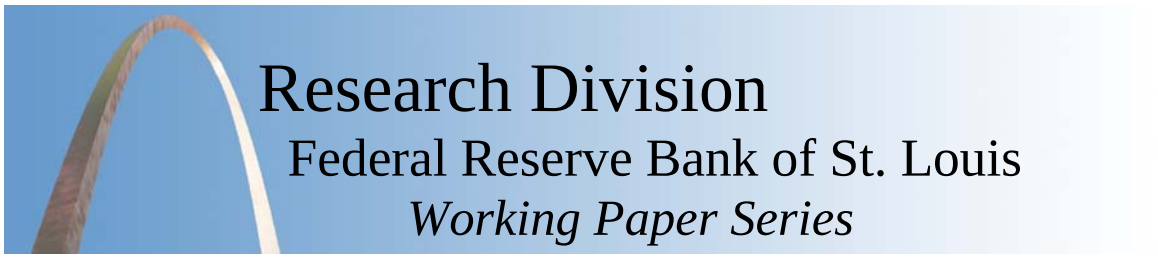

Toxic Exposure in America: Estimating Fetal and Infant Health Outcomes

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TOXIC EXPOSURE IN AMERICA:
ESTIMATING FETAL AND INFANT HEALTH OUTCOMES

ABSTRACT: We examine the effect of toxic exposure on U.S. infant and fetal mortality rates between 1989 and 2002 from toxic pollution released by facilities reporting to the Toxic Release Inventory (TRI). Unlike previous studies, we control for toxic pollution from mobile sources and from non-TRI reporting facilities. We find significant adverse effects of TRI exposure on infant mortality. There is evidence that health effects vary across media: air and water having a larger impact than land pollution. And, within air, we find that releases of carcinogens are particularly problematic for infant health outcomes. We estimate that the average county-level decreases in TRI concentrations between 1988 and 2002 saved in excess of 13,800 infant lives.

TOXIC EXPOSURE IN AMERICA:

ESTIMATING FETAL AND INFANT HEALTH OUTCOMES

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 that often arise in cross-sectional studies as a result of omitted variable bias. 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 using typical panel-data methods, such as fixed effects or first-differencing. 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 that may be attributed to both mobile sources of pollution 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 exploits the variation in toxic pollution concentrations within state-years driven by facility level response to the introduction of mandatory disclosure rules brought about by the adoption of TRI reporting requirements.

Our findings show that there are significant health consequences to infants from exposure to toxic releases, although we do not find similar outcomes for fetal health. The medium by which toxic pollutants are released into the environment plays an important role: Toxic air and water releases are significantly more harmful to infant health than land releases. Our results also suggest that of all the pollutants that we study, 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 overall 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 to ranges between $24.8B and $120B. 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, however, 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 that we conduct on the data, and in Section IX, we discuss policy implications and provide concluding remarks.

II. BACKGROUND

It is generally believed that both fetuses and infants are particularly vulnerable to exposure to toxic pollutants, although the biological mechanisms through which that occurs is 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.

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. 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.

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 4 to 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 during the 1990s.

Taking a cue from both Greenstone and Chay (2003a, 2003b) and Currie and Neidell (2005), we make use of the variation in TRI releases across location and time, induced by public disclosure of toxic pollution behavior, to identify the effects of toxic pollution on health. Our maintained assumption is that the distribution and characteristics of industries across states over time are exogenous to infant and fetal health outcomes.

To control for 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 1989-2002. Data on pregnancy outcomes are from the National Center for Health Statistics (NCHS). Data on toxic emissions are from the Toxic Release Inventory, maintained by the U. S. Environmental Protection Agency (EPA). Those two data sets are supplemented by county level data on income, job composition, transfer payments from health and unemployment benefit programs, and population, all from the U.S. Bureau of Economic Analysis. Data on land and water area are taken from the U.S. Census 2000 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

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

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. We refer to the retained observations as "internal" infant deaths.

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

Using the individual-level data described above, we compute county-level statistics based on the county of residence of the mother for infant death rates due to internal causes and death rates for fetuses with a period of gestation of more than 20 weeks. Our control variables are 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

[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.

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 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

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

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 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, linked with county-level demographic data, consists of 41,908 county-year observations. 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. This restricted sample that includes observations on (non-toxic) ambient air pollution concentrations covers 53% of the country's over-all population, 57.6% of the live births, 41.5% of aggregate TRI releases, and 39.6% of TRI on-site releases, over the sample period, and is the basis for our regression analysis.[6] Select summary statistics for this restricted data set (the "regression" sample) are presented in Tables 1 through 3, and described below. The regression sample consists of an unbalanced 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 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

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

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.[7]

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 our regression 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

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

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 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 toxic pollution on health outcomes (infant and fetal mortality) assumes that the effects of the covariates on health is linear and additive.[8] There is growing evidence, however, that suggests significant non-linearities in the effects of pollution on infant health. Because mis-specification of the functional form can lead to biased estimates, we allow for a more general specification by including quadratic terms of the toxic pollution covariates in our model.[9]

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

[9] In Section V we discuss the validity of the quadratic toxic pollution concentration term.

