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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 a value of life of $1.8M - $8.7M, the savings in lives would be valued at $45B - $217.5B. 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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TOXIC EXPOSURE IN AMERICA: ESTIMATING FETAL AND INFANT HEALTH OUTCOMES FROM 14 YEARS OF TRI REPORTING

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 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 pounds of carcinogens, 188 million pounds of developmental or reproductive toxins, 1 billion pounds of suspected neurological toxins, and 1.7 billion pounds 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

[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.

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 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 exists, however, for TRI non-reporters within the manufacturing sector or toxic polluters not required to report to the TRI (including mobile sources). 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 and explicitly include proxy variables to control for toxic pollution from both mobile sources and from facilities in the manufacturing sector that do not report to the TRI – two potentially important variables which have systematically been omitted from other studies. Our central identification strategy is based on using exogenous changes in toxic pollution concentrations within state-years 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 a value of a statistical life measure 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. We find evidence to suggest that toxic releases by non-reporting facilities may add significantly to the impact on infant health outcomes. In contrast to other studies, we do not find any measurable health effects on infants or fetuses from exposure to ambient concentrations of criteria air pollutants, specifically, 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 results. In Section VIII we describe tests for robustness, 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 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)). 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 some 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. 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 than if exposure were to occur later in life. Finally, it should be noted that for infants, both pre- and post-natal exposure to toxic pollution may be important determinants of their health outcome.

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 et al. (1992), and 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.

Fel et al. (2004) also report that 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.

The finding of low mobility rates amongst pregnant women also 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 sample selection bias if, for example, high-risk pregnant women were sorting into high pollution counties based on some set of personal characteristics which then might lead to high infant or fetal mortality rates being falsely attributed to high pollution concentrations.

Several epidemiological studies look at health outcomes for prenatal exposure to toxic pollutants. A number 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 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 childhood brain cancer when mothers live close to a TRI emitting facility. Making use of TRI data,

Marshall et. al. (1997) find 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 (non-toxic) 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 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 and 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 4to 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 2,500 fewer infant deaths.

Currie and Neidell (2005) also examine 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 a cue from both Greenstone and Chay (2003a, 2003b) and Currie and Neidell (2005), we make use of the reduction in TRI releases across location and time to identify the effects of toxic pollution on health. Our maintained assumption is that both the reductions in toxic releases and the distribution and characteristics of industries across counties over time are exogenous to determinants of infant and fetal health outcomes. We base our assumptions on the following evidence: (1) Large polluting companies of TRI "priority" substances were contacted in early 1991and 1992 and were invited to participate in a "voluntary" abatement program called the TRI 33/50. Participants of this program agreed to reduce their releases by 33% from their 1988 baselines by 1992 and 50% by 1995. In total, over 1000 companies (and their facilities) participated in this program and all target reductions were met. (2) The deadlines to meet the requirements set out under the Montreal Protocol, an international agreement that required the phase-out of several ozone-depleting chemicals that are listed substances under the TRI occurred in 1994 and 1996 (and later in 2003, and 2005). These phase-out deadlines were also met. (3) Over 26 states adopted Toxic Use Reduction Programs to encourage the reduction of toxic substance use in the production process. These programs primarily provided educational outreach to polluters to help them abate, although some states instituted (numeric) state-wide reduction goals (some with target compliance dates, as well as penalties associated with non-compliance). (4) Nationwide recessions occurred in 1990-1991 and in 2001. The contraction of manufacturing during this time period also led to a reduction in TRI releases across the country. For many industries, for example the electronic industry, the recession in 2001 was exacerbated by a build up in inventory during the 1990 boom years, which further suppressed production. And (5) because reductions in toxic releases are "voluntary" in nature, only

facilities and industries that can abate at low cost will do so. We observe large variations across industry in their reported abatement activities. For example, during our sample period, petroleum refineries reduced their releases by 74% whereas the food industry increased their releases by more than 174%. We also observe that larger facilities report larger reductions in abatement than smaller facilities. Taking these five points together, we believe that there is ample evidence that the majority of the toxic reductions may be taken as exogenous to factors that determine infant and fetal health outcomes.

To control for other potential confounding effects, we include a rich vector of 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 are also used as a proxy for toxic air pollution concentrations that are derived from mobile sources of pollution, as they are highly correlated with fuel combustion. And, unlike other studies that have made use of TRI data, we construct two unique proxy variables that allow us to control for the effects of time-varying toxic releases from *non-reporting* TRI facilities.

III. DATA

We combine data from various sources to construct a comprehensive set of measures at the county level for the period 1989 to 2002. (We restrict ourselves to these years because of changes in the data format for our primary variables post 2002 that could lead to inconsistencies in the measurement of our variables.) 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 Gazetteer Files. In this section we provide a detailed description of the primary data used in this study.

Infant and Fetal Health Outcomes Data

Our dependent variables 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. (Our estimate of pregnancies comes from adding live births and reported fetal deaths in a given year; as such it does not include terminated pregnancies.)

Infant Data: Birth certificates contain information about parentage, 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; reported 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 injury) from our measures, as they are not related to the exposure of toxic releases. We refer to the retained observations as "internal" infant deaths.

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

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

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 likewise 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, mothers that smoke tobacco, 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 data 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 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, containing 16 PBT chemicals. (See 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 subsequent changes in reporting thresholds.[4]

TRI data are reported at the facility level. Separate reports are filed for each TRI substance for which the facility meets the reporting requirements. Information is provided as to whether the toxic pollutant is released on-site or transferred off-site. We restrict our reported analysis to on-site releases, although all results are robust to the inclusion of off-site releases. Data are broken down by medium (air, water, land, etc.), and information is provided as to whether the substance is a known carcinogen. 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 chemical is simultaneously regulated under the Clean Air Act.

With these data 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 by carcinogenic, and

[4] We calculate the correlations between the balanced panel of 1988 chemicals and the newer chemicals that were added to TRI reporting requirements and find that they are low – below 23%. This suggests that bias from not including those chemicals in our analysis should be reasonably small.

developmental and/or reproductive toxic emissions.[5] (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 Gazetteer 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 pollutants, 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 95th 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 95th 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 thought to be highly correlated with

[5] Some chemicals are classified as both carcinogenic and developmental and/or reproductive toxins.

mobile source emissions of pollution and are therefore used as controls for toxic pollution concentrations from mobile sources of pollution.

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. The number of facilities by 2-digit SIC code are taken from the County-level Business Patterns data collected by the U.S. Census Bureau.

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

The TRI-internal infant death and fetal death data set consists of 43,124 county-year observations; when linked with County Business Pattern data collected by the U.S. Census Bureau, we have 42,617 county-year observations; and when we further include county-level demographic data, we have 41,908 county-year observations. The last of these data sets is referred to as the "full" sample, which we describe, below.

Between 1989 and 2002, there were over 54.3 million live births in the United States, with 410,615 internal infant deaths and 381,988 fetal deaths (post 20 weeks) recorded. More than 34.2 billion lbs of toxic pollutants were released into the environment by TRI reporters from the manufacturing sector, 28.8 billion lbs of which were released on-site. Of the on-site releases, 3.12 billion pounds were carcinogens (2.68 billion lbs in the form of air releases) and 3.27 billion lbs of which were developmental or reproductive toxins (3.24 billion lbs in the form of air releases).

Of the 41,908 county-year observations for which we have TRI, birth and infant/fetal death

information, and county-level demographic information, only 10.8%, or 4,524 county-years, also have air monitoring stations that collect PM_{10} and ozone concentrations. Over the period of study, this sample covers 53% of the country's over-all population, 57.6% of live births, 41.5% of aggregate TRI releases, and 39.6% of TRI on-site releases, and is the basis for our regression analysis. Select summary statistics for this data set (the "restricted" sample) are presented in Tables 1 through 3, and described below. The restricted sample consists of an un-balanced panel with between 273 and 376 counties, ranging in population from 2,294 to 9,800,000.

In real terms, per capita income is increasing in our restricted sample, although not monotonically. Medicaid transfers (as well as other income transfers) are also increasing over our sample period. Not surprisingly, the percentage of jobs in the manufacturing sector steadily declined, from 16.48% to 9.51%. 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 in turn may affect infant and fetal health outcomes.

With respect to parental characteristics of possible relevance to health outcomes, we note that average maternal age at birth increased slightly over time. If that is due to a reduction in teenage pregnancy, known to be associated with poorer health outcomes for both the fetuses and infants, 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 17.55% to a low of 8.11%. The consumption of alcohol during pregnancy likewise fell between 1990 and 1999, but rose dramatically thereafter. One possible explanation for that

reversal is the appearance of studies suggesting that there were positive (or no) health effects, for mother or fetus, from small amounts of alcohol consumption during pregnancy.[6]

Nationwide, mean county-level infant deaths from internal causes declined almost monotonically between 1989 and 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 the restricted sample, we observe a similar decline for infant deaths from internal causes (approximately 29%), but a much larger decline in fetal deaths (20%) 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 for fetal mortality rates. (See Figures 1 through 3.)

In 1989, average county-level on-site toxic concentrations (weighted by live-births) were approximately 3,159 lbs/sq. mile; toxic air releases (net of CAA chemicals) made up over 63% and toxic water releases some 5.7% of all on-site releases. By 2002, average county-level on-site toxic concentrations had declined 47% to 1,680 lbs/sq. mile and the contribution to releases by air and water fell to 44% and 2%, respectively. During this same period, both carcinogenic and developmental/reproductive toxin concentrations fell, suggesting that the most toxic of the TRI releases participated in the observed over-all decline. It should be noted, however, that the declines in releases (and subsequently, concentrations) have been far from monotonic. Although the annual average change in toxic concentrations over the sample period is almost -4%, the standard deviation

[6] 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.

is over 13% with changes in county-level, average annual TRI concentrations ranging between -31% and +15%. (See Figures 4 through 6.)

