenic Water Releases	0.419	0.785	2.005	1.977	0.113	0.363	0.196
Developmental/Reproductive Air Releases	0.709	1.024	1.661	0.618	0.373	1.336	0.662
Developmental/Reproductive Water Releases	0.424	0.425	0.180	0.060	0.009	0.0564	0.016
Releases of CAA Chemicals	536069	507474.4	655474.3	562178.9	267566	275911.9	272245.2

TABLE 2. CORRELATIONS OF TOXIC RELEASE CONCENTRATIONS WITH PARENT DEMOGRAPHICS AND COUNTY-LEVEL CONTROLS

Correlation of County and Aggregated Parental Factors with TRI concentrations net of Clean Air Act chemicals (lbs/sq. mile)

	Mean PM_{10} ($\mu g/m^3$)	Mean ozone (ppm)	Mother's Education	Mother's Age	Father's Age	Mother's Race: White
Air Releases	0.35%	-9.82%	4.48%	5.38%	6.21%	-4.61%
Water Releases	-1.48%	-8.71%	2.58%	6.31%	6.69%	-1.23%
Land Releases	0.07%	-3.60%	0.51%	-1.88%	-1.96%	-4.64%
Total Releases	0.07%	-5.29%	1.30%	-0.65%	-0.58%	-5.13%
Carcinogenic Air Releases	1.76%	-3.06%	0.05%	2.02%	1.78%	-9.01%
Carcinogenic Water Releases	0.37%	-6.35%	1.62%	4.82%	4.88%	-1.53%
Developmental/Reproductive Air Releases	0.35%	-3.65%	1.19%	-0.07%	1.88%	-0.02%
Developmental/Reproductive Water Releases	-2.12%	-4.47%	1.6%	3.13%	3.52%	1.22%
Releases of Clean Air Chemical	0.83%	-10.28%	-6.65%	-0.34%	1.70%	-8.21%

	Mother's Race: Black	%Alcohol	% Tobacco	Prenatal Visits	Married	Per Capita Income	Medicaid
Air Releases	3.62%	-1.85%	-0.52%	-3.52%	-2.17%	6.23%	3.05%
Water Releases	-0.40%	-1.61%	0.98%	-4.48%	1.00%	3.90%	0.30%
Land Releases	4.66%	-1.25%	-0.51%	-4.13%	-2.33%	-0.26%	1.94%
Total Releases	4.93%	-1.53%	-0.53%	-4.61%	-2.50%	0.91%	2.32%
Carcinogenic Air Releases	5.25%	-1.40%	-1.74%	-1.57%	-4.64%	4.19%	0.72%
Carcinogenic Water Releases	-0.47%	-0.37%	0.05%	-3.50%	1.47%	3.95%	-0.96%
Developmental/Reproductive Air Releases	0.70%	0.13%	2.03%	-0.33%	-1.72%	0.74%	0.21%
Developmental/Reproductive Water Releases	-1.09%	-0.41%	0.94%	-3.59%	-0.01%	1.05%	-0.58%
Releases of Clean Air Chemical	8.31%	-1.38%	-3.84%	-13.79%	-14.71%	-4.02%	18.51%

TABLE 3. WITHIN AND BETWEEN - COUNTY VARIATION FOR SELECT VARIABLES

Variable	Mean (Unweighted)	Standard Deviations		
		Overall	Between	Within
Total Births in County	7222.05	13584.87	11639.98	1336.74
Total Unterminated Pregnancies in County	7311.89	13703.38	11768.90	1349.41
Infant deaths per 100,000 live births: internal causes	748.62	306.63	233.69	227.18
Infant deaths per 100,000 live births: external causes	33.89	61.39	65.16	52.77
Fetal Death per 100,000 unterminated pregnancies	1248.19	1900.99	1854.18	507.56
County-Level Characteristics	26357.70	6114.64	6016.72	1789.94
Per Income Capital (2000 dollars)	290179.80	608665.10	560172.10	162925.50
Medicaid Transfer (2000 dollars)	14.19%	7.23%	7.84%	2.34%
% Employed in Manufacturing Industry	1767.33	16789.26	16752.40	6462.17
Parental and Demographic Characteristics	19.63	145.12	127.69	100.95
% of White Mothers	2341.64	18330.51	18711.69	6727.84
% of Black Mothers	202.22	1447.13	1566.93	753.20
% of Mothers consuming Alcohol	1.14	13.42	11.12	10.32
% of Mothers consuming Tobacco	234.85	1543.28	1615.42	855.86
% Married	774.95	15239.38	14496.67	7090.34
Concentration Level for TRI Emission (lbs/sq. mile)	0.56	15.62	16.15	8.64
Air Releases	190.65	650.98	578.31	393.04
Water Releases	18.89	136.48	111.70	95.62
Total Releases	777.45	5402.16	5843.63	2441.41
Carcinogenic Air Releases	0.90	13.55	5.55	12.31
Carcinogenic Water Releases	1.03	11.81	8.66	9.30
Developmental/Reproductive Air Releases	1.40	26.48	12.61	22.34
Developmental/Reproductive Water Releases	0.58	16.12	16.15	8.90
Releases of Clean Air Chemical	1598.99	17142.10	16543.41	6639.82