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 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 generalized 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 efficient method for correcting 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 efficient method is to use a model with time-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) after including time-fixed effects to control for $(\gamma_t - \overline{\gamma})$, we need to assume that $(\lambda_{it} - \overline{\lambda_i})$ is orthogonal to $(X_{it} - \overline{X_i})$. 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 GLS. Table 3 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 GLS 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. 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.[10] 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

[10] 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.

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 long as the variation in $(\lambda_{it} - \bar{\lambda}_i)$ within a state is low for each year in our sample. Our maintained assumption is that, by controlling for state-time interaction effects we have eliminated most sources of potential bias from our model.

An examination of the correlation between the TRI release statistics and covariates, Z_{it} and W_{it} indicate that the correlation between the levels of TRI pollution and most parental and county characteristics is low, as is the correlation with criteria air pollution concentrations (see Table 2, 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 may be used to test this directly.)

VI: Data and Econometric Issues

Toxic Pollution Concentrations

The estimating model, described in (5), assumes that measurements of concentrations of toxic pollution at the county level are available. Virtually no data exist, however, on toxic pollution

concentrations as such. So, in contrast with studies on criteria air pollutants where monitoring stations can provide concentration data, we must estimate toxic pollution concentrations for our study.

It is widely believed that the two principle sources of toxic pollution are manufacturing activities, and mobile sources. That is our maintained assumption.[11] 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 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

[11] 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.

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.[12] 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 results only for on-site releases, although our results are robust to using aggregate TRI 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, 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 overall contribution they make to toxic pollution releases within a county or to what extent they

[12] 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).

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.[13]

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. Overall, 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 (post 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 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,

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

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 we have 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 GLS estimates.

The second sort of measurement error of concern 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 (post 20 weeks) rates per 100,000 live births or 100,000 unterminated pregnancies from estimating the county-level fixed-effects model described in (5).[14] 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 full regression model includes TRI concentrations and TRI concentrations squared,[15] 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 possibility of health effects caused directly by

[14] Note that the 4 largest TRI concentration observations in our data set are treated as outliers and have been excluded from the analysis.

[15] Tests of significance on the level and quadratic term for the TRI concentration variables show joint significance in all models, except where indicated.

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.[16]

Aggregate TRI Releases

We present the results from our estimation of the health effects of aggregate TRI concentrations in Table 4. The full regression model for infant health is presented in column 1 with variations on the full model presented in the following columns. The final column presents the results for fetal health outcomes.

From the full model, 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 (column 2), county-level income variables (column 3), criteria air pollution concentrations (column 4), and non-reporting toxic concentration proxies (column 5), but are remarkably stable in magnitude across all specifications.

Although we do not report these estimates here, we also do not find any statistically significant results 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).

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

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, however, 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.[17]

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 Air and Water and Land

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 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.

In Table 5, we report estimates based on TRI concentrations partitioned by air, water, and "land," where land denotes simply 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

[17] 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.

infant, but not fetal, mortality rates. Toxic releases into the land do not appear to affect either infant or fetal mortality.

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 taken over our 14 year sample, this suggests that the decline in toxic air concentrations between 1989 and 2002 saved over 9,979 infant lives. 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 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 coefficient estimates across various model specifications to be robust in magnitude for toxic air concentrations, 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 a sample selection bias associated with county-level characteristics associated with having air monitoring stations for both PM_{10} and ozone. Another

possible explanation is that toxic water, and possibly also land concentrations, are not as well measured as toxic air concentrations using our methodology. If this is the case, the attenuation bias may be 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 affect 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 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 regression results are not driven by spurious correlation, outliers, or sample selection. Here, we discuss some

of the tests for robustness that we conducted.

The most significant loss of data was due to the small number of county-year observations for which we have PM_{10} and ozone concentration data. Although we believe that it is appropriate to include these measures because (1) they may affect infant and fetal mortality rates and (2) they proxy for toxic releases from non-manufacturing sources (e.g., mobile sources of pollution), we re-estimated all regressions excluding those variables (see column 4 in Tables 4 through 6). In doing so, the total number of county-year observations that may be included in the regressions increases from 4,520 to 42,617. (Note that these regressions also exclude parental characteristics and county income information.) The coefficient estimates in these regressions are of the same sign, basic magnitude, and general significance level for aggregate TRI concentrations, aggregate TRI air concentrations, and disaggregated TRI air concentrations. This leads us to be confident that there is no sample selection bias that is driving the results for these variables.

That is not necessarily the case, however, with TRI water concentrations (both in aggregate and disaggregated form), where we find that the significance level changes with the exclusion of mean county-level PM_{10} and ozone concentration levels. This may suggest that the criteria air pollutant variable is controlling for some important omitted variable that is correlated with toxic water releases or that there is something unique about the larger counties for which we have criteria air pollution monitoring stations that leads to a stronger health effect between toxic water pollution and infant mortality.

