Regulatory Impact: The Rise and Fall of Arsenic

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Abstract

What is the status of the first generation of pollutants in light of extensive government regulation since the 1970s? To answer this question, this paper takes stock of one such first generation industrial pollutant, arsenic. As a recognized carcinogen, arsenic is astutely toxic at certain levels and has been shown to increase the risk of lung and bladder cancers and to cause environmental damage. At the same time, while restricted in its use, arsenic has been an important input in the manufacturing of wood products, semiconductors, glass, paints, dyes, and pesticides. Commercial use of arsenic in the U.S. has ebbed and flowed in the past century, reaching peaks in the 1940s and 1990s. In the past decade, however, arsenic consumption has fallen to levels not seen since the 1920s.

I take advantage of an extensive time series to identify the impact of federal regulations on the industrial consumption of arsenic, which trends arsenic releases very well for available data from EPA's Toxics Release Inventory (1998-2008). Thus, arsenic use serves as a proxy for arsenic releases by industrial facilities. I conduct three separate analyses, the first of which is a time series analysis of total arsenic use where I control for all potential determinants of the fall and rise of arsenic use between 1929 and 2007. The second analysis is a panel analysis of arsenic consumption by end users between 1975 and 2004. The third analysis examines the causal effect of the 2001 EPA ruling on a more stringent standard for arsenic in drinking water and the 2004 voluntary ban on the use of chromated copper arsenate by the wood products industry to account for the sharp decline in arsenic use/emissions over the past decade.

Preliminary results show that, on the whole, significant events in the political economy—World War II and the voluntary ban on the use of arsenic in residential construction—have had more impact than government regulation on the ebb and flow of arsenic consumption over the past half century. With this said, selected environmental legislations, such as the Safe Drinking Water Act of 1972 and a related federal action—the 2001 EPA ruling—have accomplished what they set out to do, which is to limit contaminants in drinking water sources.

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Introduction

Since the passage of the Occupational and Safety Health Act of 1970, toxic substances have faced cradle-to-grave regulation in the U.S.: Their storage, handling, transportation, and disposal are all regulated by the U.S. federal government. What is the status of the first generation of pollutants in light of extensive government regulation since the 1970s? This paper attempts to answer the question by taking stock of one such first generation industrial pollutant, arsenic (including arsenic compounds), which is a target chemical in several federal as well as state legislations, a mainstay chemical on the EPA's list of banned or severely restricted pesticides, and the number one listed chemical for toxicity on the EPA's priority list of hazardous substances.[1]

Commercial use of arsenic in the U.S. has ebbed and flowed in the past century, reaching peaks in the 1940s and 1990s. In the past decade, however, arsenic consumption has fallen to levels not seen since the 1920s. Has the extensive and leviathan regulation of the past half century accounted for these trends? Or have instead the developments in the arsenic using sectors and economic structural changes in the U.S. underscored these trends?

As a recognized carcinogen, arsenic is astutely toxic at certain levels and has been shown to increase the risk of skin, liver, lung, and bladder cancers and to cause environmental damage.[2] Inorganic arsenic is generally more toxic than organic arsenic. However, animal studies have shown that organic arsenic (e.g., methyl and phenyl arsenates) can produce health effects similar to those produced by inorganic arsenic (e.g., chromated copper arsenate). At the same time, while restricted in its use, arsenic has been an important input in the manufacturing of wood

[1] Arsenic is also one of nine chemicals that are judged to be the most toxic to human and animal health according to the CRC Handbook of Chemistry and Physics (2008). Moreover, arsenic is listed as a "controlled" chemical by the Basel Convention on the Transboundary Movement of Hazardous Waste.
[2] Source: http://www.atsdr.cdc.gov/tfacts2.html#bookmark01.

products, semiconductors, glass, paints, dyes, and pesticides. Until recently, 85 to 90 percent of arsenic has been used by the wood products industry to protect wood from rotting due to insects and microbial agents.[3] Meanwhile, the semiconductor industry uses arsenic as a key dopant or impurity element to alter the optical/electrical proprieties of silicon for the manufacturing of electronics and as a basic building block for high-end computers and lasers. Arsenic derivatives are used as pesticides in agriculture.

In the paper, I take advantage of an extensive time series and industry panels from the U.S. Geological Survey (USGS) to identify the impact of federal regulations on the industrial consumption of arsenic, which trends arsenic releases very well for available data from the EPA's Toxics Release Inventory (TRI) (1998-2007).[4] Industrial use serves as a proxy for arsenic releases into the environment by industrial facilities. While arsenic consumption imperfectly predicts human and animal exposure (although exhibiting a similar time trend), an advantage of using arsenic consumption data is that I am able to exploit the *total* amount of industrial use rather than just the industrial releases by firms that report to the EPA.

I conduct three separate analyses, the first of which is a time series analysis of total arsenic use where I include a large set of covariates to control for potentially confounding effects of the fall and rise of arsenic use between 1929 and 2007—World War II, recessions, media attention, U.S. industrial development, and major developments in arsenic using industries—to isolate the effect of government regulation on arsenic use. In my second analysis, I conduct a panel analysis of arsenic consumption by end users between 1975 and 2004 to better understand cross sector variation of regulatory outcomes. My third analysis attempts to uncover the causal

[3] Since the mid-1930s, chromated copper arsenate (CAA) has been used as a wood preservative. Until 2004, CCA has been used widely in residential construction to treat timber.
[4] Because of additions of new sectors in the reporting of toxic emissions in 1998, there was a break in the data between 1997 and 1998. Thus, I restrict my analysis to the stable set of TRI emissions data for arsenic after 1998.

effect of two events— the 2001 EPA ruling on a more stringent standard for arsenic in drinking water and the voluntary ban on the use of chromated copper arsenate by the wood products industry—to account for the sharp decline in arsenic use/emissions over the past decade.

While my analyses are not definitive in uncovering the *causal* impact of federal regulation on arsenic use/emissions it appears to be the first attempt in the empirical literature to consider how well federal legislations have fared in time series data that capture the industrial use of toxic chemicals *before* and *after* the passage of federal regulations on toxic chemical use. Thus, I am able to isolate the effect of government regulation since I have arsenic consumption data prior to the advent of the first major regulation on toxic chemicals. This paper offers a descriptive analysis with insights into regulatory impact over time and across arsenic using sectors.

My findings in these analyses show that on the whole federal regulation has had minimal impact on arsenic use/emissions over the past half century. Significant events in the political economy—such as World War II and the voluntary ban on the use of arsenic in residential construction by the wood products industry, the largest arsenic using sector—have had more impact than government regulation on the ebb and flow of arsenic consumption over the years. With this said, selected environmental legislations, such as the Safe Drinking Water Act of 1972 and a related federal action—the 2001 EPA ruling—have accomplished what they set out to do, which is to limit contaminants in drinking water sources.

The rest of the paper is organized as follows. In Section II, I provide a brief summary of the literature. I discuss data sources that are used in the study and my methodology in section III; descriptive statistics are also presented. In Section VI, I present my results. In section V, I provide concluding remarks and next steps.

I. Background and Theory

Studies have linked toxic chemical exposure to health outcomes, including several recent studies on the impact of toxic exposure on fetuses and infants (Agarwal et al. 2009, Currie and Schmieder 2008). This line of research not only suggests that people are at risk from toxic releases but establishes a direct relationship between environmental releases of toxic substances and health impact.[5] With respect to arsenic exposure in particular epidemiological studies have shown that arsenic is a carcinogen that increases the risk of skin cancer and cancer in the liver, bladder, and lungs (ATSDR 2007).