TABLE 4. ESTIMATED EFFECTS OF AGGREGATE TRI CONCENTRATIONS ON MORTALITY RATES

Variable	Δ Internal Infant Deaths	Δ Fetal Deaths (> 20 Weeks)
Δ TRI Concentrations (lbs/sq.mile)	0.00796*	0.00146
	(0.00409)	(0.00494)
Δ Mean PM_{10} ($\mu g/m^3$)	-1.21146	-1.45204*
	(0.94310)	(0.80748)
Δ Mean Ozone (ppm)	752.32926	-523.20385
	(1,326.81966)	(1,306.33806)
Δ Maternal Tobacco Use	-45.76362	70.19407**
	(35.81560)	(29.82474)
Δ Maternal Alcohol Use	-28.17296	81.99949*
	(46.92025)	(42.66148)
Δ Per Capita Income	Y	Y
Δ Medicaid	Y	Y
Δ Other Parent Demographics	Y	Y
Year Indicators	Y	Y
State -Year Indicators	Y	Y
Observations	3895	3895
R-squared	0.21	0.19

Robust standard errors in parentheses
* significant at 10%; ** significant at 5%; *** significant at 1%

Note: Internal mortality rates are per 100,000 births and fetal mortality rates are per 100,000 pregnancies. Internal infant mortality regressions are weighted by total number of births in each county and year. Fetal mortality regression is weighted by total number of pregnancies in each county and year.

TABLE 5. ESTIMATED EFFECTS OF TRI CONCENTRATIONS BY MEDIA ON MORTALITY RATES

Variable	Δ Internal Infant Deaths	Δ Fetal Deaths (> 20 Weeks)
Δ TRI Air (lbs/sq. mile)	0.02312**	0.01121
	(0.01003)	(0.01227)
Δ TRI Water (lbs/sq. mile)	-0.00866	-0.03404
	(0.04952)	(0.04620)
Δ TRI Land (lbs/sq. mile)	0.00484	0.00016
	(0.00486)	(0.00608)
Δ Mean PM_{10} ($\mu g/m^3$)	-1.21164	-1.46100*
	(0.94443)	(0.80721)
Δ Mean Ozone (ppm)	733.22040	-527.70335
	(1,325.91786)	(1,305.25042)
Δ Maternal Tobacco Use	-45.62264	71.88280**
	(35.69114)	(30.05122)
Δ Maternal Alcohol Use	-29.54001	80.88989*
	(46.99218)	(42.68681)
Δ Per Capita Income	Y	Y
Δ Medicaid	Y	Y
Δ Other Parent Demographics	Y	Y
Year Indicators	Y	Y
State -Year Indicators	Y	Y
Observations	3895	3895
R-squared	0.21	0.19

Robust standard errors in parentheses
* significant at 10%; ** significant at 5%; *** significant at 1%

Note: Internal mortality rates are per 100,000 births and fetal mortality rates are per 100,000 pregnancies. Internal infant mortality regressions are weighted by total number of births in each county and year. Fetal mortality regression is weighted by total number of pregnancies in each county and year.