In contrast to TRI concentrations, ambient air concentrations for ozone and particulate matter are reasonably stable throughout our sample. Average county-level ozone concentrations (ppm) rose from 0.0256 to 0.0282, whereas PM_{10} concentrations ($\mu g/m^3$) fell from 36.55 to 25.48. The variance in concentrations is small, across time, across county, and within county.

V. METHODOLOGY

The approach widely used to estimate the effects of non-toxic pollution on health outcomes (infant and fetal mortality) assumes that the effects of the covariates on health is linear and additive.[7] There is evidence, however, that suggests significant non-linearities in the effects of toxic pollution on infant health, possibly due to threshold effects. Because mis-specification of the functional form can lead to biased estimates, we have a more flexible specification to allow for a non-linear dose-response function by including quadratic terms of the toxic pollution covariates in our model.[8]

We assume, then, that the true relationship between infant mortality and toxic pollution can be modeled as

$$(1) \qquad Y_{it} = \beta_1 X_{it} + \beta_2 X_{it}^2 + \theta Z_{it} + \Pi W_{it} + \epsilon_{it}$$

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

where i indexes county and t indexes year. X_{it} is our independent variable of interest, the concentrations of toxic releases; Z_{it} are a set of covariates that capture aggregate parental

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

[8] In Section VII we discuss the validity of the quadratic toxic pollution concentration term.

characteristics; and W_{it} are controls for other county-level characteristics.

Because geographic information in our infant birth/death data is at the county level, we aggregate all data to the county-year level. An ordinary least squares estimator would equally weight large and small counties. To more accurately measure the effect of pollution on infant mortality, we use an estimation strategy that weights each county-observation by the number of live births in that county-year. For weighted least squares (weighted by live births) to consistently estimate β_1 and β_2, ϵ_{it} must be orthogonal to X_{it}. If there are county-fixed unobservables α_i, time-fixed unobservables γ_t, and county-time varying unobservables λ_{it} that are correlated with X_{it} (and Y_{it}), ϵ_{it} will no longer be orthogonal to X_{it}. Including county-time interaction terms would be one method that would correct all such possible biases if the data structure allowed for it. That approach is foreclosed, however, by a constraint on the available degrees of freedom because the covariates in our model are aggregated to the county-year level.

While it is therefore not possible to correct for all sources of bias from county-time varying unobservables, it is straightforward to correct for biases stemming from only county-fixed or time-fixed unobservables. One approach would be to use a model with county-demeaned variables to remove the county-level unobserved fixed effects, and to include dummy variables to correct for bias from the time-fixed unobservables. To do so, we take the difference between county-level observations at period *t* and mean county-level observation across all years to obtain

$$(3) \qquad Y_{it} - \overline{Y_i} = \beta_1 (X_{it} - \overline{X_i}) + \beta_2 (X^2_{it} - \overline{X^2_i}) + \theta (Z_{it} - \overline{Z_i}) + \Pi (W_{it} - \overline{W_i}) + (\epsilon_{it} - \overline{\epsilon_i})$$

$$(4) \qquad \epsilon_{it} - \overline{\epsilon_i} = \lambda_{it} - \overline{\lambda_i} + \gamma_t - \overline{\gamma} + u_{it} - \overline{u_i},$$

where $\overline{X_i} = \frac{\Sigma_t X_{it}}{T}$, etc.

For consistent estimation of (3) by WLS after including time-fixed effects to control for $(\gamma_t - \bar{\gamma})$, we need to assume that $(\lambda_{it} - \bar{\lambda}_i)$ is orthogonal to $(X_{it} - \bar{X}_i)$ conditional on time fixed-effects. This implies that the annual deviation in levels of pollution concentration by manufacturing plants in a particular county is not correlated with annual deviations in other (uncontrolled) factors that are correlated with infant health in that county. Since we control for county-fixed and time-fixed unobservables, these factors are exclusively those with significant variation across time within each county. Presumably, many of those factors are constant across all counties within a single state-year. For example, changes in policy within a state in a given year may affect both infant health and toxic pollution. So, to control for effects that are neither fixed within a county or across time, but are fixed within state-time groups, we include state-time variables in our demeaned model.

If the size of the residual county-time varying unobservables that are correlated with toxic pollution is not large and the within state-time variation is large enough, we can consistently estimate β_1 and β_2 using WLS. Table 2 presents the within state-time variation of the key variables in the model. The within state-time standard deviation of the demeaned variable of our county-level and parental demographic characteristics is less than a fifth of the overall standard deviation in most cases. We conclude that a model that accounts for county-fixed and state-time interaction effects will adequately control for unobservables that may induce bias in the WLS estimator. While the within state-time variation is not high for county characteristics, the within state-time standard deviation of each of our measures (in terms of county-demeaned variables) of toxic pollution concentration and the infant health statistic is at least a third of the overall standard deviation. This

gives us confidence that correcting for state-time interaction effects, in addition to county-fixed and time-fixed effects, has not purged our model of the variation that would be necessary for identification. As described more fully in Section II, we believe that the source of within state-time variation in the demeaned toxic pollution concentration stems from the distribution of manufacturing industries in the counties of a state.[9] Over time within a county, there is variation in the level of pollution abatement by different industries, induced by TRI reporting and other factors exogenous to health outcomes. This variation can be used to identify the effect of the concentration of toxic pollution of infant and fetal health. We therefore estimate the following model in which observations are weighted by live births:

$$(5) \qquad Y_{it} - \overline{Y_i} = \beta_1 (X_{it} - \overline{X_i}) + \beta_2 (X^2_{it} - \overline{X^2_i}) + \theta (Z_{it} - \overline{Z_i}) + \Pi (W_{it} - \overline{W_i}) + \xi_{st} + \nu_{it},$$

where s indexes the state of county i. ξ_{st} are state-time indicators and ν_{it} is an orthogonal error term.

For consistent estimation of (5), we assume that $E[(X_{it} - \bar{X}_i) \cdot \upsilon_{it}] = 0$ and $E[(X^2_{it} - \bar{X}^2_i) \cdot \upsilon_{it}] = 0$.

Intuitively, this says that the time demeaned 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 that are not captured in ξ_{st}, $Z_{it,}$ or W_{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. This assumption will also be reasonable as

[9] We also test this directly by examining whether industry level dummies have any explanatory power to predict variations in toxic releases at the county-level (where state-year fixed effects are included). The resulting F-statistic is sufficiently large to allow for rejection of the null hypothesis at the 1% significance level.

long as the variation in ($\lambda_{it} - \bar{\lambda}_i$) within a state is low for each year in our sample. By controlling for state-time interaction effects, we believe we have eliminated most sources of potential bias in 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 3, panel II). Only for Medicaid benefits and mother's race (black) 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. A Hausman test for exogeneity also may be used to test this hypothesis directly (this is discussed in Section VII).

Finally, we are concerned that errors both across time within a county and across counties within a state might not be independent. If TRI releases are serially correlated within a county or state-wide effects cause dependence in releases between two counties within a state, assuming that ν_{it} are distributed i.i.d. might yield inconsistent standard errors and invalidate any statistical inference. To avoid these problems, we cluster our standard errors within state-groups so that when computing standard errors, if *i* and *j* are two counties in the same state, we allow for Corr(ν_{it}, ν_{jt}) $\neq 0$. This error specification may also help to correct for spacial correlation that exists due to spillover effects across county borders within states, but not across states.

VI: DATA ISSUES

Toxic Pollution Concentrations

The estimating model described in (5) assumes that measurements of toxic pollution concentrations are available at the county-level. Virtually no data exist, however, on toxic pollution concentrations. So, in contrast with studies on criteria air pollutants where monitoring stations can provide concentration data, we must estimate toxic pollution concentrations.

It is widely believed that the two principle sources of toxic pollution are manufacturing activities, and mobile sources. That is our maintained assumption.[10] Even with that assumption, however, we can observe toxic releases only from TRI reporting facilities within the manufacturing sector and not from non-reporting TRI facilities or from mobile sources of toxic pollution. Not accounting for such factors obviously leads to a serious risk of omitted variable bias in our model. The problem, therefore, is how to control for these unobserved contributors to toxic releases.

Toxic releases from mobile sources of pollution are generated predominantly by internal combustion and therefore are correlated with non-toxic pollutants that are simultaneously generated in the same process. Here, then, we proxy for their releases through observed concentrations of PM_{10} and ozone, of which internal combustion is know to be a major source.

Controlling for toxic releases from non-TRI reporting sources is more complicated. Our

[10] TRI reporting requirements after 1998 were expanded to include a small number of non-manufacturing industries, including electric utilities and mining. We do not include these industries in our analysis; however, for the years in which we have TRI data for them, we calculate the correlations between releases from the "new" industries and releases from the "original" industries. The correlation between the new and original industries is under 14% for all TRI release types (by media and category), so we do not expect a significant bias from omitting these industries.

strategy is to construct two proxy variables for each county-year. Our first proxy variable captures the percentage of non-reporting TRI facilities in the manufacturing sector. The second takes into account both the number of *non*-reporting facilities by 2-digit SIC code in manufacturing and the relative dirtiness of those industries based on national annual TRI releases by reporting facilities. The construction of these variables is described more fully below.

Toxic Concentrations from TRI Reporting Facilities: County-level toxic pollution concentrations that originate from the manufacturing sector are measured as pounds of toxic releases per square mile.[11] Toxic release data are available for facilities in a specified range of manufacturing SIC codes that have at least ten full-time employees and that either use or manufacture more than a threshold level of a specified toxic pollutant under the TRI. For our analysis, we restrict ourselves to the 1988 balanced panel of both toxic pollutants and industries covered by the TRI.

As noted in the data section, the TRI provides information on whether the toxic releases are released "on-site" or are transported "off-site." Aggregate releases are defined as being the sum of both on-site and off-site releases that are produced at the facility. For this paper, we report the results only for on-site releases, although our results are robust to using aggregate releases as well.