In light of these studies, my paper considers an earlier chain in the causal linkage. I ask: how has extensive federal regulation since the 1970s account for industrial use/emissions of arsenic over the past half century? In this section, I provide a brief review of the literature on government regulation and the legislative history of "social" regulation in the U.S. I also offer hypotheses on the direction of effects of the federal legislations that have regulated arsenic and other toxic chemicals.

Joskow et al. (1994) and May (2002) define "social" regulation as regulation that controls polluting by-products of production, sets health and safety standards for products and workplaces, restricts the content of information provided by sellers through advertising and other means of describing products to consumers, and establishes requirements to protect buyers from fraudulent, discriminatory, or incompetent behavior by sellers.[6] These policies potentially affect

[5] Because it is difficult to draw a relationship between a disease such as cancer and toxic exposures in a particular location given that cancer develops over a long period and people are mobile toxic exposure studies have exploited the use of infant health outcomes to see if existing environmental releases have detectable negative effects for human health. Birth outcomes are ideal because they are likely to be highly affected by conditions during the brief interval of pregnancy, all else equal.

[6] By contrast, "economic" regulation is aimed at ensuring competitive markets for goods and services and at avoiding consumer and other harms when such markets are not feasible. This is generally accomplished through regulating prices and/or conditions for firms entering specific markets The line between economic and social

prices, costs, product quality, the dynamics of business competition, and the allocation of resources in the economy.

Throughout most of U.S. history, state and local governments have had primary responsibility for health-and-safety regulation, including environmental protection, but since 1970 the federal government has played an increasingly important role in environmental regulation (Revesz 2001). Congress's first major effort came in 1969 with the passage of the National Environmental Policy Act, which laid out broad environmental goals and required federal agencies to assess the environmental impacts of their programmatic actions (Klyza and Sousa 2008). From 1970 through 1990, Congress passed and the President signed seven major federal social regulatory statutes that pertained to arsenic and other toxic chemicals in the industrial manufacturing process. These legislations include the Occupational and Safety Health Act (OHSA) of 1970, the Clean Water Act of 1972, the Safe Drinking Water Act (SDWA) of 1974, the Resource Conservation and Recovery Act (RCRA) of 1976, the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) (otherwise known as the Superfund Act) of 1980, the Emergency Planning and Community Right-to-Know Act of 1986 (which established the Toxics Release Inventory), and the Clear Air Act of 1990. The federal government has also taken several additional regulatory actions on arsenic: the 1993 EPA decision to ban the use of arsenic acid for treating cotton crops and the 2001 adoption by the EPA of a more stringent arsenic limit for drinking water. See Table 1 in the Appendix for a description of each of these regulatory actions.

The bulk of these statues are command-and-control regulation, with the exception of the Emergency Planning and Community Right-to-Know Act 1986 and the creation of sulfur dioxide

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regulation becomes blurred when economic regulation is used to achieve social goals and when economic instruments are used as part of social regulation (May 2002).

(SO2) emissions markets in the Clean Air Act of 1990 (the latter of which does not directly pertain to arsenic). In fact, the command-and-control approach has been the predominant form of US environmental regulation over the past several decades (Case 2001, Esty 2004). Economic theory maintains this leviathan approach is an efficient mechanism if firms are homogenous with respect to pollution abatement costs, and if government can correctly identify the source(s) of the problem, adequately supply regulations which compel firms to reduce pollution, and effectively monitor and enforce compliance (Revesz and Stavin 2007). Because such assumptions about firm and government capacities do not always hold across policy issue areas, the leviathan form of regulation has often generated high direct costs (Bui and Mayer 2003).

Since the mid 1980s, during an era of increased devolution of federal to state regulatory responsibilities, new regulatory tools have been proposed to substitute or supplement command-and-control forms of regulation (Fiorino 1999, Coglianese and Nash 2001).[7] More recent regulation has moved toward market-based methods and other more indirect and flexible approaches (Bae et al. 2008, Revesz and Stavin 2007). These include market-based instruments such as cap and trade systems (Tietenberg 2006) and pollution charges, market-friction reductions, and government subsidy reductions (Revesz 1997),[8] information-based regulation such as the Toxics Release Inventory Program (Konar and Cohen 1997), which is a byproduct of the Emergency Planning and Community Right-to-Know Act 1986, and voluntary programs (Prakash and Potoski 2006, Kotchen, M. and Klaas van 't Veld. 2009).

[7] The Reagan administration established a stringent (on paper at least) system of regulatory review that required more extensive cost-benefit tests of new regulations. In 1995, in response to concerns voiced by state and local officials about federal mandates, Congress enacted the Unfunded Mandates Reform Action. This put in place procedures for assessing the impacts of major federal mandates on other levels of government and the private sector (May 2002).

[8] Market-based instruments encourage behavior through market signals; these regulation "harness market forces," because if they are well designed and properly implemented, they encourage firms or individuals to undertake pollution control efforts that are in their own interests and that collectively meet policy goals.

Notwithstanding, by and large first generation pollutants such as arsenic and other toxic chemicals continue to be regulated under traditional command-and-control legislations enacted several decades ago. These laws have granted wide discretion to the EPA to set tolerance levels for various pollutants and limits on contaminants allowed in drinking water, and to create a system of strict joint-and-several liability for parties responsible for abandoned hazardous waste sites, among other explicit directives regarding pollution control levels or methods (Revesz and Stavin 2007).

The mainstay rationale for prescriptive policies as described is based on externalities, which dates back to Pigou (1920). An externality implies that actors such as firms do not fully internalize the costs and benefits of their actions. Government command-and-control regulations are designed to compel firms and other actors to internalize the negative externalities of their actions. In particular, Pigou had proposed that the government should impose a tax on emissions equal to the cost of the related damages at the efficient level of control.

In his critical response to the Pigovian perspective, Coase (1960) attributes externality problems to the absence of property rights. Coase conceptualizes pollution as a property rights problem: if one were to allocate property rights to the atmosphere, for example, pollution problems might be solved by more efficiently by private bargaining, instead of governmental intervention.

The Coase Theorem states that in a bargaining environment without transaction costs, parties will reach socially desirable agreements and that the overall amount of pollution will be independent of the legal rules (i.e., the assignment of property rights) chosen to structure their relationship. Thus, regardless of the initial legal rule, bargaining will produce two results: (1) it will lead to the same amount of pollution; and (2) it will lead to the maximization of social

welfare.[9] The Coase Theorem has served as a fundamental basis for market-based environmental policies.

The Coase Theorem is said to hold if there are no transaction costs, no wealth or income effects, private rather than public goods, and no third-party impacts (i.e., all affected parties participate in the negotiation). At least some of these conditions are unlikely to hold in the case of most environmental problems (Hanley et al. 1997). Hence, private negotiation will not—in general—fully internalize environmental externalities. And when market transactions—including Coasian bargaining—do not generate socially efficient allocations of resources, government regulation may be necessary to improve environmental quality.

On the other hand, although government regulation may be necessary to improve environmental quality when market transactions fail to generate socially efficient allocations of resources, such regulation is by no means sufficient to improve welfare or even environmental quality. This is because government regulation itself may not be efficient, that is, government may under-regulate or over-regulate, and/or it may regulate in ways that require unnecessarily large costs of compliance (Keohane et al. 1998, Revesz and Stavin 2007).