TABLE 6. ESTIMATED EFFECTS OF TRI CONCENTRATIONS BY MEDIA AND TYPE ON MORTALITY RATES

Variable	Δ Internal Infant Deaths	Δ Fetal Deaths (> 20 Weeks)
Δ TRI Developmental/Reproductive Toxins: Air (lbs/sq. mile)	-0.06861	-0.22194**
	(0.10630)	(0.09268)
Δ TRI Carcinogens: Air (lbs/sq. mile)	0.33127**	0.01375
	(0.15189)	(0.26905)
Δ TRI Other: Air (lbs/sq. mile)	0.00319	0.00159
	(0.00978)	(0.01171)
Δ TRI Developmental/Reproductive Toxins: Water (lbs/sq. mile)	-0.65850**	-0.55733
	(0.26058)	(0.35560)
Δ TRI Carcinogens: Water (lbs/sq. mile)	0.23693	0.05050
	(0.25344)	(0.36044)
Δ TRI Other: Water (lbs/sq. mile)	0.02063	-0.00421
	(0.03269)	(0.07323)
Δ TRI Developmental/Reproductive: Land (lbs/sq. mile)	0.10165	-0.39360
	(0.21038)	(0.28557)
Δ TRI Carcinogens: Land (lbs/sq. mile)	0.00780	0.00579
	(0.00961)	(0.01183)
Δ TRI Other: Land (lbs/sq. mile)	0.00522	0.00004
	(0.00504)	(0.00601)
Δ Mean PM_{10} ($\mu g/m^3$)	-1.18709	-1.45647*
	(0.94594)	(0.80470)
Δ Mean Ozone (ppm)	543.62941	-314.08287
	(1,357.83770)	(1,305.96114)
Δ Maternal Tobacco Use	-44.50171	75.43777**
	(35.78864)	(30.31320)
Δ Maternal Alcohol Use	-24.58004	78.98279*
	(47.24545)	(42.69350)
Δ Per Capita Income	Y	Y
Δ Medicaid	Y	Y
Δ Other Parent Demographics	Y	Y
Year Indicators	Y	Y
State -Year Indicators	Y	Y
Observations	3895	3895
R-squared	0.21	0.19

Robust standard errors in parentheses
* significant at 10%; ** significant at 5%; *** significant at 1%

Note: Internal mortality rates are per 100,000 births and fetal mortality rates are per 100,000 pregnancies. Internal infant mortality regressions are weighted by total number of births in each county and year. Fetal mortality regression is weighted by total number of pregnancies in each county and year.

TABLE 7. ESTIMATED ELASTICITIES AND LIVES SAVED OR LOST: AVERAGE ANNUAL COUNTY-LEVEL VALUES

Variable	Mean (lbs/sq. mile)	Mean Change in Concentration	Estimated β	Standard Error	Aggregate: Point Estimate of Lives Lost (Saved)	Aggregate: Low Estimate of Lives Lost (Saved)	Aggregate: High Estimate of Lives Lost (Saved)
Total	673.2810	-1.20%	0.0080	0.0040	-25237	-49969	-505
Air	169.4333	-4.86%	0.0230	0.0100	-73776	-136645	-10906
Water	17.2978	-4.57%	-0.0090	0.0500	2769	-27374	32910
Carcinogenic Air	1.1702	0.84%	0.3310	0.1500	1264	831	6500
Developmental or Reproductive Water	0.2060	-6.94%	-0.6590	0.2600	3666	-1732	5340
Mean Internal Deaths (per 100,000 live births)			775.6				
Total Births (000,000)			27.8				
Total Pregnancies (000,000)			28.1				

FIGURE 1. AVERAGE ANNUAL COUNTY-LEVEL INTERNAL INFANT DEATH RATE PER 100,000 LIVE BIRTHS BY TRI QUARTILE

FIGURE 2. AVERAGE ANNUAL COUNTY-LEVEL FETAL DEATH RATE PER 100,000 LIVE BIRTHS BY TRI QUARTILE (> 20 WEEKS GESTATION)