Toxic Concentration Proxies for Non-TRI Reporters: A facility in a "designated" SIC code may be a non-reporter for several reasons: they may not have had 10 or more full-time employees,

[11] An alternative approach might be to look at the exact distance between a mother's residence (address) and a toxic plant to obtain a possibly better measure of exposure. This approach has been taken by some epidemiologists (see, for example, Choi (2004)) and is currently being explored by Janet Currie in preliminary, unpublished work that focuses on infant health, environmental justice, and toxic pollution exposure in New Jersey, Florida, Pennsylvania, and Texas (IHEA Conference, Summer 2007, Copenhagen). The nature of our data precludes us from taking this approach.

they may have fallen below the reporting threshold, or they may simply have failed to report. Although it is generally thought that non-reporters are small polluters, there is little evidence as to what over-all contribution they make to toxic pollution releases within a county or to what extent they may be correlated with reported releases. To address the issue of potential omitted variable bias in our estimation, we make use of the County Business Pattern data collected from the U.S. Census Bureau to construct two separate variables that we use to control for non-reporter toxic release concentrations.[12]

For the first variable, we determine the *total* number of facilities in operation by county in the manufacturing sector (SIC 20-39). From the TRI data we calculate the number of TRI *reporting* facilities by county and 2-digit SIC code. From these we construct a variable that is the percentage of non-reporting facilities within a county. Within the regression sample of 4524 county-year observations, 204 county-years had no reporting facilities, and 22 county-years had no non-reporting facilities. Over all, the average percentage of non-reporters within a county year is 92.8%, with a standard deviation of 8.1%. Counties with higher percentages of non-reporting facilities (above the mean value) tend to be counties with much lower TRI concentrations, lower percentages of employment in the manufacturing sector, and higher per capita income levels. These counties also have lower rates of fetal (> 20 weeks gestation) and infant mortality – both internal and external.

Because releases vary greatly both across industries and over time, and not just by the number of facilities, we construct a second variable that controls for the relative "dirtiness" of non-reporting facilities, depending on the distribution of non-reporters within a county over time. That is done by

[12] We thank Wayne Gray for suggesting the use of this data set, which allowed us to construct these proxy variables.

constructing an annual national index based on aggregating TRI data by 2-digit SIC codes and calculating average facility-level TRI releases. For each county and year, we then take the number of non-reporting facilities in each 2-digit SIC code and multiply it by a "dirtiness" index – namely the national "dirtiness" rank of that 2-digit SIC code. That value is summed over all industries in the county in each year to construct our second control variable. This variable assumes that the rank distribution of TRI releases by non-reporting facilities across industries and time is the same as for reporting facilities. To obtain a "pseudo-concentration" value, we divide the control variable by land area. This variable will be largest for counties with many non-reporting facilities in the dirtiest industries and smallest for counties with few non-reporting facilities in the cleanest industries.

As a check on the validity of our two variables to proxy for the contribution of toxic releases from non-reporting facilities, we construct the same two variables for *reporting* facilities. We then regress aggregate, *actual county-level TRI concentrations* on the newly constructed control variables and all other exogenous variables in our health-outcome model (the first-stage regression). Given the very large F-statistic from the first-stage regression, we conclude that they are strong instruments. This suggests that our proxy variables may be sound controls for toxic pollution contributions from unobserved *non-reporters*.

Measurement Error

There are two types of measurement error to consider. The first is classical measurement error that arises because we do not have "true" toxic pollution exposure or concentration measures. Instead, we make use of toxic pollution releases that we modify into a "concentration" measure by normalizing pounds of releases by county land area. This leads to attenuation bias in our estimates.

The second type of measurement error is non-classical measurement error that arises from

using survey data. Evidence in the labor literature shows that errors in survey data may be substantial and problematic when used for estimation purposes, and the direction of any bias may be difficult to predict. Although this type of measurement error almost surely exists in TRI data, we assume that TRI survey respondents are providing TRI release estimations that are based on their best available information and, more importantly, are making those estimations independent of county-level infant (or fetal) mortality rates. Under these conditions, the non-classical measurement error in TRI releases may be described as "optimal prediction errors" in the regressor, and no additional bias should be introduced into the estimators from this source of measurement error (see Hyslop and Imbens (2000)).

VII. Estimation Results

Tables 4 to 6 summarize the effects of TRI concentrations on infant mortality and fetal mortality (> 20 weeks) rates per 100,000 live births or 100,000 unterminated pregnancies from estimating the county-level fixed-effects model described in (5). Infant mortality regressions are weighted by total number of live births in each county and year, whereas fetal mortality regressions are weighted by the total number of unterminated pregnancies. We report standard errors that are robust to correlation between observations from within-state groups.

The primary regression model which is estimated using the restricted sample includes TRI concentrations and TRI concentrations squared,[13] as well as controls for parental characteristics, real per capita income and medicaid transfers. As described above, air pollution concentrations for PM_{10} and ozone are included to control for mobile sources of toxic pollution, allowing as well for the

[13] Tests of significance on the level and quadratic term for the TRI concentration variables show joint significance in all models.

possibility of health effects caused directly by those pollutants; our control for the percentage of non-reporting facilities and our proxy for TRI pollution releases (per sq. mile) from non-reporters are included to account for aggregate toxic pollution concentrations attributable to non-reporters. Hausman tests were used to test the exogeneity assumption required for (5) to yield consistent estimators for the preferred regression; in each specification described below, the null hypothesis of exogeneity for the TRI concentration variables of interest could not be rejected at a 5% level of significance.[14]

Aggregate TRI Releases

We present the results from our estimation of the health effects of aggregate TRI concentrations in Table 4. Column 1 is the primary model based on (5) and contains all of the covariates described above and is estimated using the restricted sample. Column 2 re-estimates the primary model but excludes county-level demographic variables and parental characteristics. Column 3 is based on the full sample and excludes county-level demographic variables, parental characteristics, PM_{10}, and ozone. Column 4 presents the results for the model given in Column 3 further excluding TRI non-reporter controls. Column 5 summarizes the results for fetal health outcomes using the restricted sample.

From column 1, our estimates suggest that aggregate TRI concentrations from reporting facilities in the manufacturing sector, although positive, do not have a statistically significant effect on infant or fetal health outcomes. These results are robust to the exclusion of parental characteristics and county-level income variables in the restricted sample (column 2), and parental

[14] The Hausman test consists of running the regression including leads on all variables of interest and conducting a Wald test on their joint significance.

characteristics, county-level income, non-reporting toxic concentration proxies, and criteria air pollution concentrations (column 3 and 4) in the full sample. Note that the coefficient estimates are remarkably stable in magnitude across all specifications and samples.

Although we do not report the estimates here, we do not find statistically significant effects on infant mortality rates for PM_{10} or ozone concentrations, which is consistent with the California results in Currie and Neidell (2005) but not with Greenstone and Chay (2003a). It should be noted, however, that there is very little variation in the concentrations of PM_{10} and ozone both across county and over time in our sample. This could make their effects on infant and fetal health outcomes difficult to identify. And, as found in earlier health-pollution studies, per capita income levels and income transfers also do not appear to have a measurable effect on infant and fetal health outcomes. We do find positive and (sometimes) statistically significant results for our two TRI non-reporter controls, which suggests that as both the percentage of non-reporters and the number of non-reporters in dirtier industries increases within a county, infant mortality rates rise.[15]

One possible explanation for why we do not find any health effects from aggregate TRI concentrations is that this measure obscures important heterogeneity in health effects either across pollution media, toxic chemical categories, or both. We turn now to these possibilities.

TRI by Land, Sea, and Air

The first question of interest is whether different pollution media have differential effects on health. For example, infants undergo direct exposure to air pollution and their less-developed

[15] There is multicollinearity between one of the proxy variables and some of the county-level demographic characteristics which sometimes lowers the significance level, however, joint tests of significance between the proxy variables and the county demographic variables show statistical significance. These findings are consistent throughout our results.

pulmonary capacity may adversely affect their ability to deal with inhaled 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.

We preface the discussion of our results here with a cautionary word: although we believe that our measure for toxic air pollution concentrations (pounds of toxic pollution divided by county-square miles) is reasonable, we are less certain about the accuracy of this measure for either water or land pollution. It is highly likely that the relevant area around a polluting source for the determination of exposure is much smaller for water and land pollution than for air pollution. We therefore expect that the degree of measurement error associated with our water and land pollution variables would lead to greater attenuation bias and more imprecise estimators.

In Table 5, we report estimates based on TRI concentrations partitioned by air, water, and "land," where land simply denotes the residual releases once air and water releases have been accounted for. We include quadratic terms for all TRI concentration variables. What we observe now is that both TRI air and water concentrations have strong, statistically significant effects on infant, but not fetal, mortality rates. Toxic releases into the land do not appear to affect either infant or fetal mortality.

Focusing on air releases, we observe that TRI air concentrations have strong, statistically significant effects on infant, but not fetal, mortality rates. From the estimates in column 1 of Table 5, we calculate the implied county-level, annual toxic air concentration elasticity (or, more precisely, the toxic air concentration from TRI reported on-site releases elasticity on infant mortality), measured at the mean, as 0.03. With an annual average decline in toxic air concentrations of

approximately 9.47% per year (measured over our 14 year sample), this suggests that the decline in toxic air concentrations between 1989 and 2002 saved over 9,979 infant lives. Using a value of a statistical life of between $1.8M and $8.7M, the cost savings would be approximately $18B to $86.8B. Similarly for water concentrations, we estimate an implied county-level, annual toxic water concentration elasticity, measured at the mean, of 0.004. Given an annual average decline of 12.4% in toxic water concentrations, we estimate that the decline in toxic water concentrations during our sample period led to a savings of approximately 1,716 infant lives. Taken together, approximately 11,694 infant lives were saved. Using a value of a statistical life of between $1.8M and $8.7M, the cost savings would be approximately $21.05B to $101.7B. (See Table 7.)