As such, it is an empirical question whether command-and-control regulations in general and the set of laws that have regulated arsenic and other toxic chemicals in particular, have *in fact* realized what they have aimed to achieve—the reduction of emissions into the environment through stringent restrictions on the storage, handling, transportation, and disposal of the toxic substances in question. How have regulatory actions by the federal government on arsenic affected the industrial use/emissions of arsenic over time? I propose the following null and alternative hypotheses.

[9] Of course, the choice of legal rules can determine which party makes payments and which party receives them, a distributional concern, though not one of efficiency.

H_0: The enactment of the seven federal environmental statutes and the EPA's 1993 ban on arsenic acid and the 2000 ruling on arsenic, ceteris paribus, have led to statistically significant reductions in arsenic use/emissions, respectively.

H_1: The abovementioned regulations and government actions either a) have had no statistically significant effect or b) have led to statistically significant increases in arsenic use/emissions, respectively.

Table 1 contains descriptions of each of the seven major federal legislations and the EPA's arsenic ruling in 2000. The third column of Table 1 summarizes my hypothesis regarding each of the regulation's effect, ceteris paribus, on the industrial consumption/emissions of arsenic.

[Insert Table 1]

II. Data and Methods

I combine data from various sources to construct (1) a time series dataset of industrial consumption of arsenic at the national level for the period 1929 to 2007 and (2) a panel dataset of industrial consumption of arsenic across arsenic using sectors for the period 1975 to 2004.
In this section I describe the dependent variables and the explanatory variables in turn. I conclude the section with a description of my estimation strategy and methods.

Dependent variables

The latent outcome variable of interest is the industrial emission of arsenic in the U.S. over the past century, while the observed variable is the industrial consumption of arsenic. Industrial use serves as a proxy for arsenic releases into the environment by industrial facilities over the period of my analysis (1929-2007). Consistent data for arsenic emissions from the EPA's Toxic Release Inventory (TRI) are restricted to 1998 and onwards,[10] a decade in which only one new regulatory action on arsenic—the EPA's limit on arsenic in drinking water in 2000—was instituted. To isolate the effect of the extensive government regulation on arsenic since the 1970s I need historical data. The U.S. Geological Survey (USGS) houses data on the production, trade, and industrial use of over 80 mineral commodities in the U.S. dating back to 1900. On a limited basis (across time and scope), the USGS also keeps end-use data on arsenic and other mineral commodities. I exploit this extensive time series for my analysis.

Figure 1 plots arsenic use and releases between 1998 and 2008. Notably the two series trend each other considerably well; the two series exhibit a Pearson's correlation coefficient, r, of about 0.90. Measured in metric tons, releases are on average ninefold larger than consumption figures on an annual basis. This may be because releases account for past as well as current consumption; this may also be why while the two series move together, contemporaneous arsenic use lags arsenic emissions by a year or two.

[Insert Figure 1]

[10] Data on toxic emissions of arsenic are available from the EPA's TRI starting in 1989; however, because of the addition of new industry sectors there is a break in the data in 1998. Thus, I restrict my analysis to the stable set of TRI emissions data for arsenic after 1998. Starting in the 1998 reporting year, seven new industry sectors, including metal mining, coal mining, electric utilities, chemical wholesale distributors, petroleum bulk storage/terminals, hazardous waste management facilities and solvent recovery facilities, were required to report to the EPA. These industries are excluded from the dataset covering years prior to 1998.

A major drawback with the USGS data, however, is that it measures chemical use rather than releases into the environment, the latter of which have been shown in the literature to have caused detrimental effects on human health, despite the similar time trends exhibited by the two series. With that said, a key advantage of using arsenic consumption data other than the extended history is that I am able to exploit the *total* amount of industrial use rather than just the industrial releases by firms that report to the EPA to identify the effects of federal regulatory action on arsenic.[11] While the chemical releases of facilities that are small enough not to have to report to the EPA are negligible, researchers have shown evidence of systematic underreporting of toxic releases by facilities to the EPA due to changes in plant operations or lax enforcement, among other factors (Brehm and Hamilton 1996, Natan and Miller 1998, Poje and Horowitz 1990).

Thus, given the advantages I describe above and the similar trends exhibited by the two series over the past decade I make the assumption in my analysis (see section IV) that the historical series of arsenic use, of which there are no companion toxic release data, are appropriate proxy measures for arsenic emissions into the environment.

The USGS arsenic use data are calculated figures, according to the general formula: Production + Imports - Exports ± (Stock Change). Domestic arsenic production in the U.S. ceased after 1985. Thus, 1986 and onwards industrial consumption has come from imports. By 2006, 86% of all arsenic metal imports in the U.S. come from China; Japan is the next largest source of arsenic metal (13%).[12] Imports of arsenic trioxide come from China (63%), Morocco

[11] According to the EPA,"Facilities must report release and other waste management information pursuant to EPCRA Section 313 if they: (1) have 10 or more full-time employees or the equivalent; (2) are in a covered NAICS code; and (3) exceed any one threshold for manufacturing (including importing), processing, or otherwise using a toxic chemical listed in 40 CFR Section 372.65. Reporting thresholds is at 10,000 lbs (annually) for most chemicals.

[12] Source: http://minerals.usgs.gov/minerals/pubs/myb.html.

(25%), Hong Kong (4%), Chile (3%) and other countries (5%). Exports and stocks make up negligible shares of arsenic use in the U.S.

Figure 2 depicts historical trends of the commercial use of arsenic in the U.S. Of note, the time series is a faintly inverted U shape, reminiscent of the environmental Kuznets curve. Commercial use of arsenic in the U.S. has ebbed and flowed in the past century, reaching peaks in the 1940s and 1990s. In the past decade, however, arsenic consumption has fallen to levels not seen since the 1920s.

[Insert Figure 2]

Data between 1975 and 2004 are available for the following arsenic end-use sectors: agricultural chemicals, glass, nonferrous alloys and electronics, pressure treated wood, and other industrial uses. USGS estimates end-use distributions based on apparent demand information collected from industry contacts and Internet sources.[13] End-use figures sum up to total use figures. Figure 3 Panel A and Panel B depict the time trends of these various end-use sectors.

[Insert Figure 3]

Until 2004, 85 to 90 percent of industrial consumption of arsenic had occurred in the wood products industry where chromated copper arsenate (CAA) was used as a wood preservative to treat timber for residential and industrial construction. There was an upward

[13] This information is provided in an email exchange on May 11, 2009 by Dr. William E. Brooks, the commodity specialist at the USGS for Silver, Mercury, and Arsenic.

trend in arsenic use in wood products until the late 1990s; since then arsenic use has fallen. By 2004, the wood products industry accounted for 65 percent of total arsenic use. The next largest end-user of arsenic is the agriculture sector where arsenic is a chemical input in the production of pesticides: in particular, insecticides, herbicides, algaecides, and growth stimulants for plants and animals. Panel A of Figure 3 depicts a clear decline of arsenic use in the agriculture industry between 1978 and 2004. Agricultural use made up 12.5 percent of total industrial consumption of arsenic in 2004.

By contrast, there is a slight upward trend in arsenic use in the semiconductor industry despite a sharp dip and recovery in the mid 1990s as depicted by Panel B. Arsine gas (AsH_3) is used as a dopant in the production of semiconductors, and gallium arsenide (GaAs) is used in integral components of discrete microwave devices, lasers, light-emitting diodes, photoelectric chemical cells, and semiconductor devices. Arsenic is also an industrial input for clarifying glass and ceramics. Arsenic use has been relatively flat in the glass sector over the last three decades.