There are also concerns with the accuracy of TRI reported releases in the early years of reporting, as well as with the quality of the infant birth and death files for small counties. As a check on these potential problems, we make use of the linked birth-death records for infants that exist for

the years 1996 through 2001. The linked birth-death files exclude all births and deaths that cannot be linked because of low data quality. On average in a given year, over 95% of all infant death records are linked with the corresponding birth certificate. The public use linked files contain information on infant births and deaths for all counties with populations greater than 250,000. This data set consists of a balanced panel of 199 counties, accounting for approximately 58% of all live births in the country (8.12 million births of 14 million, nationwide, from 1996 through 2001). Using this much smaller and more restricted data set, our basic regression results remain robust.

We must also be concerned 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 not provided here, but are available upon request. In all cases, we find no statistically significant results on any of our TRI variables.

As a check for omitted variable bias associated with failing to control for non-time varying fixed-effects that are not captured directly through county fixed effects, we also compare our regression results with those estimated using a first-difference model. Results are of the same sign and general order of magnitude using aggregate TRI concentrations, and TRI concentrations broken down by medium, albeit the coefficient estimates are somewhat smaller, which is not unexpected given that, with measurement error, we would expect the attenuation bias to be exacerbated.

The coefficient estimates, however, differed significantly for the more disaggregated model.

No conclusions can be drawn from this result, though, as the regressors in this particular first-difference model failed to pass the exogeneity test, so the estimators are known to be biased.

Finally, as previously noted, we exclude a small number (4) of outliers from our regression analysis. To ensure robustness over our sample, 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 with increases in toxic concentrations, which would be obscured by looking only at aggregate TRI releases because of heterogeneity in health effects across pollution media and chemical categories. We find that infants are more sensitive to air-borne and water-borne concentrations of toxins than to landborne concentrations, over-all, and that they are particularly vulnerable to carcinogens. Between 1989 and 2002, we estimate that the decline in county-level TRI concentrations in the manufacturing sector saved over 13,800 infant lives, at an estimated value of between $25B and $120B. It is important to note, however, that the above number of lives saved may be significantly under-estimated. 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-makers are 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 is needed also 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.

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

SELECT REFERENCES

Ashenfelter, Orley, and Michael Greenstone. (2004) "Using Mandated Speed Limits to Measure the Value of a Statistical Life." *Journal of Political Economy*, Vol. 112(1) pp. S226-67.

Baker, D. B., S. Greenland, J. Mendlein, P. Harmon. (1988). "A Health Study of Two Communities Near the Stringfellow Waste Disposal Site. *Archives of Environmental Health.* Vol. 43:325-334.

Bove, Frank J., Mark C. Fulcomer, Judith B. Klotz, Jorge Esmart, Ellen M. Dufficy, Jonathan E. Savrin. (1995). "Public Drinking Water Contamination and Birth Outcomes." *American Journal of Epidemiology*. Vol. 141, No. 9.

Carpenter, D.O. (1994). "How Hazardous Wastes Affect Human Health." *Central European Journal of Public Health.* 2(supple):6-9.

Choi, Hannah, Youn K. Shim, Wendy E. Kaye, P. Barry Ryan. (2006). "Potential Residential Exposure to Toxic Release Inventory Chemicals during Pregnancy and Childhood Brain Cancer." *Environmental Health Perspectives* Vol.114(7):1113-1118.

Croen, L.A., G.M. Shaw, L.Sanbonmatsu, S. Selvin, P. Buffler. (1997). "Maternal Residential Proximity to Hazardous Waste Sites and Risk for Selected Congenital Malformations." *Epidemiology*. Vol. 8:347-354.

Currie, Janet, and Jonathan Gruber. (1996) "Saving Babies: The Efficacy and Cost of Recent Expansions of Medicaid Eligibility for Pregnant Women." *The Journal of Political Economy*. December.

Currie, Janet, and Matthew Neidell. (2005). "Air Pollution and Infant Health: What Can We Learn from California's Recent Experience?" *Quarterly Journal of Economics*. Vol. 120(3):1003-30.

Fell, Deshayne, Linda Dodds, Will King. (2004). "Residential Mobility During Pregnancy." *Pediatric and Perinatal Epidemiology*. Vol. 18:408-414.

Fielder, H. M., C.M. Poon-King, S. R. Palmer, N. Moss, G. Coleman. (2000). "Assessment of Impact on Health of Residents Living Near the Nant-y-Gwyddon Landfill Site: Retrospective Analysis." *British Medical Journal*. Vol. 320:19-22.

Grandjean, P., P.J. Landrigan. (2006). "Developmental Neurotoxicity of Industrial Chemicals." *The Lancet*. Vol. 368.

Greenstone, Michael, Kenneth Chay. (2003a) "The Impact of Air Pollution on Infant Mortality:

Evidence from Geographic Variation in Pollution Shocks Induced by a Recession." *Quarterly Journal of Economics*. Vol. 118(3), 1121-67.