In the medium-based partitioned regression, we continue to find no statistically significant effects of criteria air pollution concentrations, per capita income, or transfers. And, consistent with our findings using aggregate TRI concentrations, the coefficients on our two controls for non-TRI reporter concentrations are positive and statistically significant here, as well.

We find our estimators for toxic air concentrations to be robust across various model specifications, although somewhat less so for toxic water concentrations once criteria air pollution concentrations are no longer included in the model. This might suggest correlation across these variables or sample selection bias associated with county-level characteristics associated with having air monitoring stations for both PM_{10} and ozone. A more likely explanation, however, is that toxic water and land concentrations are not as well measured as toxic air concentrations using our methodology and the attenuation bias more pronounced for these estimators.

TRI Carcinogens, Developmental, and Reproductive Toxins

Exposures to carcinogens and to developmental/reproductive toxins are thought to be

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

Because our earlier findings show that different pollution media have differential effects on health, we now parse aggregate TRI releases by both media (air, water, and land) and chemical category (carcinogenic, developmental/reproductive, "other"), including a separate variable for each of the 9 different categories. In doing so, however, we recognize that we may not obtain statistically significant results, as we lose a great deal of variation in these more narrowly defined chemical categories by media. Regression results are summarized in Table 6.

Of toxic air releases, carcinogenic air concentrations have the largest adverse effect on infant mortality, whereas developmental/reproductive toxins do not appear to have any measurable effect. With a coefficient estimate of 0.29 on the linear term and -0.0032 on the quadratic term, the implied elasticity for carcinogenic air concentrations is 0.0027. The average annual reduction in carcinogenic air concentrations during our sample period was 23.6%. Accumulated over 14 years, this suggests a reduction in infant lives lost of 2,179, or a valuation of between $3.9B and $19B. (See Table 7.)

Air toxins that are neither carcinogens nor developmental/reproductive toxins also have a significant effect on infant mortality. This result is robust over all of our estimated specifications, with coefficient estimates on the toxic air concentration variables remaining quite stable. We estimate that given an annual county-level decline of 9.3% over 14 years, the reduction in non-carcinogenic/developmental/reproductive toxins saved approximately 9,860 infant lives. Taken together with the lives saved from the reduction in carcinogenic air concentrations, we estimate an

aggregate reduction in lives lost from the reduction in toxic air concentrations of approximately 12,039, valued at between $21.7B and $104.7B.

We also find that concentrations of non-carcinogenic, non-developmental/reproductive toxins in water may also have an adverse effect on infant mortality, although the robustness of this result disappears if criteria air pollutant concentrations are not included in our model. This is similar to the pattern that we observed when we had TRI concentrations broken down only by pollution medium, and may suggest some important correlations across the toxic water variables and the criteria air pollution concentration variables, sample selection issues, or attenuation bias.

If we include criteria air pollution concentrations in our model, we find that toxic water pollution concentrations that are not carcinogenic or developmental/reproductive toxins also affect infant mortality. The coefficient estimates here are similar to those found for non-carcinogenic, non-developmental/reproductive air releases. Over the 14-year sample period, we estimate that over 1,774 infant lives were saved from the approximately 12.2% average annual county-level decline in toxic water concentrations.

VIII. Additional Checks for Robustness

Because of the complicated nature of our data, it is important to ensure that our findings are not driven by sample selection, spurious correlation, or outliers. We discuss these issues, below.

Timing: A potential concern that we must address is the timing of births and deaths. In our analysis, year t mortality is regressed on year t pollution concentrations; however, if mortality occurs at the beginning of the year, then year *t-1* pollution concentrations might be more relevant to the health outcome than year *t*'s. Without being able to match up calendar-year births and deaths accurately, it is not possible to test the potential problem of "timing" directly, without introducing

other methodological problems such as sample selection bias. With this understanding, we construct a data set that limits the set of births and deaths that we study to one that satisfies the following conditions for infants: if a death occurs, the death must occur before the end of December in a given calendar year, and the infant must have been conceived during the *same calendar year*. If the infant were carried to full term, this would mean that the infant must have been born no earlier than September of that same year. By constructing the data in this way, all pre- and post-natal exposure will occur in the same year as the death; hence there is no "timing" issue. Caution should be used in interpreting the coefficient estimates from this "September-cohort" data as there may be sample selection bias inherent in the construction of the sample. As a second check, we also re-run our original model including leads and lags for all toxic pollution variables. A priori, we would expect that if timing is not an issue, neither leads nor lags will be statistically significant. Results for these robustness tests are summarized in Table 8.

Note that for simplicity, we focus only on the model specification that breaks TRI concentrations down by media. Columns 1 and 2 use the September-cohort and estimates the effects of TRI concentrations on infant mortality. Column 1 contains all covariates except for PM_{10} and ozone concentrations to allow for the maximum number of county-years to be included in the sample; column 2 only contains the TRI concentration variables. In both cases, the coefficient estimators on TRI air are statistically significant and are very similar in magnitude to the coefficient estimators reported in Table 5. So, even when restricting ourselves to a September-cohort where timing issues do not exist, our coefficient estimators remain approximately the same. In columns 3 through 5, we re-estimate the models presented in Table 5 including leads and lags. In all but one specification do we find a statistically significant coefficient on a lead or lag variable. We believe

that given these results, timing issues are not problematic in our estimates.

Spurious Correlation: We must be concerned also about the possibility of spurious correlation driving our results. To ensure that this is not the case, we follow Greenstone and Chay's (2003a) methodology and re-estimate our model using *external* infant deaths as our dependent variable. External infant deaths include those from automobile accidents, murder, and trauma – deaths that should not be related to toxic pollution concentrations. Our TRI concentration variables should not be statistically significant in a regression with external infant mortality rates as the dependent variable if our results are not driven by spurious correlation. Regression results are provided in Tables 4 through 6, column 7. Note that these regressions are run on the restricted sample as they include controls for PM_{10} and ozone, although the results do not change if the unrestricted sample is used for the estimation. In only one case do we find a statistically significant coefficient on any of our TRI variables. This is in the specification which breaks TRI releases by medium and pollution category and is for the coefficient on the squared term of the residual water concentration (non-carcinogenic, non developmental or reproductive toxin). The coefficient is significant only at the 10% level, so we conclude that overall, our results are not driven by spurious correlation.

Outliers: Finally, we exclude a small number (4) of outliers from our regression analysis. These observations lie approximately 3 standard deviations away above the mean value of aggregate TRI releases, and appear to be due to data entry errors that had not been corrected as of 2005. To ensure robustness over our sample, however, we checked the stability of our results over different outlier criteria; results are robust over all specifications.

IX. CONCLUSION

Although the release of toxic chemicals is not directly regulated, the potential health effects could be significant. Our objective has been to study those health effects 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. Our analysis of the data suggests that there are potentially large, statistically significant effects on infant mortality rates at the county-level with increases in toxic concentrations, which would be obscured by looking only at aggregate TRI releases because of heterogeneity in health effects across media and chemical categories. Between 1989 and 2002, we estimate that the decline in county-level TRI concentrations in the manufacturing sector saved over 12,000 infant lives, at an estimated value of between $22B and $104B. It is important to note, however, that the above number of lives saved may be significantly underestimated. By constructing proxy variables to control for toxic releases from non-TRI reporting sources, we find statistical evidence that their contribution to toxic concentrations may also have an adverse effect on health outcomes.

From a policy perspective, our findings suggests 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 findings also suggest that much more information should be collected from current non-reporting facilities. Even if each non-reporting facility released a very small amount of toxic pollution into the environment, given the sheer number of non-reporters in the manufacturing sector, their aggregate contribution would be significant. Current TRI policy is contemplating the reduction of reporting requirements by TRI facilities, which would include allowing *fewer* facilities to report

their toxic releases to the public. Such a policy clearly would be detrimental to improving our understanding of how toxic releases affect health outcomes.

Our results are based on crude measures of concentration and exposure and more precise measures could help to refine our findings. Further study also is needed to determine whether there are specific chemicals that are driving the results, or, whether it is the general mix of chemicals that are released into the environment that is doing the harm. Spatial analysis may be important to determine whether proximity to a TRI-producing facility or an "off-site" treatment facility may lead to higher levels of adverse health outcomes, as well as to whether there are "cross-border" spill-overs – whether the border is at the zip-code, county, or state level. We suspect that with more refined geographic information on the residence of the pregnant woman that we could obtain more statistically reliable results on the effects of toxic water and land pollution exposure on fetal and infant health.

The lack of general regulatory oversight 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.