Explanatory variables

The main explanatory variables of interests are the major federal environmental legislations and related policy actions (*reg_OHSA, reg_SDWA, reg_RCRA, reg_Superfund, reg_TRI, reg_CAA, reg_As, reg_AsAcid*) that have regulated arsenic since 1970s. Table 1 of Section II presents a brief description of each of these regulations. The regulation variables are created as indicator variables whereby each variable is coded as 1 starting in the year the legislation or action is enacted. As I hypothesize (see section I), if government regulation has had a large effect on arsenic use/emission, as is intended by the laws, there should be a

statistically significant *negative* relationship between the dependent variable and the key explanatory variables of interest, ceteris paribus.

To isolate the impact of federal regulation on toxic releases, I include a vector of covariates as controls—these are other factors that might affect arsenic use/emissions, thus confounding the effect of government action. For both the time series and panel analyses, categories of time-varying factors that appear as explanatory variables in the regression model include: economic factors and key developments in arsenic using sectors. In the time series analysis, I also include a variable that proxy for public awareness, which might affect the use/releases of arsenic over time. In the panel analysis, time-invariant factors are controlled by sector-specific fixed effects. I also include time fixed effects to account for time varying factors that happen in a given year that affect all arsenic using sectors not already controlled for by the other variables. Finally, all explanatory variables are lagged in order to link the impacts of the explanatory variables to arsenic use/release levels for subsequent years.

With respect to economic variables, a measure of U.S. industry structure (i*ndstructure*) is included as an explanatory variable because some studies (notably Bradford et al. 2000) have shown evidence of the environmental Kuznets curve (EKC)—the proposition that there is an inverse relationship between income level and pollution—for arsenic and some selected pollutants.[14] Additionally, arsenic's slightly U shaped time trends (see Figure 1) suggest controlling for industry stricture is warranted. If the EKC exists, industrial structure should exert a *negative* influence on the use/release of arsenic over time in both the time series and panel analyses. I construct a measure of industrial structure to be

$$indstructure_t = \alpha_0 + \alpha_2 (\ln gdpc)_t + \alpha_2 (\ln gdpc)_t^{\,2} + \alpha_3 \ln pop + \alpha_4 (\ln pop)_t^2 + \ln gdpc_t * \ln pop_t$$ in

[14] Bradford et al. (2000) find some evidence of the environmental Kuznets curve for arsenic, COD, dissolved oxygen, lead and SO2, while finding less evidence in the cases of PM and some other measures of pollution.

the time series analysis, where *gdpc* is GDP per capita from the Bureau of Economic Analysis and *pop* is population data from the Bureau of the Census. In the panel analysis, industrial structure is measured as $indstructure_{it} = \alpha_0 + \alpha_2 (\ln rvalue)_{it} + \alpha_2 (\ln rvalue_{it})^2$ with *rvalues* as a measure of the "value added" by arsenic end-use sectors in 2000 U.S. dollars from the Bureau of Economic Analysis.

Moreover, in my analyses I control for the ebb and flow of the business cycle by including a binary indicator variable which is unity if there is a recession in a particular year (*recession*).[15] The recession variable is in both the time series and panel data analyses. In the time series analysis I also include a vector of prices that measure the unit value per metric ton of arsenic in 2000 U.S. dollars (*lnprice00*). I expect prices to be positively correlated with arsenic use/emissions. Price data are estimated by the USGS using the market price in U.S. dollars per ton of arsenic trioxide.[16]

Another set of covariates that potentially confound the impact of federal regulatory actions are the major developments in arsenic using sectors as well as elsewhere in the political economy that could affect the use/emission of arsenic. The first of these factors is World War II (*WWII*) in the time series analysis; an indicator variable accounts for the presence of WWII in 1940-45. The second Great War was a period of tremendous economic growth in the U.S., as the

[15] NBER designated recessions that are relevant to this study include August 1929-March 1933; May 1937-June 1938; February 1945-October 1945; November 1948-October 1949; July 1953-May 1954; August 1957-April 1958; April 1960-February 1961; December 1969-Nobember 1970; November 1973-March 1975; January 1980-July 1980; July 1981-November 1982; July 1990-March 1991; and March 2001-November 2001. Since my unit of analysis is on an annual basis I translate the recession dates into annual data. My decision rule is a year would be designated a recession year if a recession officially begins in January up through August of that year. A recession that begins after August of a given year would not count as a recession year but the year after would be designated a recession year. Moreover, if a recession ends through March that year in which it ends would not be counted as a recession year. Recession years are as follow: 1929, 1931, 1930, 1931, 1932, 1933, 1937, 1938, 1945, 1949, 1953, 1954, 1957, 1958, 1960, 1970, 1974, 1980, 1981, 1982, 1990, 2001, and 2008.

16 The market price of arsenic trioxide was converted to a value for arsenic stock by dividing the arsenic trioxide price by the percentage of arsenic contained in arsenic trioxide (75.7 percent). Source: USGS.

nation's manufacturing sectors produced for the war efforts. Therefore, I expect World War II to exert a positive influence on arsenic use/emissions.

Over the half past century, there have been several major developments in the arsenic using sectors that have impacted arsenic use/emissions. The first of these was the introduction of chromated copper arsenate-C formulation (*CAA_C*), the most widely used form of CAA in the U.S. in 1938; CAA has been a key input in the production of pesticides as well as wood preservatives for residential and industrial construction. Another key development is the discovery of gallium arsenide (*GaAs*) in the semiconductor industry in 1986. In 1986, Morris Young, a physicist at Lawrence Livermore National Laboratory discovered how to make gallium arsenide crystals (hydrogen and arsenic), which have replaced the silicon crystals as a basic building block for high-end computers and lasers. I create indicator variables to account for both of these developments; I expect both variables to be positively correlated with arsenic use/emissions since the demand for arsenic necessarily increases as the demand for products that contain arsenic as key inputs increases.

The third major development in the arsenic using sectors is a voluntary ban (*voluntaryban*) by the wood products industry—the sector that accounts for the largest share of arsenic use—to phase-out and discontinue the use of CAA to treat lumber products for residential construction. As of December 31, 2003, no further sales of CCA lumber were allowed for residential purposes in the U.S.[17] I expect this indicator variable to be negatively correlated with arsenic use; in fact, I suspect the voluntary ban plays a large role in the 67 percent drop in arsenic use between 2003 and 2008.

[17] Canada's wood products industry followed the U.S.'s lead in the same year. The European Union (EU)'s ban on arsenic in wood preservative took effect on June 30, 2004. Of note, Germany and Sweden had both independently banned the sales of CAA-treated wood in 1975 and 1993. Australia followed with a ban in 2005.

Another factor that could potentially shape the ebb and flow of arsenic use over the time periods investigated is the change in public attention regarding arsenic use/emissions over time. For example, an increase in public attention regarding the adverse effect of arsenic on human health and the environment could lead to increased collective action by the American populace to oppose the use of arsenic for industrial purposes, which could lead to reductions in arsenic use and the switch to substitutes given enough public pressures. I proxy for public attention in the time series by including a variable which is a count of the number of news/media reports (*media*) published by the *New York Times* about the adverse effect of arsenic between 1939 and 2007.