Greenstone, Michael, Kenneth Chay. (2003b) "Air Quality, Infant Mortality, and the Clean Air Act of 1970." National Bureau of Economics Working Paper #9844.

Hyslop, Dean R. And Guido W. Imbens. (2000) "Bias from Classical and Other Forms of Measurement Error," NBER Technical Working Paper Series WP 0257.

Kahn, Matthew. (2004) "Domestic Pollution Havens: Evidence from Cancer Deaths in Border Counties." *Journal of Urban Economics*, July 2004, Vol. 56(1):51-69.

Kharrazi, M., B.J. Von, M. Smith, T. Lomas, M. Armstrong, R. Broadwin, et al. (1997). "A Community-Based Study of Adverse Pregnancy Outcomes Near A Large Hazardous Waste Landfill in California." *Toxicology and Industrial Health*. Vol. 13:299-310.

Khoury, M.J, W. Stewart, A. Weinstein, S. Panny, P. Lindsay, M. Eisenberg. (1988). "Residential Mobility During Pregnancy: Implications for Environmental Teratogenesis." *Journal of Clinical Epidemiology*. Vol. 41:15-20.

Landrigan, P., C. Kimmel, A. Correa, B. Eskenazi. (2004). "Children's Health and the Environment: Public Health Issues and Challenges for Risk Assessment." *Environmental Health Perspectives.* Vol. 112(2): 257-265.

Landrigan, P., W. Suk, R. Amler. (1999). "Chemical Wastes, Children's Health, and the Superfund Basic Research Program." *Environmental Health Perspectives.* Vol. 107:423-427.

Lave, Lester, Eugene Seskin. (1977). *Air Pollution and Human Health.* Resources for the Future, Washington, D.C.

Marshall, E.G., L.J. Gensburg, D. A. Deres, N.S. Geary, M.R. Cayo. (1997). "Maternal Residential Exposure to Hazardous Wastes and Risk of Central Nervous System and Musculoskeletal Birth Defects." *Archives of Environmental Health.* Vol. 52:416-425.

Mueller, B., C. Kuehn, C.K. Shapiro-Mendoza, K.M. Tomashek. (2007). "Fetal Deaths and Proximity to Hazardous Waste Sites in Washington." *Environmental Health Perspectives.* Vol. 115, #5.

Needham, L., Dana Barr, S.P. Caudill, J.L. Pirkle, W.E. Turner, J. Osterloh, R.L. Jones, E.J. Sampson. (2005). "Concentrations of Environmental Chemicals Associated with Neuro-developmental Effects in U.S. Population." *Neurotoxicology.* 26(4):531-45.

Neumann, C.M., D.L. Foreman, J.E. Rothlein. (1998). "Hazard Screening of Chemical Releases

and Environmental Equity Analysis of Populations Proximate to Toxic Release Inventory Facilities in Oregon." *Environmental Health Perspectives.* Vol. 106:217-216.

Nickerson, Krista. (2006). "Environmental Contaminants in Breast Milk." *Journal of Midwifery and Women's Health.* Vol. 51, No. 1, January/February.

Shaw, G.M. L.H. Malcoe. (1992). "Residential Mobility During Pregnancy for Mothers of Infants With or Without Congenital Cardiac Anomalies: a reprint." *Archives of Environmental Health.* Vol. 47:236-238.

Sosniak, W.A., W. E. Kaye, T.M. Gomez. (1994). "Data Linkage to Explore the Risk of Low Birth Weight Associated with Maternal Proximity to Hazardous Waste Sites from the National Priorities List." *Archives of Environmental Health.* Vol 49:251-255.

Zender, R., A. M. Bachand, J.S. Reif. (2001). "Exposure to Tap Water During Pregnancy." *Journal of Exposure Analysis and Environmental Epidemiology*. Vol. 11:224-230.

TABLE 1: DESCRIPTIVE STATISTICS FOR REGRESSION SAMPLE

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 Regression 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							
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 (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 (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 (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 (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) (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 FOR REGRESSION SAMPLE, 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 Regression 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							
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 (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 (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 (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 (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) (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. CORRELATIONS OF TOXIC RELEASE CONCENTRATIONS WITH PARENT DEMOGRAPHICS AND COUNTY-LEVEL CONTROLS

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 3. WITHIN STATE-TIME VARIATION FOR SELECT VARIABLES

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 4. ESTIMATED EFFECTS OF AGGREGATE TRI CONCENTRATIONS ON INFANT AND FETAL MORTALITY RATES