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TABLE 1: DESCRIPTIVE STATISTICS

Year	1989	1990	1991	1992	1993	1994	1995
Number of Counties in Full Sample	3138	3137	3137	3136	3139	3140	3140
Total unterminated pregnancies	4,106,988	4,227,266	4,178,607	4,140,357	4,075,704	4,023,016	3,966,182
Total live births	4,045,693	4,162,917	4,115,342	4,069,428	4,004,523	3,956,925	3,903,012
Infant deaths (external) per 100,000 live births	33.74	33.49	30.64	30.72	32.66	30.10	29.13
Infant deaths (internal) per 100,000 live births	948.89	886.81	856.55	819.23	794.73	765.17	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 Restricted Sample	273	302	312	329	355	365	363
Total unterminated pregnancies	2,300,939	2,507,635	2,402,515	2,411,194	2,471,157	2,456,792	2,413,694
Total live births	2,273,005	2,473,685	2,373,036	2,377,723	2,432,488	2,420,710	2,379,440
Infant deaths (external) per 100,000 live births	32.86	31.17	27.69	29.15	30.38	29.41	26.14
Infant deaths (internal) per 100,000 live births	978.88	902.06	866.02	828.61	800.09	778.57	730.59
Fetal deaths per 100,000 unterminated pregnancies	1214.03	1353.87	1227.01	1388.15	1564.81	1468.66	1419.15
Mean County-Level Characteristics (Restricted Sample)							
Per Capita Income (2000)	25,696.62	25,662.07	24,997.65	25,270.46	24,992.35	25,295.11	25,477.19
Medicaid Transfers (2000)	192,823.79	211,094.36	225,444.07	256,249.89	274,178.07	282,771.91	295,287.03
% of Jobs in Manufacturing Sector	16.48%	16.36%	15.78%	15.63%	15.30%	15.18%	14.88%
Land Area (sq. miles)	1344	1261	1267	1242	1241	1195	1200
Water Area (sq. miles)	114	107	101	103	108	102	99
Population	480795	463987	442367	433320	421427	420131	426843
Mean Parental and Demographic Characteristics (Restricted Sample, Weighted by Live Births)							
Years of Mother's Education	12.44	12.42	12.41	12.45	12.49	12.54	12.61
Mother's Age	26.58	26.70	26.71	26.84	26.94	27.03	27.14
Father's Age	29.91	29.90	29.92	30.02	30.12	30.20	30.27
% of White Mothers	75.11%	75.77%	76.43%	76.08%	75.68%	75.56%	76.14%
% of Black Mothers	19.65%	19.00%	18.34%	18.56%	18.84%	18.78%	18.03%
% Mother's Consumption of Alcohol	4.61%	3.77%	3.81%	2.77%	3.97%	3.44%	2.98%
% Mother's Consumption of Tobacco	17.55%	16.56%	15.96%	15.38%	14.29%	13.42%	12.23%
Number of Prenatal Visits	10.72	10.79	10.93	11.09	11.13	11.28	11.39
Percentage Married	69.85%	69.12%	67.92%	67.38%	66.34%	64.83%	65.73%

Mean Infant Health Endowment (Restricted Sample, Weighted by Live Births)							
Birth Weight (gms)	3326.51	3331.92	3327.62	3330.07	3321.48	3319.50	3318.56
Gestation Period (weeks)	39.10	39.07	39.03	39.03	38.95	38.93	38.92
Mean Fetal Health Endowment (Restricted Sample, Weighted by Live Births)							
Birth Weight (gms)	1466.12	1415.62	1403.82	1411.81	1347.41	1338.48	1340.23
Gestation Period (weeks)	28.40	27.77	27.97	27.78	27.12	26.92	26.82
Mean Concentration Level for Pollution (Restricted Sample, Weighted by Live Births)							
Ozone - 8 hr (ppm)	0.0256	0.0247	0.0259	0.0244	0.0250	0.0260	0.0269
PM10 24-hr (µg/m3)	36.55	32.94	33.30	29.25	28.76	28.87	27.68
Mean Concentration Level for TRI Releases by Manufacturing Industries (lbs/sq. miles) (Restricted Sample, Weighted by Live Births)							
Total Onsite releases	3158.573	2757.896	2488.981	2880.275	1986.141	1897.177	1635.504
Air Releases	2009.079	1597.872	1371.826	1225.091	1017.201	1013.555	866.445
Water Releases	178.965	193.387	191.788	169.109	107.978	85.582	46.736
Carcinogenic Air Releases	25.610	12.577	7.915	6.998	7.757	6.729	5.659
Carcinogenic Water Releases	9.763	8.728	6.788	5.369	5.483	4.199	2.964
Developmental/Reproductive Air Releases	28.419	26.799	13.720	13.408	9.456	3.822	4.234
Developmental/Reproductive Water Releases	1.681	1.403	2.883	0.801	0.980	0.659	0.334

TABLE 1: DESCRIPTIVE STATISTICS CONT'D

Year	1996	1997	1998	1999	2000	2001	2002
Number of Counties in Full Sample	3139	3140	3140	3139	3140	3141	3139
Total unterminated pregnancies	3,960,037	3,948,331	4,008,630	4,027,340	4,126,955	4,085,973	4,082,657
Total live births	3,894,874	3,884,329	3,945,192	3,963,465	4,063,823	4,031,531	4,027,376
Infant deaths (external) per 100,000 live births	30.73	29.84	28.49	34.24	33.22	33.39	33.69
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	1645.52	1620.99	1582.54	1586.03	1529.75	1332.41	1354.05
Number of Counties in Restricted Sample	376	374	341	289	281	283	277
Total unterminated pregnancies	2,403,439	2,320,646	2,277,093	2,064,808	1,890,658	1,910,580	1,895,966
Total live births	2,367,951	2,290,749	2,247,445	2,040,164	1,867,408	1,890,269	1,877,578
Infant deaths (external) per 100,000 live births	28.17	27.63	27.72	32.15	32.29	34.39	33.82
Infant deaths (internal) per 100,000 live births	705.25	700.43	686.91	687.05	678.43	681.91	696.48
Fetal deaths per 100,000 unterminated pregnancies	1476.55	1288.31	1302.01	1193.53	1229.73	1063.08	969.85
Mean County-Level Characteristics (Restricted Sample)							
Per Capita Income (2000)	25,615.16	26,163.54	27,548.15	27,726.69	28,432.88	28,260.60	27,882.85
Medicaid Transfers (2000)	293,128.75	275,592.01	303,439.31	335,439.64	327,910.65	375,998.55	394,886.84
% of Jobs in Manufacturing Sector	14.98%	14.53%	13.75%	12.73%	13.35%	10.29%	9.51%
Land Area (sq. miles)	1253	1203	1261	1402	1092	1187	1281
Water Area (sq. miles)	98	100	103	105	94	92	91
Population	416124	413366	444356	471508	443266	454518	464706
Mean Parental and Demographic Characteristics (Restricted Sample, Weighted by Live Births)							
Years of Mother's Education	12.62	12.69	12.74	12.68	12.79	12.78	12.78
Mother's Age	27.17	27.24	27.32	27.19	27.25	27.30	27.37
Father's Age	30.31	30.37	30.45	30.35	30.42	30.47	30.52
% of White Mothers	76.27%	76.31%	76.48%	75.21%	74.90%	74.57%	75.60%
% of Black Mothers	17.81%	17.72%	17.45%	18.30%	19.13%	19.53%	18.23%
% Mother's Consumption of Alcohol	2.46%	2.67%	1.82%	1.37%	6.46%	4.48%	6.11%
% Mother's Consumption of Tobacco	11.89%	11.75%	11.97%	10.74%	9.95%	10.27%	8.11%
Number of Prenatal Visits	11.43	11.53	11.51	11.58	11.47	11.45	11.49
Percentage Married	65.56%	65.67%	65.36%	64.19%	64.13%	63.44%	63.21%

Mean Infant Health Endowment (Restricted Sample, Weighted by Live Births)							
Birth Weight (gms)	3316.79	3312.47	3313.25	3306.02	3300.48	3289.02	3283.78
Gestation Period (weeks)	38.92	38.83	38.79	38.75	38.74	38.68	38.65
Mean Fetal Health Endowment (Restricted Sample, Weighted by Live Births)							
Birth Weight (gms)	1345.95	1321.22	1309.65	1278.44	1263.89	1271.86	1237.69
Gestation Period (weeks)	26.85	27.09	26.93	27.35	26.98	27.24	27.22
Mean Concentration Level for Pollution (Restricted Sample, Weighted by Live Births)							
Ozone - 8 hr (ppm)	0.0265	0.0267	0.0280	0.0280	0.0266	0.0273	0.0282
PM10 24-hr (µg/m3)	26.64	26.80	26.54	27.72	26.04	25.57	25.48
Mean Concentration Level for TRI Releases by Manufacturing Industries (lbs/sq. miles) (Restricted Sample, Weighted by Live Births)							
Total Onsite releases	1634.223	1888.138	1905.801	1975.801	2154.782	1747.925	1680.158
Air Releases	822.450	825.871	812.137	820.395	784.857	756.025	736.299
Water Releases	34.725	43.519	44.344	40.260	32.640	40.489	32.594
Carcinogenic Air Releases	6.382	3.609	2.893	2.764	3.034	3.244	3.525
Carcinogenic Water Releases	3.198	1.692	1.533	1.488	1.195	1.220	1.473
Developmental/Reproductive Air Releases	2.143	2.088	2.201	1.379	0.952	1.041	0.911
Developmental/Reproductive Water Releases	0.181	0.206	0.266	0.238	0.542	1.363	0.218

TABLE 2. WITHIN STATE-TIME VARIATION FOR SELECT VARIABLES (RESTRICTED SAMPLE)

Variable	Mean (Weighted By Live Births)	Overall Standard Deviation	Within State-time Standard Deviation	Within State-time Standard Deviation of Demeaned Variable
Health Statistics				
Infant deaths per 100,000 live births: internal causes	770.79	251.62	171.26	104.51
Infant deaths per 100,000 live births: external causes	30.05	26.61	22.81	20.10
Fetal Death per 100,000 unterminated pregnancies	695.60	217.71	152.15	95.98
County-Level Characteristics				
Per Income Capital (2000 dollars)	28563.72	7042.91	6207.70	1177.03
Medicaid Transfer (2000 dollars)	1243989.10	1960629.00	1496427.00	389867.70
% Employed in Manufacturing Industry	13.22%	5.76%	4.29%	1.55%
Parental and Demographic Characteristics				
% of White Mothers	75.76%	16.08%	11.30%	1.11%
% of Black Mothers	18.51%	16.25%	10.45%	0.97%
% of Mothers consuming Alcohol	3.56%	7.96%	5.21%	4.83%
% of Mothers consuming Tobacco	13.05%	8.28%	6.60%	5.43%
% Married	66.04%	10.60%	8.63%	1.66%
Concentration Level of TRI Releases (lbs/sq.mile)				
Total Onsite	2141.81	6136.72	5187.64	2703.26
Air	1063.27	1876.20	1540.26	579.76
Water	92.16	427.61	376.21	252.00
Carcinogenic Air	7.26	31.75	23.23	20.38
Carcinogenic Water	4.10	27.61	24.27	16.86
Developmental/Reproductive Air	8.33	123.23	118.71	90.02
Developmental/Reproductive Water	0.86	11.27	10.14	8.84