Table 2 presents descriptive statistics of the vector of covariates for both the time series and panel data analyses. Panel A presents the mean, min, and max values of the explanatory variables included in the time series analysis. Panel B presents the same statistics for the covariates in the panel data analysis.

[Insert Table 2]

In the panel data analysis, I control for *time invariant*, sector specific unmeasured factors that could affect the values of the covariates but have no direct bearing on arsenic use/emissions. I also include year-specific fixed effects to account for such factors as changes in the regulatory environment, such as monitoring and sanctioning capacities of the government, which are not already accounted for in the set of regulatory variables. Other time variant factors include the increase in the awareness of health hazards associated with arsenic over time.

Estimation strategy and methods

I conduct three separate analyses to isolate the effect of extensive government action on the industrial use/emissions of arsenic over the past century. The first analysis is a time series specification that details an historical account of the arsenic use/emissions in the U.S. between 1929 and 2007. The second analysis is a panel data analysis (1975-2007) using a fixed effects model. In the third analysis, I focus on arsenic emissions over the past decade (1998-2007), a period in which arsenic use/emissions have fallen from their peak to levels not seen since 1920s, to estimate the causal effect of a government and an industry initiated decisions. For the third analysis, lead serves as the control/comparison chemical in a difference-in-difference specification.[18]

Time series and panel data specifications

My empirical strategy in the time series and panel data analyses is to control for as many "natural" causes of the ebb and flow of arsenic use/emission as possible, and search for the effects of government regulation in the parameter estimates. With respect to the covariates, as described above I painstakingly account for major developments in arsenic using industries, including commercial innovations and other developments that may increase or decrease the use of arsenic as an input in the production process, which could confound the effect of government regulation. I also control for U.S. industrial development and other economic as well as noneconomic factors that could obscure the impact of environmental legislations on arsenic use/emissions. I search for the effect of government regulation using variation over time (arsenic

[18] Here I use toxic emissions data from the EPA's TRI for both arsenic and lead since I am only investigating the previous decade worth of data. The third analysis is currently least developed. Constructive comments and suggestions welcomed.

consumption has ebbed and flowed over the years) and across sectors (the use of arsenic as an input varies across sectors).

Equation (1) represents the time series specification, where *t* denotes year:

$$\begin{aligned}\ln arsenic_t = {} & \beta_0 + \beta_1 reg\_OHSA_{t-1} + \beta_2 reg\_CWA_{t-1} + \beta_3 reg\_SDWA_{t-1} + \\ & \beta_4 reg\_RCRA_{t-1} + \beta_5 reg\_\sup erfund_{t-1} + \beta_6 reg\_TRI_{t-1} + \beta_7 reg\_CAA_{t-1} + \\ & \beta_8 reg\_As_{t-1} + \beta_9 reg\_AsAcidban_{t-1} + \beta_{10} indstructure_{t-1} + \beta_{11} recession_{t-1} + \\ & \beta_{12} \ln prices00_{t-1} + \beta_{13} WWII_{t-1} + \beta_{14} CAA\_C_{t-1} + \beta_{15} GaAs_{t-1} + \beta_{16} voluntaryban_{t-1} \\ & + \beta_{17} \ln media_{t-1} + \varepsilon_t\end{aligned} \quad (1)$$

I estimate the model using the Newey West method, which is the OLS estimator with Newey West standard errors. The Newey West standard errors account for heteroskedasticity and autocorrelation up to the third lag. Tests of the AR(p) model on the error term suggest that the disturbances are correlated over the first 3 lags but not beyond that; after the first few lags the noise die down.

Equation (2) represents the fixed effects specification, where *i* indexes the industry of interest, *t* indexes year, α_i indexes sector fixed effects, and $\{T_t\}$ indexes a set of time fixed effects:

$$\begin{aligned}\ln arsenic_{it} = {} & \alpha_i + \beta_0 + \beta_1 reg\_\sup erfund_{i,t-1} + \beta_2 reg\_TRI_{i,t-1} + \\ & \beta_3 reg\_CAA_{i,t-1} + \beta_4 reg\_As_{i,t-1} + \beta_5 reg\_AsAcidban_{i,t-1} + \beta_6 indstructure_{i,t-1} + \\ & \beta_7 recession_{t-1} + \beta_8 \ln prices00_{t-1} + \beta_9 GaAs_{i,t-1} + \beta_{10} voluntaryban_{i,t-1} + \sum_t \theta_t T_t + v_{it}\end{aligned} \quad (2)$$

I fit equation (2) by using feasible generalized least squares (FGLS). FGLS allows estimation in the presence of AR(1) autocorrelation within panels and cross-sectional correlation and heteroskedasticity across panels. Given the model is correctly specified FGLS yields more efficient and correct standard errors.

Difference-in-difference specification

My empirical strategy for the third analysis is difference-in-difference specifications in which lead serves as a control chemical for arsenic, the treatment chemical. These specifications investigate the causal effect of two decisions—one government and one industry initiated actions, respectively—which I posit are responsible for the decline of arsenic use/emissions over the past decade. These two decisions are (i) the EPA's adoption in 2001 of a more stringent water quality standard that only targeted arsenic and (ii) the voluntary decision by the wood products industry in 2004 to discontinue the use of CAA in residential construction. To isolate the effect of the 2001 EPA decision, I estimate equation (3), where $EPA2001_i$ is an indicator variable for being an EPA targeted chemical for drinking water limits in 2001:

$$\ln emissions_t = \beta_0 + \beta_1 postEPA2001_t + \beta_2 EPA2001_i + \beta_3 EPA2001treat_i + \varepsilon_{it} \quad (3)$$

$postEPA2001_i$ is essentially a vector of year fixed effects, which inherently includes a dummy for the introduction date of the 2001 EPA policy. $EPA2001treat_i$ is an indicator variable that equals one for arsenic after the 2001 EPA ruling, such that β_3 measures the treatment effect of the 2001 EPA ruling.

To isolate the effect of the wood products industry's decision to voluntarily ban CAA, I estimate equation (4), where $voluntaryban_i$ is an indicator for being the chemical that observes voluntary ban:

$$\ln emissions_t = \beta_0 + \beta_1 postvoluntaryban_t + \beta_2 voluntaryvan_i + \beta_3 voluntarybantreat_i + \varepsilon_{it} \quad (4)$$

$postvoluntaryban_i$ is a vector of year fixed effects, which includes a dummy for the introduction date of the voluntary ban. $voluntarybantreat_i$ is an indicator variable that equals one

for arsenic the voluntary ban has taken effect, such that β_3 measures the treatment effect of the voluntary ban by the wood products industry.

Identification will ultimately come from the assumption that, after controlling for changes in observables and time-invariant unobserved heterogeneity between the two chemicals, emission levels would change the same over the time frame studied in the treatment and control chemicals, in the absence of the 2001 EPA decision and the 2004 voluntary ban on arsenic. However, for the preliminary analyses I have not added a vector of chemical or time-specific covariates.

Relative to other chemical substances lead is probably the best chemical to serve as a control for arsenic because like arsenic it is regulated under all the major federal environmental legislations discussed in section I. Back in the early 1990s the drinking water limit for lead was set at 15 ppb. In a sense, the EPA ruling in 2001 on arsenic catches up to (and goes beyond by 5 ppb) its ruling on lead in drinking water. Moreover, lead is the number two listed chemical for toxicity on the EPA's priority list of hazardous substances (versus number one for arsenic). Likewise, lead is one of nine chemicals, along with arsenic that are judged to be the most toxic to human and animal health according to the CRC Handbook of Chemistry and Physics (2008). Furthermore, lead like arsenic is listed as a "controlled" chemical by the Basel Convention on the Transboundary Movement of Hazardous Waste. Of course, chemically the two substances are not the same, but the two chemicals' toxicity and adverse impact on human health and the environmental are viewed under law to be similar in severity.