Variable	**Internal Infant Deaths**					**Fetal Deaths**
TRI Concentrations (lbs/sq.mile)	0.0006	0.0012	0.0018	0.0023	0.0023	-0.0013
	(0.002)	(0.003)	(0.0028)	(0.002)	(0.002)	(0.002)
(TRI Concentrations)^2	-6.99e-9	-1.17e-8	-1.80e-8	-2.24e-8	-2.23e-8	5.59e-9
	(2.06e-8)	(2.13e-8)	(2.42e-8)	(1.69e-8)	(1.69e-8)	(1.89e-8)
Non-Reporter Controls	Y	Y	Y	Y	N	Y
Mean PM_{10} ($\mu g/m^3$)	Y	Y	Y	N	N	Y
Mean Ozone (ppm)	Y	Y	Y	N	N	Y
County Income Controls	Y	Y	N	N	N	Y
Parental Characteristics	Y	N	N	N	N	Y
State -Year Indicators	Y	Y	Y	Y	Y	Y
County Fixed Effects	Y	Y	Y	Y	Y	Y
Observations	4520	4698	4698	42617	43124	4520
Adjusted R-squared	0.7908	0.7882	0.7858	0.4118	0.4149	0.7549

Robust standard errors in parentheses

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

Note: Internal mortality rates are per 100,000 births and fetal mortality rates are per 100,000 pregnancies. Internal infant mortality regressions are weighted by total number of births in each county and year. Fetal mortality regression is 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 ON INFANT AND FETAL MORTALITY RATES BY POLLUTION MEDIUM

Variable	Internal Infant Deaths					Fetal Deaths
TRI Air Concentrations (lbs/sq.mile)	0.0250**	0.0269**	0.0309**	0.0214**	0.0213***	-0.0032
	(0.0111)	(0.0234)	(0.0131)	(0.0072)	(0.0072)	(0.0085)
(TRI Air Concentrations)^2	-1.11e-6*	-1.06e-6*	-1.17e-6*	-5.77e-7**	-5.75e-7**	1.56e-7
	(6.01e-7)	(5.60e-7)	(5.98e-7)	(2.52e-7)	(2.52e-7)	(4.25e-7)
TRI Water Concentrations (lbs/sq.mile)	0.0352**	0.0480**	0.0516**	0.0111	0.0110	0.0078
	(0.0156)	(0.0228)	(0.0240)	(0.0113)	(0.0112)	(0.0201)
(TRI Water Concentrations)^2	-4.64e-6**	-6.35e-06**	-6.87e-6**	-1.48e-7	-1.46e-7	-1.98e-6
	(1.99e-6)	(3.01e-6)	(3.13e-6)	(1.67e-7)	(1.67e-7)	(2.51e-6)
TRI Land Concentrations (lbs/sq.mile)	-0.0022	-0.0023	-0.0023	-0.0017	-0.0017	-0.0009
	(0.0023)	(0.0024)	(0.0024)	(0.0013)	(0.0014)	(0.002)
(TRI Land Concentrations)^2	1.92e-8	2.13e-8	2.08e-8	1.69e-8	1.68e-8	1.62e-9
	(1.97e-8)	(2.04e-8)	(2.09e-8)	(1.22e-8)	(1.22e-8)	(2.00e-8)
Non-Reporter Controls	Y	Y	Y	Y	N	Y
Mean PM_{10} ($\mu g/m^3$)	Y	Y	Y	N	N	Y
Mean Ozone (ppm)	Y	Y	Y	N	N	Y
County Income Controls	Y	Y	N	N	N	Y
Parental Characteristics	Y	N	N	N	N	Y
State -Year Indicators	Y	Y	Y	Y	Y	Y
County Fixed Effects	Y	Y	Y	Y	Y	Y
Observations	4520	4698	4698	42617	43124	4520
Adjusted R-squared	0.7924	0.7905	0.7888	0.4127	0.4158	0.7547

Robust standard errors in parentheses

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

Note: Internal mortality rates are per 100,000 births and fetal mortality rates are per 100,000 pregnancies. Internal infant mortality regressions are weighted by total number of births in each county and year. Fetal mortality regression is 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 ON INFANT AND FETAL MORTALITY RATES BY POLLUTION CATEGORY AND MEDIUM