TABLE 3. CORRELATIONS BETWEEN TOXIC CONCENTRATIONS, PARENT DEMOGRAPHICS, AND COUNTY-LEVEL CONTROLS (RESTRICTED SAMPLE)

PANEL I: VARIABLES IN LEVELS

	Mean PM10	Mean ozone	Mother's Education	Mother's Age	Father's Age	Mother's Race: White
Air	13.60%	-11.45%	-7.66%	-15.02%	-8.31%	-31.70%
Water	7.49%	-6.39%	-2.86%	-9.33%	-8.50%	-9.28%
Land	4.91%	0.64%	-3.77%	-9.79%	-7.75%	-5.09%
Total	13.01%	-4.43%	-4.19%	-11.78%	-6.69%	-15.84%
Carcinogenic Air	10.30%	-5.97%	-2.93%	-4.37%	-1.57%	-8.21%
Carcinogenic Water	2.18%	-3.47%	1.25%	-1.42%	0.04%	-2.56%
Developmental/Reproductive Air	0.72%	-4.43%	3.06%	3.68%	2.36%	-1.47%
Developmental/Reproductive Water	1.93%	-1.28%	-0.09%	-2.47%	-2.60%	0.09%

	Mother's Race: Black	%Alcohol	% Tobacco	Prenatal Visits	Married	Per Capita Income	Medicaid
Air	35.51%	0.89%	-0.59%	-5.35%	-24.50%	-0.86%	10.43%
Water	12.03%	-0.76%	5.48%	-3.60%	-8.26%	-3.93%	-0.21%
Land	6.98%	-0.85%	-1.39%	0.10%	-3.38%	-5.50%	-2.14%
Total	19.62%	-0.69%	3.64%	-2.32%	-14.65%	-5.07%	7.06%
Carcinogenic Air	9.92%	3.10%	4.11%	-7.38%	-7.17%	-1.61%	5.21%
Carcinogenic Water	4.39%	1.40%	1.93%	-1.82%	-0.88%	-0.29%	0.69%
Developmental/Reproductive Air	1.86%	1.07%	0.18%	-3.75%	1.58%	2.89%	0.35%
Developmental/Reproductive Water	0.91%	-0.82%	1.61%	-1.00%	3.15%	0.00%	-0.85%

PANEL II: DE-MEANED VARIABLES (DE-MEANED FOR STATE-TIME AND COUNTY FIXED EFFECTS)

	Mean PM10	Mean ozone	Mother's Education	Mother's Age	Father's Age	Mother's Race: White
Air	2.21%	2.42%	-2.19%	-7.72%	-10.62%	-11.67%
Water	2.12%	-0.04%	-2.40%	0.58%	-1.84%	-3.94%
Land	1.98%	1.01%	-3.36%	-3.36%	-2.10%	1.40%
Total	2.63%	1.52%	-4.01%	-4.92%	-4.52%	-1.48%
Carcinogenic Air	3.06%	1.55%	1.85%	5.30%	-1.62%	-3.69%
Carcinogenic Water	-0.17%	0.25%	0.92%	1.88%	1.68%	0.85%
Developmental/Reproductive Air	-0.72%	-7.36%	-3.25%	-7.56%	-6.75%	7.20%
Developmental/Reproductive Water	-0.97%	-1.92%	1.02%	-2.04%	-4.88%	1.20%

	Mother's Race: Black	%Alcohol	% Tobacco	Prenatal Visits	Married	Per Capita Income	Medicaid
Air	20.20%	0.84%	5.45%	-8.03%	-6.87%	-4.59%	-17.92%
Water	5.59%	-0.18%	1.26%	-3.85%	-0.04%	2.36%	-2.91%
Land	-1.92%	0.04%	-1.15%	-2.55%	0.12%	-0.30%	1.08%
Total	2.95%	0.20%	0.15%	-4.60%	-1.35%	-1.06%	-3.05%
Carcinogenic Air	4.81%	-0.38%	1.71%	-1.95%	-0.12%	4.75%	-7.58%
Carcinogenic Water	0.11%	1.31%	1.33%	5.37%	1.38%	6.19%	-0.49%
Developmental/Reproductive Air	-6.03%	0.03%	-0.48%	4.23%	-2.20%	-2.96%	-0.20%
Developmental/Reproductive Water	-0.93%	-0.53%	0.19%	2.74%	3.32%	1.45%	1.59%

TABLE 4. ESTIMATED EFFECTS OF AGGREGATE TRI CONCENTRATIONS

Variable	Internal Infant Deaths				Fetal Deaths	External Deaths
	Restricted Sample (with PM_{10}, ozone)		Full Sample (without PM_{10}, ozone)			
Aggregate TRI (lbs/sq.mile)	0.0006	0.0018	0.0023	0.0023	-0.0013	-5.50e-7
	(0.002)	(0.0028)	(0.002)	(0.002)	(0.002)	(0.0005)
(Aggregate TRI)^2	-6.99e-9	-1.80e-8	-2.24e-8	-2.23e-8	5.59e-9	-2.53e-9
	(2.06e-8)	(2.42e-8)	(1.69e-8)	(1.69e-8)	(1.89e-8)	(4.33e-9)
Non-Reporter Controls	Y	Y	Y	N	Y	Y
Mean PM_{10} ($\mu g/m^3$)	Y	Y	N	N	Y	Y
Mean Ozone (ppm)	Y	Y	N	N	Y	Y
County Income Controls	Y	N	N	N	Y	Y
Parental Characteristics	Y	N	N	N	Y	Y
State -Year Indicators	Y	Y	Y	Y	Y	Y
County Fixed Effects	Y	Y	Y	Y	Y	Y
Observations	4520	4698	42617	43124	4520	4520
Adjusted R-squared	0.7908	0.7858	0.4118	0.4149	0.7549	0.2498

State-level clustered 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 for gestational period > 20 weeks and is weighted by total number of pregnancies in each county and year.

TABLE 5. ESTIMATED EFFECTS OF TRI CONCENTRATIONS BY POLLUTION MEDIUM

Variable	Internal Infant Deaths				Fetal Deaths	External Deaths
	Restricted Sample (includes PM_{10}, ozone)		Full Sample (without PM_{10}, ozone)			
TRI Air (lbs/sq.mile)	0.0250**	0.0309**	0.0214**	0.0213***	-0.0032	0.00047
	(0.0111)	(0.0131)	(0.0072)	(0.0072)	(0.0085)	(0.0016)
$(TRI\ Air)^2$	-1.11e-6*	-1.17e-6*	-5.77e-7**	-5.75e-7**	1.56e-7	-9.79e-10
	(6.01e-7)	(5.98e-7)	(2.52e-7)	(2.52e-7)	(4.25e-7)	(1.31e-7)
TRI Water (lbs/sq.mile)	0.0353**	0.0516**	0.0111	0.0110	0.0078	-0.0046
	(0.1056)	(0.0240)	(0.0113)	(0.0112)	(0.0201)	(0.0033)
$(TRI\ Water)^2$	-4.65e-6**	-6.87e-6**	-1.48e-7	-1.46e-7	-1.98e-6	6.51e-7
	(1.99e-6)	(3.13e-6)	(1.67e-7)	(1.67e-7)	(2.51e-6)	(4.21e-7)
TRI Land (lbs/sq.mile)	-0.0022	-0.0023	-0.0017	-0.0017	-0.0009	0.00002
	(0.0023)	(0.0024)	(0.0013)	(0.0014)	(0.002)	(0.0005)
$(TRI\ Land)^2$	1.92e-9	2.08e-8	1.69e-8	1.68e-8	1.62e-9	-2.91e-9
	(1.973-8)	(2.09e-8)	(1.22e-8)	(1.22e-8)	(2.00e-8)	(4.96e-9)
Non-Reporter Controls	Y	Y	Y	N	Y	Y
Mean PM_{10} ($\mu g/m^3$)	Y	Y	N	N	Y	Y
Mean Ozone (ppm)	Y	Y	N	N	Y	Y
County Income Controls	Y	N	N	N	Y	Y
Parental Characteristics	Y	N	N	N	Y	Y
State -Year Indicators	Y	Y	Y	Y	Y	Y
County Fixed Effects	Y	Y	Y	Y	Y	Y
Observations	4520	4698	42617	43124	4520	4520
Adjusted R-squared	0.7924	0.7888	0.4127	0.4158	0.7547	0.2496

State-level clustered 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 for gestational period > 20 weeks and is weighted by total number of pregnancies in each county and year.