Aside from these qualifications, whether lead is a good control for arsenic is determined by whether the emissions trends of the two chemical substances are similar in the absence of the treatments. The treatments in theory induce a deviation from this common trend. Figure 4

depicts the historical trends of the industrial consumption of the two chemicals between 1980 and 2007. The two chemicals appear to trend each other quite well (although not perfectly) until the 1990s and early 2000s. There appears to be a break around that time and the trends diverged.

Figure 5 zooms in on the divergent trends of arsenic and lead over the past decade; the data displayed here and used in estimating equations (3) and (4) are toxic emissions data from the EPA's TRI. The 2000 EPA decision and the wood product's voluntary ban are represented by the shaded gray bars. Like arsenic, consistently measured emissions data for lead starts in 1998.

[Insert Figure 5]

III. Estimation Results

In this section I present the estimation results from the time series, fixed effects, and difference-in-difference models. Table 3 summarizes the results from estimating equation (1). The time series results show that of the seven major federal legislations and two federal actions on arsenic four of these regulations exert statistically significance influence at the five percent level ($p=0.05$) on arsenic use/emissions over the time period of the analysis. One of these legislations—the Safe Drinking Water Act (SDWA) of 1974—shows the hypothesized result of a negative coefficient on the indicator variable. This result appears to confirm the pedestrian knowledge in the environmental policy field that SDWA has been by and large effective in ensuring the quality of drinking water in the U.S. SDWA applies to every public water system in the United States. One of SDWA's main mandates is to set Maximum Contaminant Levels for a

priority set of contaminants; arsenic is one such substance. SDWA is the law that enabled the EPA to raise the stringency of allowable concentration of arsenic in drinking water in 2001.

[Insert Table 3]

What appear surprising at first is that the coefficient on the indicator variable which represents the Emergency Planning and Community Right-to-Know Act of 1986 (i.e., TRI) exhibits the "wrong" sign. According to the estimation results, the advent of TRI has led to an *increase* rather than a decrease in arsenic use/emissions. This runs counter to the theoretical intention and expectation of an information disclosure policy, which is that out of the fear of reprimand by the market firms that must public disclose their toxic emissions will be driven to improve their environmental performance. However, there has been literature that shows disclosure policies such as the TRI do not always produce the intended results, in part because of information overload from a highly complex, voluminous database. When community groups and others are not able to use the data properly they are less able to keep firms accountable (Bae et al. 2008, Dranove et al. 2003).[19]

The signs on the coefficients associated with the Clean Water Act of 1972 and the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) /Superfund of 1980 are unexpected. The results show these two laws have led to increases in arsenic use/emissions, which are not as hypothesized. These results warrant more investigation because the results are unintuitive from my general knowledge of these laws.

[19] Bae et al. (2008) show that at the state level simple dissemination of TRI data was ineffective (and even counterproductive in some instances), and that the states' data processing efforts have played a critical role in achieveing the TRI's underlying goals.

Of the other explanatory variables included in the time series specification, coefficients on World War II and the voluntary ban by the wood products sector exhibit statistical significance at the five percent level. As expected, *WWII* had a positive influence on arsenic use/emissions. The wood products industry's voluntary decision to discontinue the use of CCA led to the decline of arsenic use/emissions, which is expected, given the wood products industry's historically large share of arsenic use.

Finally, there is one explanatory variable that is statistically significant at the ten percent level. Industrial structure yields a negative relationship with arsenic use/emissions, which confirms Bradford et al. 2000's finding that arsenic, along with a few other pollutants show evidence of the environmental Kuznets curve (EKC). Figure 2 lends some visual evidence of this phenomenon.

Table 4 summarizes the results from estimating equation (2). The sector fixed effects specification shows that one federal action—the EPA's decision to ban the use of arsenic acid in cotton farming—has led to a statistically significant decline in arsenic use/emissions during the period (1974-2004) under study. No other federal regulatory action yields statistical significance. The voluntary ban by the wood products industry is negatively correlated with arsenic use/emissions; this suggests that while the ban officially took effect in 2004 users of CAA have transitioned out the chemical even before then.

Of note, a couple of the year fixed effects (e.g., 1979 and 1982) yield statistically significant positive effects on the outcome variable. From Figures 2 and 3 one can see that these were years of tremendous growth for arsenic consumption, particularly for the wood products industry, and to a lesser extent the glass and electronics sectors.

[Insert Table 4]

Table 5 Panels A and B present preliminary results from estimating equations (3) and (4). As noted, the models currently are bare boned; I have not included a vector of covariates to control for potential confounders. Consequently, the results need to be interpreted with caution. However, I expect the qualitative results not to change much when I add the other explanatory variables. The difference-in-difference results show that the two significant actions by the EPA and the wood products industry—the 2001 EPA ruling on a more stringent standard for arsenic in drinking water and the voluntary ban on the use of CAA by the wood products industry—have led to statistically significant declines in arsenic use/emissions. While I am not able to claim causality in the time series and panel data specifications the difference-in-difference models imply that as long as lead is an appropriate control for arsenic there is a *causal* relationship between the two decisions and the sharp decline in arsenic use/emissions over the past decade.

[Insert Table 5]

IV. Conclusion

Arsenic, along with other toxic chemicals has been tightly controlled by federal environmental legislations over the past half century. Arsenic is among the most toxic chemicals of all times; exposure to arsenic can cause cancer and other respiratory diseases. While a tightly

controlled substance arsenic remains a legal substance with wide ranging industrial applications. Toward this end, arsenic is an important input in the preserving of wood, manufacturing of computers and other electronics, production of pesticides, and in glass making. Arsenic's multi-facet characteristics and uses pose a major challenge for understanding the factors that account for the ebb and flow of arsenic use/emissions over the past half century, particularly the sharp decline over the past decade. What effect, if any, has had government regulation on the industrial consumption/release of arsenic? The sustained decrease in arsenic use/emissions in recent years is particularly puzzling given that the consumption and environmental releases of lead, a comparable chemical substance to arsenic with respect to the severity of impact on human health and the environment, have exhibited an upward trend in the same time period when historically the two series by and large moved together.

I conduct three separate analyses, the first of which is a time series analysis of aggregate arsenic use (1929-2007) and the second analysis is a panel analysis of arsenic consumption by end users between 1975 and 2004. My findings in these analyses show that on the whole federal regulation has had minimal impact on arsenic use/emissions over the past half century. With this said, selected environmental legislations, such as the Safe Drinking Water Act of 1972 and a related federal action—the 2001 EPA ruling on a more stringent standard for arsenic in drinking water—have accomplished what they set out to do, which is to limit contaminants in drinking water sources.

The time series and panel data specifications are not definitive in uncovering the causal impact of federal regulation on arsenic use, but they offer a descriptive analysis with insights into regulatory impact over time and across arsenic using sectors. At this stage of research, there is

still work to be done to sort out the meaning of some of the results. Robustness tests need to be conducted.