Variable	Internal Infant Deaths					Fetal Deaths
TRI Carcinogenic Air Concentrations (lbs/sq.mile)	0.2942*	0.3732***	0.4572***	0.4854***	0.4828***	-0.0243
	(0.1490)	(0.1349)	(0.1457)	(0.1661)	(0.1668)	(0.2467)
(TRI Carcinogenic Air Concentrations)^2	-0.0003**	-0.0004***	-0.0005***	-0.0006***	-0.0006***	0.0003
	(0.0001)	(0.0001)	(0.0001)	(0.0002)	(0.00001)	(0.00025)
TRI Developmental/Reproductive Air Concentrations (lbs/sq.mile)	0.0010	0.0055	0.01467	0.00800	0.00793	-0.04489
	(0.0488)	(0.0506)	(0.0553)	(0.0528)	(0.0533)	(0.0440)
(TRI Developmental/Reproductive Air Concentrations)^2	1.63e-6	1.35e-6	-3.01e-8	4.58e-7	5.19e-7	1.58e-6
	(9.23e-6)	(0.00001)	(0.00001)	(0.00001)	(0.00001)	(8.23e-6)
TRI Residual Air Concentrations (lbs/sq.mile)	0.0234**	0.0254**	0.0289**	0.0204***	0.0203***	-0.00360
	(0.0108)	(0.0113)	(0.0134)	(0.0071)	(0.0071)	(0.00744)
(TRI Residual Air Concentrations)^2	-1.08e-6*	-1.04e-6*	-1.14e-6*	-5.53e-7**	-5.52e-7**	1.23e-7
	(6.27e-7)	(6.01e-7)	(6.45e-7)	(2.47e-7)	(2.46e-7)	(4.18e-7)
TRI Carcinogenic Water Concentrations (lbs/sq.mile)	0.3025	0.3762	0.3555	0.6464	0.6375	-0.3772*
	(0.3732)	(0.5112)	(0.5285)	(0.5485)	(0.5527)	(0.2182)
(TRI Carcinogenic Water Concentrations)^2	-0.0007	-0.0009	-0.0008	-0.0013	-0.0013	0.00032
	(0.0007)	(0.0009)	(0.0009)	(0.0010)	(0.0010)	(0.0004)
TRI Developmental/Reproductive Water Concentrations (lbs/sq.mile)	-1.0739	-1.343	-1.360	-1.527**	-1.5429**	-0.0257
	(0.9702)	(0.9907)	(1.0010)	(0.7567)	(0.7643)	(0.5372)
(TRI Developmental/Reproductive Water Concentrations)^2	0.0019	0.0024	0.0024	0.0029*	0.0030*	-0.0004
	(0.0021)	(0.0021)	(0.0021)	(0.0016)	(0.0016)	(0.0012)
TRI Residual Water Concentrations (lbs/sq.mile)	0.0389**	0.0515**	0.0548**	0.0098	0.0098	0.0207
	(0.0189)	(0.0222)	(0.0231)	(0.0106)	(0.0106)	(0.0214)
(TRI Residual Water Concentrations)^2	-5.12e-6**	-6.79e-6**	-7.30e-6**	-1.29e-7	-1.29e-7	-3.48e-6
	(2.34e-6)	(2.93e-6)	(3.05e-6)	(1.57e-7)	(1.57e-7)	(2.73e-6)
TRI Land Concentrations (lbs/sq.mile)	-0.0020	-0.0021	-0.0020	-0.0013	-0.0013	-0.0009
	(0.0022)	(0.0023)	(0.0023)	(0.0014)	(0.0014)	(0.0021)
(TRI Land Concentrations)^2	1.76e-8	1.90e-8	1.77e-8	1.34e-8	1.33e-8	5.79e-10
	(1.88e-8)	(1.90e-8)	(1.92e-8)	(1.23e-8)	(1.23e-8)	(2.0e-8)
Non-Reporter Controls	Y	Y	Y	Y	N	Y
Mean PM_{10} ($\mu g/m^3$)	Y	Y	Y	N	N	Y

Mean Ozone (ppm)	Y	Y	Y	N	N	Y
County Income Controls	Y	Y	N	N	N	Y
Parental Characteristics	Y	N	N	N	N	Y
State -Year Indicators	Y	Y	Y	Y	Y	Y
County Fixed-Effects	Y	Y	Y	Y	Y	Y
Observations	4520	4698	4698	42617	43124	4520
Adjusted R-squared	0.7924	0.7909	0.7894	0.4132	0.4162	0.7555