TABLE 6. ESTIMATED EFFECTS OF TRI CONCENTRATIONS BY POLLUTION CATEGORY AND MEDIUM

Variable	Internal Infant Deaths				Fetal Deaths	External Deaths
	Restricted Sample (includes PM_{10}, ozone)		Full Sample (without PM_{10}, ozone)			
TRI Carcinogenic Air (lbs/sq.mile)	0.2942*	0.4572***	0.4854***	0.4828***	-0.0243	-0.0134
	(0.1490)	(0.1457)	(0.1661)	(0.1668)	(0.2467)	(0.0587)
(TRI Carcinogenic Air)^2	-0.0003**	-0.0005***	-0.0006***	-0.0006***	0.0003	-0.00002
	(0.0001)	(0.0001)	(0.0002)	(0.00001)	(0.00025)	(0.0001)
TRI Dev/Rep Air (lbs/sq.mile)	0.0010	0.01467	0.00800	0.00793	-0.04489	0.00268
	(0.0488)	(0.0553)	(0.0528)	(0.0533)	(0.0440)	(0.00857)
(TRI Dev/Rep Air)^2	1.63e-6	-3.01e-8	4.58e-7	5.19e-7	1.58e-6	-1.54e-6
	(9.23e-6)	(0.00001)	(0.00001)	(0.00001)	(8.23e-6)	(1.60e-6)
TRI Residual Air (lbs/sq.mile)	0.0234**	0.0289**	0.0204***	0.0203***	-0.00360	0.0008
	(0.0108)	(0.0134)	(0.0071)	(0.0071)	(0.00744)	(0.0016)
(TRI Residual Air)^2	-1.08e-6*	-1.14e-6*	-5.53e-7**	-5.52e-7**	1.23e-7	-2.73e-9
	(6.27e-7)	(6.45e-7)	(2.47e-7)	(2.46e-7)	(4.18e-7)	(1.33e-7)
TRI Carcinogenic Water (lbs/sq.mile)	0.3025	0.3555	0.6464	0.6375	-0.3772*	0.0400
	(0.3732)	(0.5285)	(0.5485)	(0.5527)	(0.2182)	(0.0467)
(TRI Carcinogenic Water)^2	-0.0007	-0.0008	-0.0013	-0.0013	0.00032	-0.00004
	(0.0007)	(0.0009)	(0.0010)	(0.0010)	(0.0004)	(0.00009)
TRI Dev/Rep Water (lbs/sq.mile)	-1.0739	-1.360	-1.527**	-1.5429**	-0.0257	0.0503
	(0.9702)	(1.0010)	(0.7567)	(0.7643)	(0.5372)	(0.1181)
(TRI Dev/Rep Water)^2	0.0019	0.0024	0.0029*	0.0030*	-0.0004	-0.0002
	(0.0021)	(0.0021)	(0.0016)	(0.0016)	(0.0012)	(0.0003)
TRI Residual Water (lbs/sq.mile)	0.0389**	0.0548**	0.0098	0.0098	0.0207	-0.0055
	(0.0189)	(0.0231)	(0.0106)	(0.0106)	(0.0214)	(0.0034)
(TRI Residual Water)^2	-5.12e-6**	-7.30e-6**	-1.29e-7	-1.29e-7	-3.48e-6	7.66e-7*
	(2.34e-6)	(3.05e-6)	(1.57e-7)	(1.57e-7)	(2.73e-6)	(4.05e-7)
TRI Land (lbs/sq.mile)	-0.0020	-0.0020	-0.0013	-0.0013	-0.0009	3.67e-6
	(0.0022)	(0.0023)	(0.0014)	(0.0014)	(0.0021)	(0.0005)
(TRI Land)^2	1.76e-8	1.77e-8	1.34e-8	1.33e-8	5.79e-10	-2.67e-9
	(1.88e-8)	(1.92e-8)	(1.23e-8)	(1.23e-8)	(2.0e-8)	(5.00e-9)
Non-Reporter Controls	Y	Y	Y	N	Y	Y

Mean PM_{10} ($\mu g/m^3$)	Y	Y	N	N	Y	Y
Mean Ozone (ppm)	Y	Y	N	N	Y	Y
County Income Controls	Y	N	N	N	Y	Y
Parental Characteristics	Y	N	N	N	Y	Y
State -Year Indicators	Y	Y	Y	Y	Y	Y
County Fixed-Effects	Y	Y	Y	Y	Y	Y
Observations	4520	4698	42617	43124	4520	4520
Adjusted R-squared	0.7924	0.7894	0.4132	0.4162	0.7555	0.2495

State-level clustered 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 for gestational period > 20 weeks and 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 Change in Concentration	Estimated Elasticity	Estimated Number of Lives Saved (Lost)
TRI Air	-9.469%	0.031198	9,979
TRI Water	-12.36%	0.004109	1,716
Carcinogenic Air	-23.65%	0.002728	2,179
Non-Carcinogenic, Non-Developmental/Reproductive Air	-9.25%	0.031553	9,860
Non-Carcinogenic, Non-Developmental/Reproductive Water	-12.20%	0.004303	1,774
Mean Internal Deaths (per 100,000 live births)		770.7866	
Total Births (000,000)		31.3	

TABLE 8. ESTIMATED EFFECTS OF TRI CONCENTRATIONS BY POLLUTION MEDIUM INCLUDING LEADS AND LAGS ON INTERNAL DEATHS

Variable	September Cohort		Full Sample with Leads and Lags		
TRI Air (lbs/sq.mile)	0.0203*	0.0276**	0.02106***	0.0256***	0.0253***
	(0.0114)	(0.0105)	(0.0079)	(0.0087)	(0.0087)
$(TRI\ Air)^2$	-4.10e-07	-5.86e-07**	-7.10e-07**	-8.43e-07 **	-8.38e-07***
	3.203-07	(2.45e-07)	(3.46e-07)	(3.85e-07)	(3.85e-07)
TRI Air (lbs/sq.mile) (t+1)			-0.0023	0.00003	0.0002
			(0.0094)	(0.0103)	(0.0103)
TRI Air (lbs/sq.mile) (t-1)			-0.0015	0.0019	0.0018
			(0.004)	(0.005)	(0.005)
$(TRI\ Air)^2$: (t+1)			1.07e-07	9.49e-08	9.02e-08
			(3.56e-07)	(3.92e-07)	(3.90e-07)
$(TRI\ Air)^2$: (t-1)			-2.72e-08	-7.17e-08	-7.06e-08
			(6.36e-08)	(7.41e-08)	(7.42e-08)
TRI Water (lbs/sq.mile)	0.0305	0.0335	0.0091	0.0072	0.0072
	(0.0294)	(0.0299)	(0.0113)	(0.0110)	(0.0111)
$(TRI\ Water)^2$	-5.09e-07	-5.56e-07	-4.75e-08	-3.43e-08	-3.67e-08
	(4.16e-07)	(4.25e-07)	(1.39e-07)	(1.42e-07)	(1.42e-07)
TRI Water (lbs/sq.mile):(t+1)			0.0047	0.0079	0.0080
			(0.0097)	(0.0108)	(0.0109)
TRI Water (lbs/sq.mile):(t-1)			-0.0103	-0.008	-0.0077
			(0.0067)	(0.0069)	(0.0070)
$(TRI\ Water)^2$: (t+1)			2.20e-08	1.58e-08	1.43e-08
			(6.47e-08)	(7.80e-08)	(7.85e-08)
$(TRI\ Water)^2$: (t-1)			2.06e-08	-1.58e-08	-1.64e-08
			(7.20e-08)	(7.48e-08)	(7.52e-08)
TRI Land (lbs/sq.mile)	-0.0034	-0.0029	-0.0020	-0.0021	-0.0021
	(0.0039)	(0.0036)	(0.0017)	(0.0017)	(0.0017)
$(TRI\ Land)^2$	6.15e-08	5.38e-08	1.57e-08	1.80e-08	1.77e-08
	(3.653-08)	(3.54e-08)	(1.55e-08)	(1.55e-08)	(1.55e-08)
TRI Land (lbs/sq.mile): (t+1)			0.0013	0.0016	0.0016

			(0.0010)	(0.0011)	(0.0011)
TRI Land (lbs/sq.mile): (t-1)			0.0006	0.001	0.0007
			(0.0007)	(0.0006)	(0.0006)
$(TRI\ Land)^2$: (t+1)			-9.09e-10	-4.64e-09	-4.85e-09
			(1.05e-08)	(1.14e-08)	(1.14e-08)
$(TRI\ Land)^2$: (t-1)			-2.17e-09	-2.62e-09	-2.59e-09*
			(1.42e-09)	(1.40e-09)	(1.40e-09)
Non-Reporter Controls	Y	Y	Y	Y	N
Mean PM_{10} ($\mu g/m^3$)	N	N	N	N	N
Mean Ozone (ppm)	N	N	N	N	N
County Income Controls	Y	N	Y	N	N
Parental Characteristics	Y	N	Y	N	N
State -Year Indicators	Y	Y	Y	Y	Y
County Fixed Effects	Y	Y	Y	Y	Y
Observations	40336	42194	35910	36537	36964
Adjusted R-squared	0.0606	0.0585	0.4017	0.3994	0.4039