In the third analysis, I am able to exploit the comparability of arsenic and lead in a difference-in-difference model to investigate causally the sharp decline in arsenic use/emissions over the past decade. Initial results suggest that the 2001 decision by the EPA to increase the stringency of the limit on arsenic in drinking water and the wood products industry's voluntary decision to cease the use of CCA in preserving wood for residential construction are responsible for the sharp declines observed in the data.

The results presented in this paper are preliminary. With respect to future work, in addition to robustness tests on the time series and panel data analyses, I plan to expand the third analysis to consider factors that could confound the relationship between the "treatment" and the outcome of interest. Factors that influence the supply and demand of arsenic and lead as commodities would be obvious candidate variables. In particular, a closer look at the increasing use of substitutes in arsenic using sectors is warranted and should shed light on the phasing out of arsenic in these sectors. Substitutes for arsenic are on steadily on the rise; price decreases in these substitutes and thus their accessibility over time will have profound impact on arsenic as an industrial input. Currently, viable substitutes include the use of tributylarsine, a lower toxicity substance, in semiconductor manufacturing. Moreover, two wood preservatives are now being marketed as substitutes for CCA pressure treated lumber. These include Ammoniacal Copper Quat Type B (ACQ-B) and Copper Azole Type A (CBA-A). In addition, untreated wood (cedar and redwood) and non-wood alternatives, such as plastics, metal, and composite materials are also viable substitutes for arsenic in residential and industrial construction. A practical question would be how to proxy for and statistically control for the rise of substitutes.

I plan also to extend my analyses to consider the impact of government regulation on the use/emissions of other toxic chemicals. No doubt some policies have been more effective than others in restricting the use and handling of toxic substances. How have the environmental legislations over the past century differentially impacted the use/emissions of arsenic, selenium, cadmium, antimony, tellurium, mercury, thallium, lead, and inorganic cyanides, the nine chemicals that are judged to be the most toxic to human and animal health according to the CRC Handbook of Chemistry and Physics (2008)?[20] What are the factors that have facilitated or impeded these regulations?

Finally, while my results are preliminary they suggest two policy relevant questions that are normative in nature. The first question is: what is it about the institutional design of SDWA that makes this legislation particularly effective in controlling the use/emissions of arsenic (and other toxic chemicals)? An examination of the incentive structures and/or monitoring and sanctioning mechanisms would be fruitful for uncovering lessons for future policy design for controlling the use of and exposure to highly toxic substances. Secondly, the significance of the voluntary ban by the wood products industry in reducing the manufacturing sector's reliance on arsenic suggest there is much to be gained in engaging firms and other nongovernment actors in policymaking that move beyond the traditional control-and-command approach; as noted in Section I this is now the trend rather than the exception. How might the government design

[20] When comparing chemical I will need to consider their relative toxicities rather than just their use/release levels. This is important because chronic human health risks not only depend on the quantities of chemicals released but also on the characteristics of each chemical (e.g., their differential effect on long term human health), such as its toxicity or the media type where it was emitted. The EPA has created two models to calculate the "toxic risk" of chemicals based on their toxicity scores: The US EPA's Risk Screening Environmental Indicators (RSEI) is recommended for estimating impacts to human health and the EPA's Tool for the Reduction and Assessment of Chemical Impacts (TRACI) is recommended for estimating impacts to human health and the environment.

programs to effectively encourage firms to voluntarily invest in environmentally friendly activities that maximize societal welfare as well as profits?

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Appendix

Table 1
U.S. Federal Regulation on Arsenic and Arsenic Compounds, 1970-2000

Legislation	Description	Hypothesized Effect on Arsenic Use/Emissions
Occupational and Safety Health Act (OHSA) of 1970	OSHA establishes the federal regulatory framework for the control of workplace safety. OSHA's goal is to make sure employers provide their workers a place of employment free from recognized hazards to safety and health, including exposure to toxic chemicals. OSHA establishes permissible exposure limits (PELs) to regulate workplace exposure to air contaminants. OSHA stipulates "the employer shall assure that no employee is exposed to inorganic arsenic at concentrations greater than 10 micrograms per cubic meter of air (10 ug/m(3)), averaged over any 8-hour period."[21]	Negative (-)
Clean Water Act/Water Pollution Control Act of 1972	CWA is an amendment to the federal Water Pollution Control Act of 1972, establishes the basic structure for regulating discharges of pollutants into the waters of the United States. Section 307 defines a list of priority pollutants for which the U.S. EPA must establish ambient water- quality criteria (the basis of state water-quality standards) and effluent limitations (rules controlling environmental releases from specific industrial categories based on the "best available technology economically achievable").	Negative (-)
Safe Drinking Water Act (SDWA) of 1974	Enacted to protect the quality of drinking water in the U.S., SWDA requires the U.S. EPA to establish primary drinking-water regulations for contaminants in public water systems that may have adverse effects on people's health. Such regulations typically include a media quality standard that defines legally allowable concentrations of toxic chemicals, called maximum	Negative (-)

[21] Source: http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=STANDARDS&p_id=10023.

	contaminant levels (MCLs). The EPA had originally adopted an arsenic limit of 50 ppb for drinking water.	
Resource Conservation and Recovery Act (RCRA) of 1976	RCRA is the primary environmental law governing the proper disposal of hazardous wastes. While RCRA handles many regulatory functions of hazardous and non-hazardous waste, one of its most notable provisions regard the Subtitle C program which tracks the progress of hazardous wastes from their point of generation, their transport, and their treatment and/or disposal, known overall the "cradle to grave" system.	Negative (-)
Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980	CERCLA created the Superfund Program to clean up uncontrolled or abandoned hazardous-waste sites and to respond to accidents, spills, and other emergency releases of pollutants and contaminants, including long-term remedial action to deal with toxic releases. Section 101 defines a list of hazardous chemicals for which the EPA must establish regulations. Releases of CERCLA hazardous substances in amounts greater than their "reportable quantity" must be reported to the National Response Center and to state and local government officials.	Negative (-)
Emergency Planning and Community Right-to-Know Act (EPCRA) of 1986	EPCRA established the Toxics Release Inventory (TRI). TRI requires manufacturing companies in certain industrial sectors (SIC codes 20-39) to publicly report environmental releases and transfers of chemicals on a list established by Section 313. Chemicals are listed if they are known to cause or can reasonably be anticipated to cause significant adverse acute effects on health at concentrations likely beyond facility boundaries; cancer, teratogenic effects, reproductive effects, neurological effects, heritable genetic mutations, or other chronic effects on health; or significant damage to the environment.	Negative (-)
1988 EPA designation of CAA as a restricted pesticide	EPA concludes its Special Review of CCA and issues a new	Negative (-)

	registration standard for inorganic arsenicals, which imposes additional conditions on CCA's usages, but no restrictions on its use in treated wood . CCA is now classified as a restricted use pesticide.	
Clean Air Act of 1990	Congress amended the Clean Air Act in 1990 to address a large number of air pollutants that are known to cause or may reasonably be anticipated to cause adverse effects to human health or adverse environmental effects. 188 specific pollutants and chemical groups were initially identified as hazardous air pollutants (HAPs), and the list has been modified over time.	Negative (-)
1993 EPA ban on the use of arsenide acid	The EPA bans use of arsenic acid (a pesticide containing inorganic arsenic, which is also a component of CCA) for treating cotton crops	Negative (-)
2000 EPA ruling on arsenic standards	The EPA adopts a more stringent arsenic limit from 50 ppb to 10 ppb for drinking water; this ruling pertains to both organic and inorganic arsenic.	Negative (-)

Source: The Scorecard/Environmental Defense (http://www.scorecard.org/about/about.tcl), the U.S. EPA (http://www.epa.gov/), and the U.S. Occupational Safety & Health Administration (www.osha.gov).