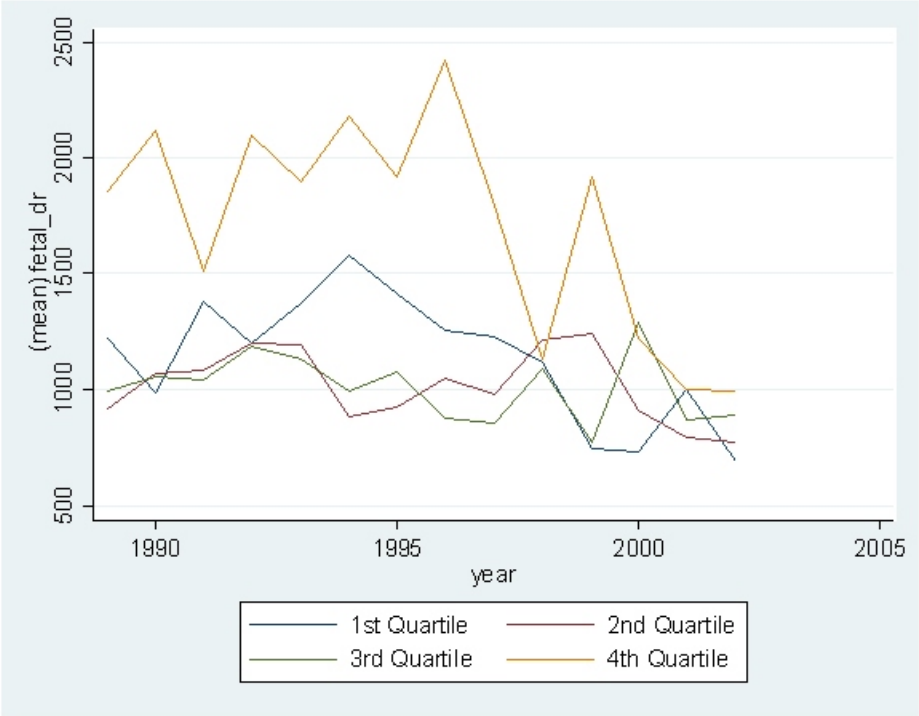

FIGURE 3. AGGREGATE TRI RELEASES BY MEDIA

FIGURE 4. AGGREGATE TRI AIR RELEASES BY TYPE

FIGURE 5. AGGREGATE TRI WATER RELEASES BY TYPE

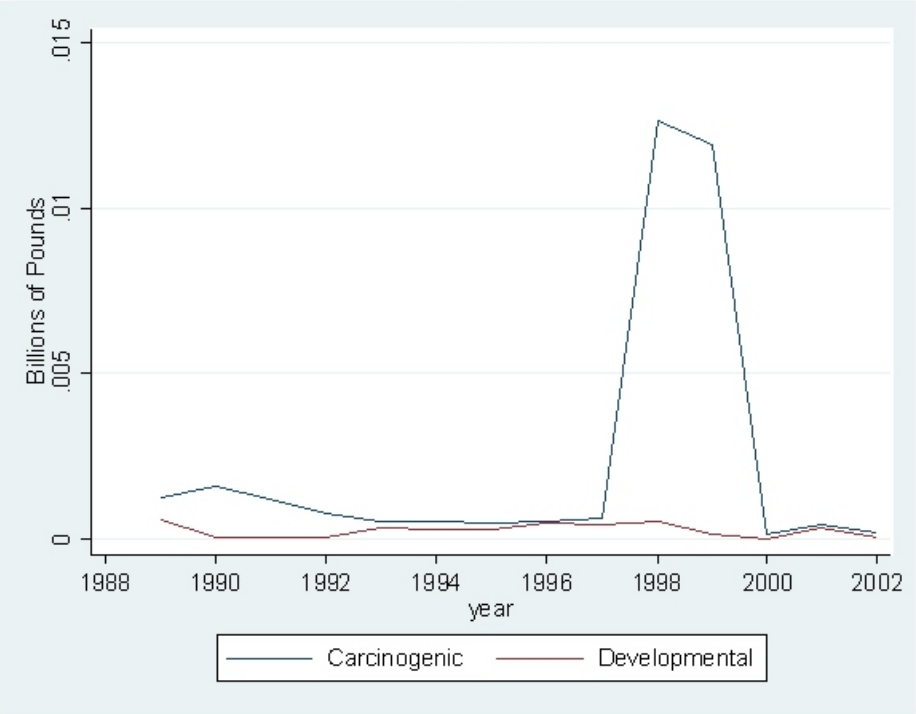

FIGURE 6. AVERAGE ANNUAL COUNTY-LEVEL TRI CONCENTRATION BY MEDIA