State-level clustered standard errors in parentheses

* significant at 10%; ** significant at 5%; *** significant at 1%

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

FIGURE I

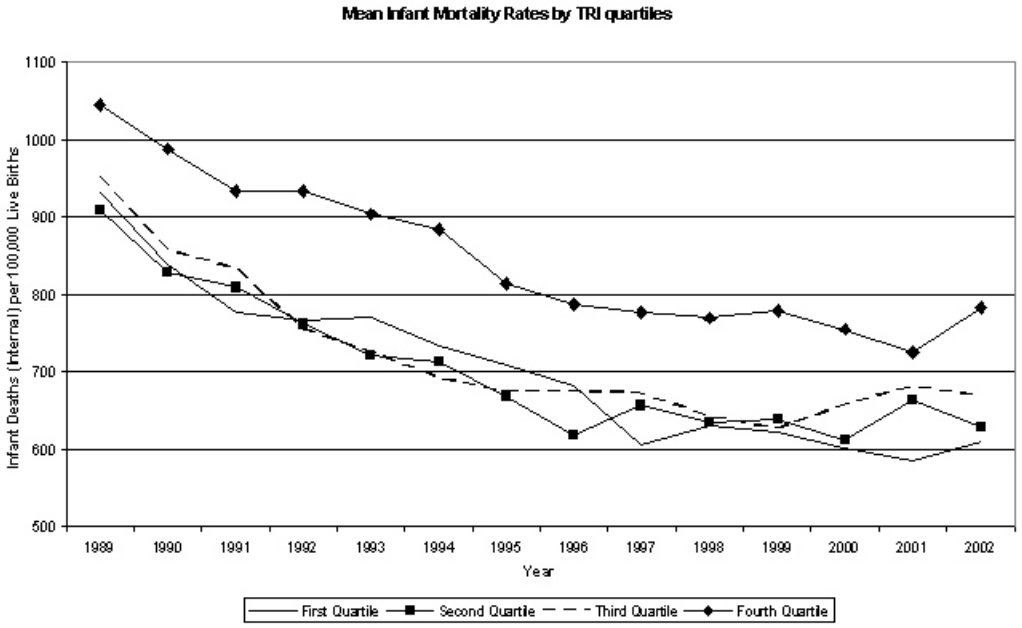

FIGURE II

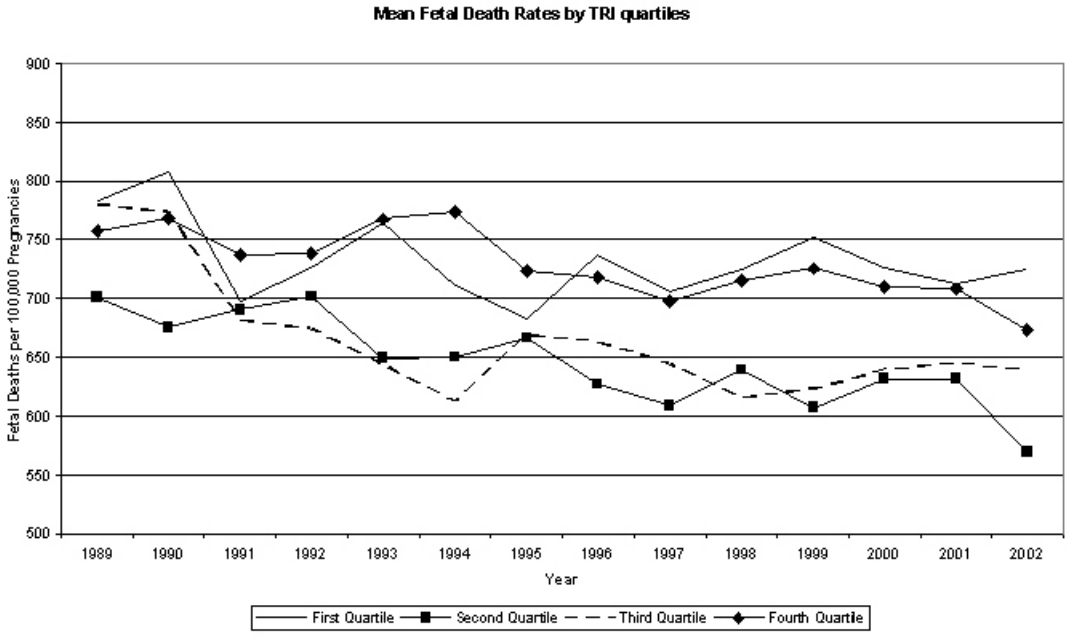

FIGURE III

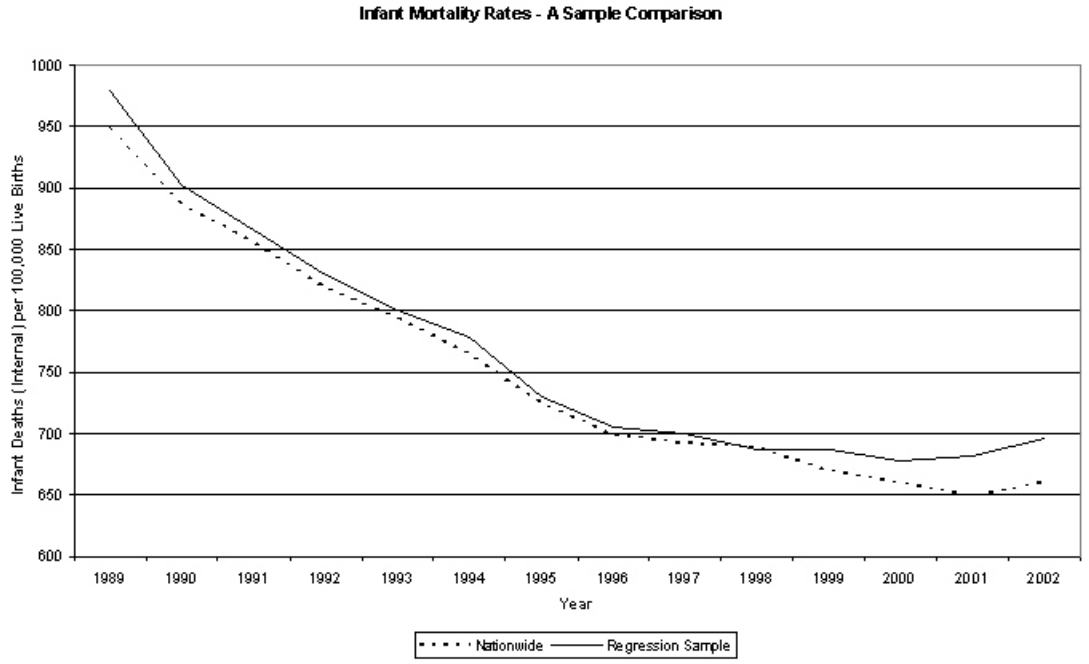

FIGURE IV

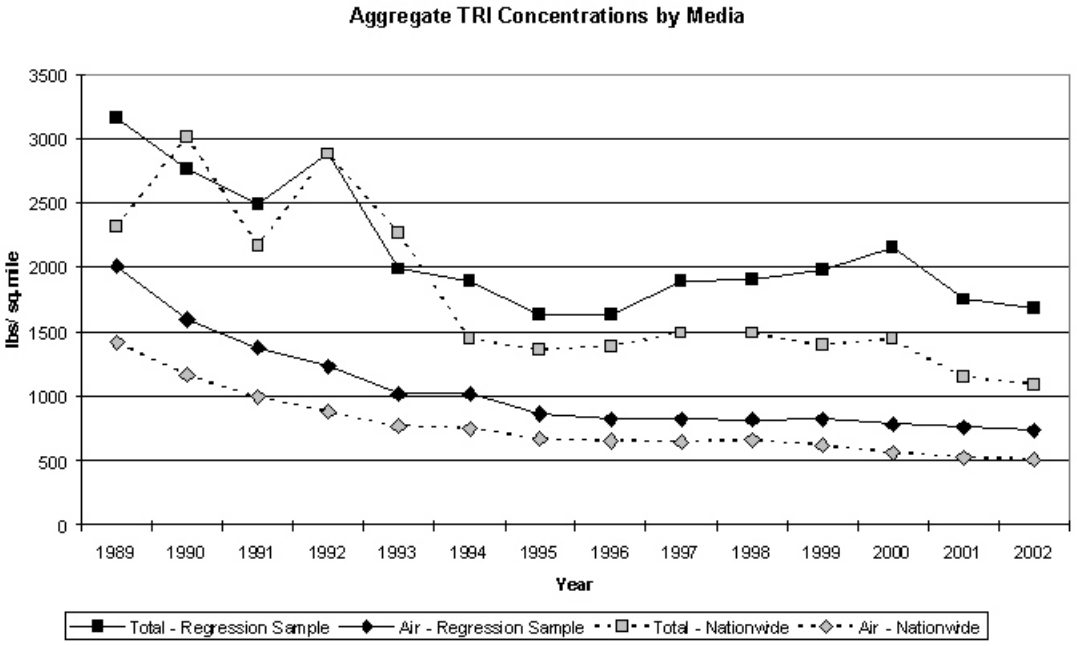

FIGURE V

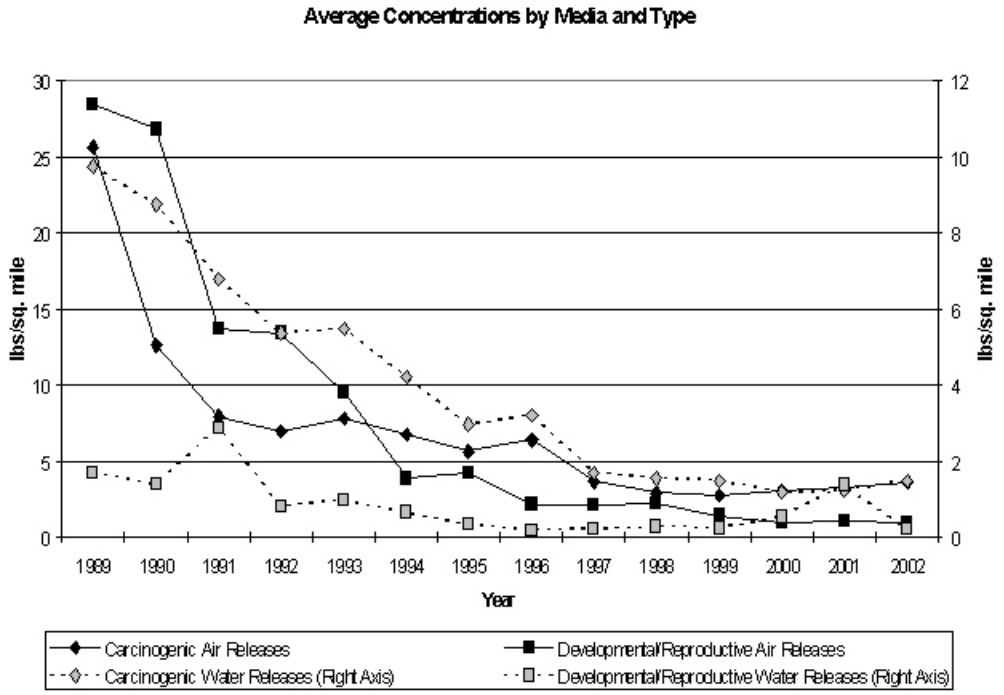

FIGURE VI