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

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

FIGURE I

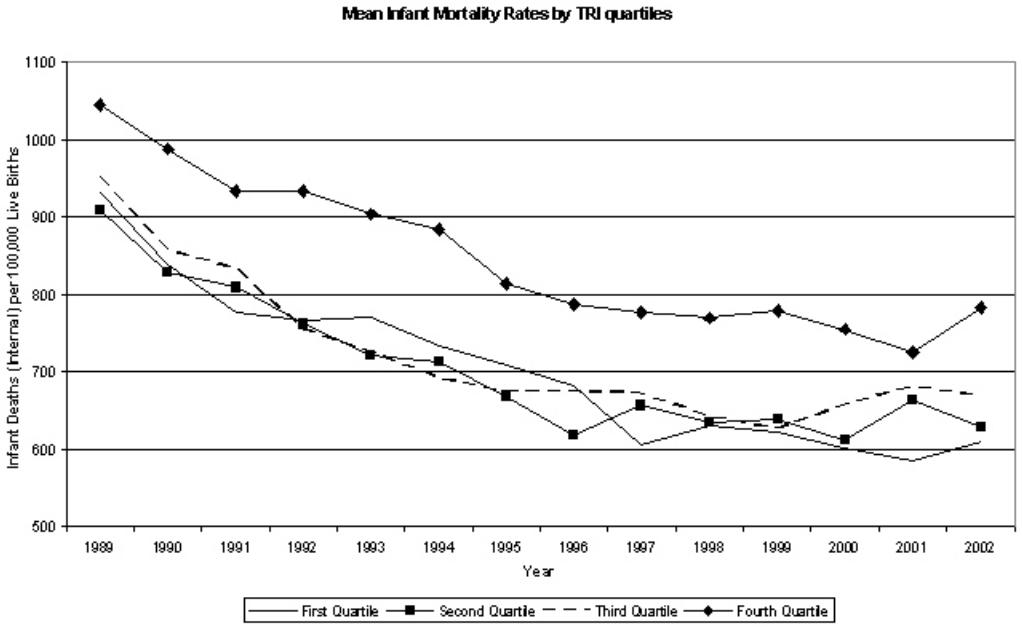

FIGURE II