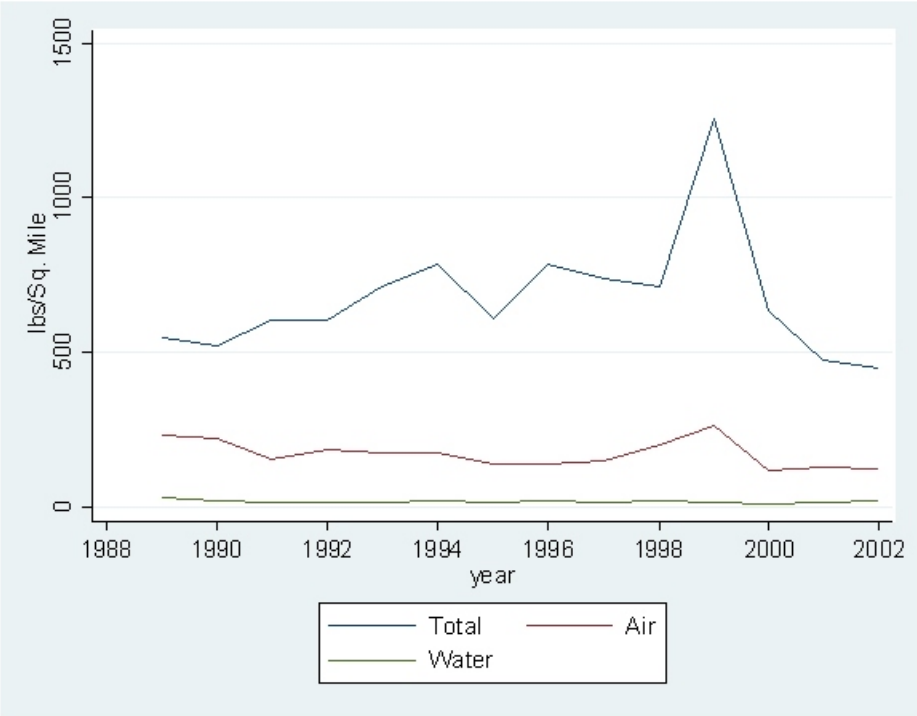

FIGURE 7. AVERAGE ANNUAL COUNTY-LEVEL TRI AIR CONCENTRATION BY TYPE

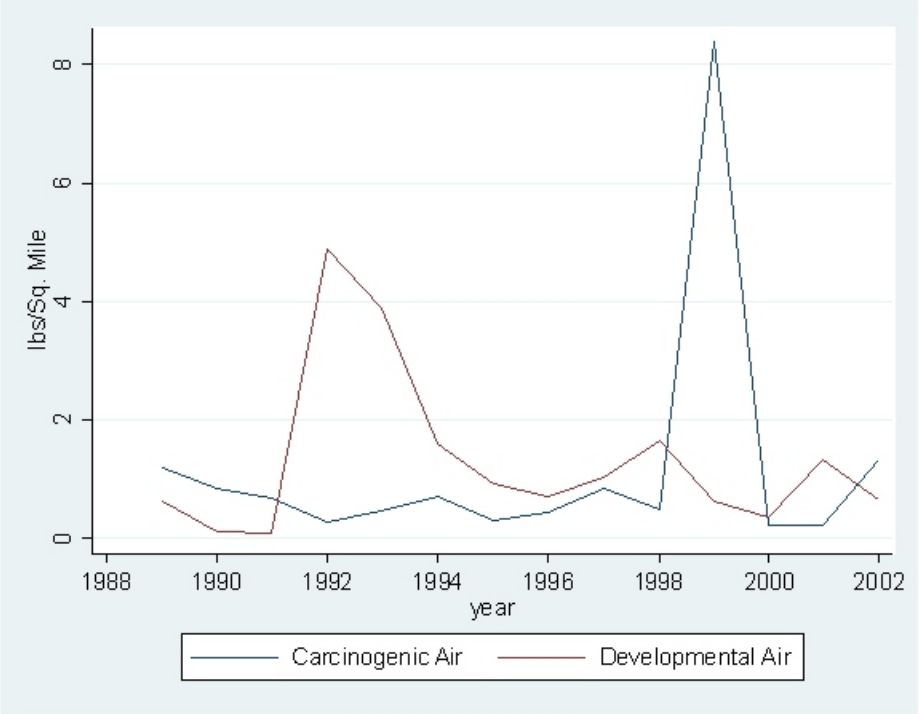

FIGURE 8. AVERAGE ANNUAL COUNTY-LEVEL TRI WATER CONCENTRATION BY TYPE