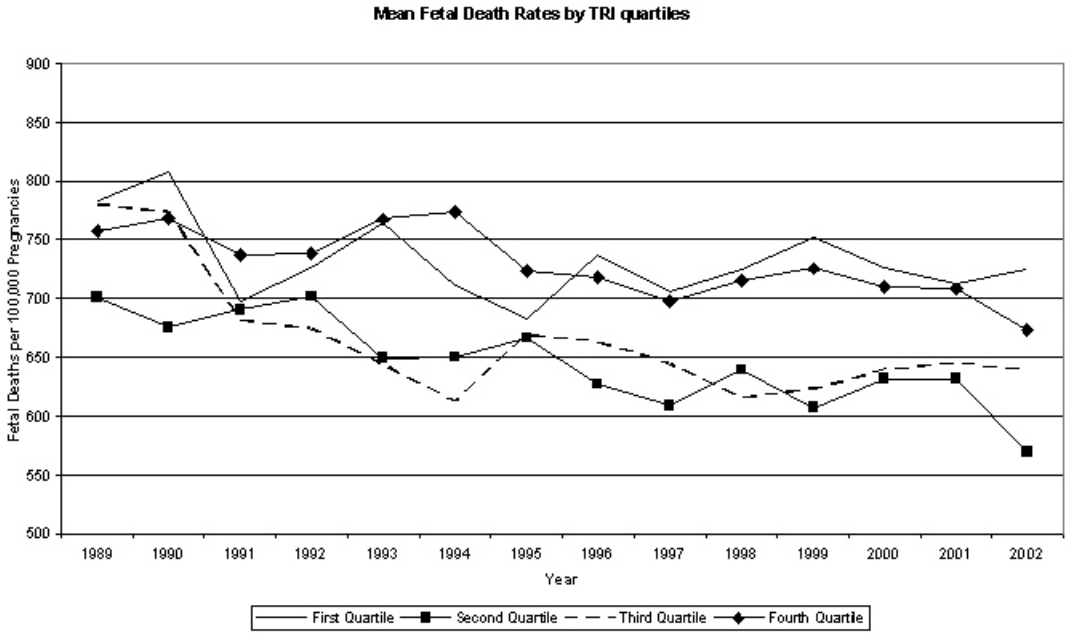

FIGURE III

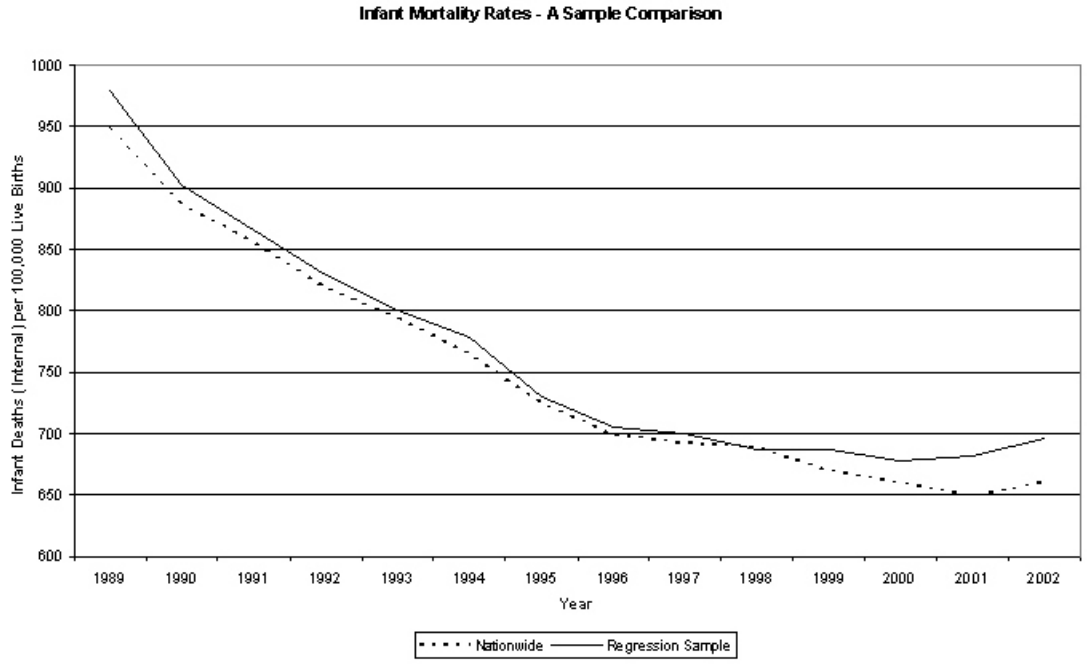

FIGURE IV

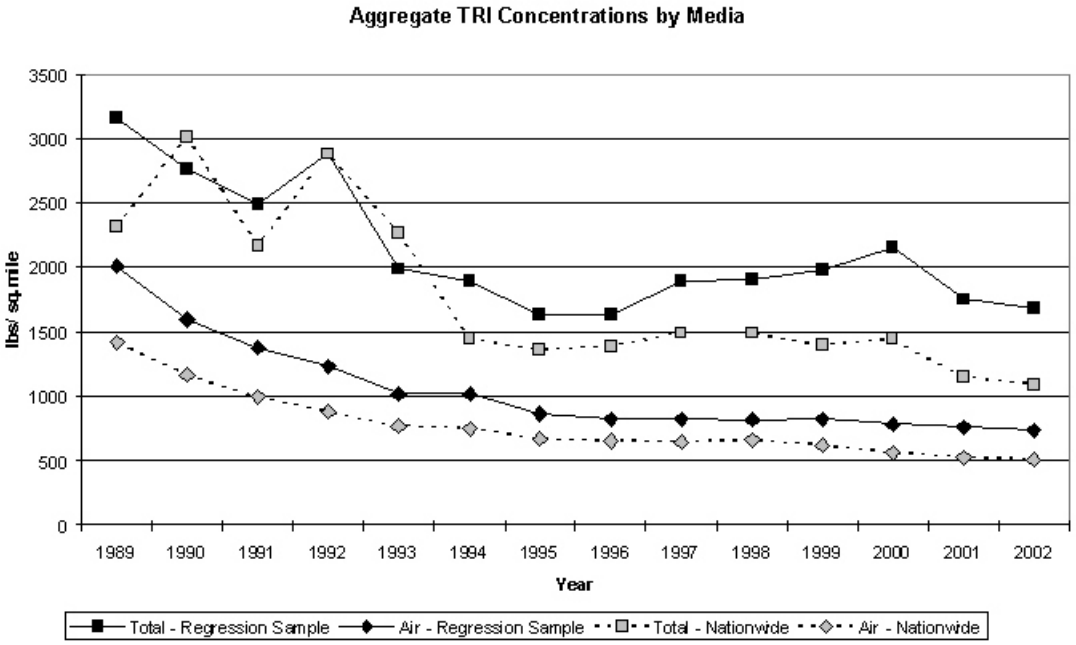

FIGURE V

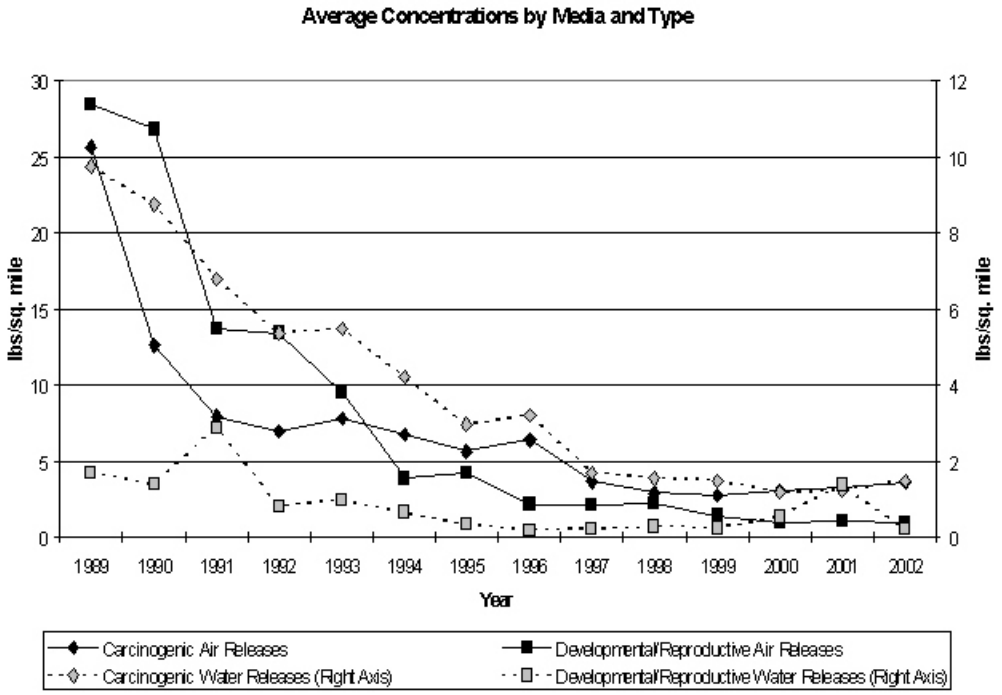

FIGURE VI