Table 2
Descriptive Statistics

Panel A: Time Series

Variable	Obs	Mean	Std. Dev.	Min	Max
year	79	1968	22.94922	1929	2007
countrycode	0				
recession	79	.2658228	.4445932	0	1
voluntaryban	79	.0632911	.2450417	0	1
regulation	79	.3725136	.4306529	0	1
reg_OHSA	79	.4810127	.502832	0	1
reg_CWA	79	.4556962	.5012157	0	1
reg_SDWA	79	.4303797	.4982931	0	1
reg_RCRA	79	.4050633	.4940411	0	1
reg_Superf~d	79	.3544304	.4813969	0	1
reg_TRI	79	.278481	.4511157	0	1
reg_CAA	79	.2278481	.4221243	0	1
reg_As	79	.0886076	.2859924	0	1
reg_pest	79	.2531646	.4376029	0	1
reg_AsAcid~n	79	.1898734	.3947069	0	1
lnmedia	79	3.128526	.542127	1.791759	4.564348
WWII	79	.0759494	.2666099	0	1
lnarsenic	79	9.743588	.3622382	8.571681	10.38282

indstructure	79	673.4158	35.4815	606.7469	728.6075
GaAs	79	.2658228	.4445932	0	1
CCA_C	79	.8860759	.3197492	0	1
lnprice00	79	6.838017	.2982008	6.002748	7.581641

Panel B: Panel Data

Ag Sector

Variable	Obs	Mean	Std. Dev.	Min	Max
year	28	1990.5	8.225975	1977	2004
arsenic	28	3900.357	2833.099	750	9500
rvalue	28	111527.8	12530.95	87525.69	142471.7
lnarsenic	28	7.93505	.8973198	6.620073	9.159047
lnrvalue	28	11.61597	.1121956	11.37969	11.8669
indstructure	28	146.5588	2.71855	140.877	152.6902
voluntaryban	28	0	0	0	0
GaAs	28	0	0	0	0
recession	28	.1785714	.390021	0	1
reg_Superf~d	28	.8928571	.3149704	0	1
reg_TRI	28	.6785714	.4755949	0	1
reg_CAA	28	.5357143	.5078745	0	1
reg_As	28	.1428571	.3563483	0	1
reg_AsAcid~n	28	.4285714	.5039526	0	1

Electronics Sector

Variable	Obs	Mean	Std. Dev.	Min	Max
year	28	1990.5	8.225975	1977	2004
arsenic	28	652.1429	260.8726	250	1300
rvalue	28	117886.1	30751.42	64521.26	185563
lnarsenic	28	6.404983	.3968391	5.521461	7.17012
lnrvalue	28	11.64338	.2701409	11.07475	12.13115
indstructure	28	147.2821	6.537743	133.7248	159.2959
voluntaryban	28	0	0	0	0
GaAs	28	.6428571	.48795	0	1
recession	28	.1785714	.390021	0	1
reg_RCRA	28	1	0	1	1
reg_Superf~d	28	.8928571	.3149704	0	1
reg_TRI	28	.6785714	.4755949	0	1
reg_CAA	28	.5357143	.5078745	0	1
reg_As	28	.1428571	.3563483	0	1
reg_AsAcid~n	28	.4285714	.5039526	0	1

Glass Sector

Variable	Obs	Mean	Std. Dev.	Min	Max
year	28	1990.5	8.225975	1977	2004
arsenic	28	766.4286	123.6846	600	1000
rvalue	28	35431.15	6425.958	23732.03	46085.54
lnarsenic	28	6.629387	.1595258	6.39693	6.907755
lnrvalue	28	10.4596	.1806249	10.07458	10.73825
indstructure	28	119.8943	3.961209	111.5718	126.0484
voluntaryban	28	0	0	0	0
GaAs	28	0	0	0	0

recession	28	.1785714	.390021	0	1
reg_Superf~d	28	.8928571	.3149704	0	1
reg_TRI	28	.6785714	.4755949	0	1
reg_CAA	28	.5357143	.5078745	0	1
reg_As	28	.1428571	.3563483	0	1
reg_AsAcid~n	28	.4285714	.5039526	0	1

Wood Products Sector

Variable	Obs	Mean	Std. Dev.	Min	Max
year	28	1990.5	8.225975	1977	2004
arsenic	28	14048.21	6406.673	2400	26500
rvalue	28	26505.46	4319.552	15775.13	34237.45
lnarsenic	28	9.396671	.6406653	7.783224	10.1849
lnrvalue	28	10.17102	.175636	9.66619	10.44108
indstructure	28	113.6503	3.719872	103.1014	119.4571
voluntaryban	28	.0714286	.2622653	0	1
GaAs	28	0	0	0	0
recession	28	.1785714	.390021	0	1
reg_Superf~d	28	.8928571	.3149704	0	1
reg_TRI	28	.6785714	.4755949	0	1
reg_CAA	28	.5357143	.5078745	0	1
reg_As	28	.1428571	.3563483	0	1
reg_AsAcid~n	28	.4285714	.5039526	0	1

Table 3
Time Series Analysis

```
Regression with Newey-West standard errors          Number of obs  =        78
maximum lag: 3                                      F( 17,    60)  =    103.93
                                                    Prob > F       =    0.0000

------------------------------------------------------------------------------
             |             Newey-West
   lnarsenic |      Coef.   Std. Err.      t     P>|t|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
    reg_OHSA |
          L1. |  -.0522109   .1023267    -0.51    0.612    -.2568948    .1524731
     reg_CWA |
          L1. |   .5524801   .0592175     9.33**  0.000     .4340275    .6709327
    reg_SDWA |
          L1. |  -.5219832   .1660249    -3.14**  0.003    -.8540825   -.1898838
    reg_RCRA |
          L1. |   .0725559   .1320983     0.55    0.585      -.19168    .3367918
reg_Superf~d |
          L1. |   .4005922   .0877936     4.56**  0.000     .2249789    .5762056
     reg_TRI |
          L1. |   .1907113   .0615035     3.10**  0.003      .067686    .3137366
     reg_CAA |
          L1. |   .0365203    .059743     0.61    0.543    -.0829834     .156024
      reg_As |
          L1. |   -.102857   .0886997    -1.16    0.251    -.2802829    .0745689
reg_AsAcid~n |
          L1. |    .083677   .0593226     1.41    0.164    -.0349858    .2023398
indstructure |
          L1. |  -.0059482   .0033791    -1.76*   0.083    -.0127074    .0008111
   recession |
          L1. |  -.0901115   .0762594    -1.18    0.242    -.2426531      .06243
   lnprice00 |
          L1. |  -.1470604   .1653499    -0.89    0.377    -.4778095    .1836887
         WWII |
          L1. |   .4591621   .1171539     3.92**  0.000     .2248195    .6935048
       CCA_C |
          L1. |   .0941362   .1509241     0.62    0.535     -.207757    .3960294
        GaAs |
          L1. |  -.0085687   .0248398    -0.34    0.731    -.0582558    .0411183
voluntaryban |
          L1. |  -1.245734   .0802063   -15.53**  0.000     -1.40617   -1.085297
     lnmedia |
          L1. |   .0293812   .0443177     0.66    0.510    -.0592673    .1180298
        _cons |   14.39338   2.848599     5.05**  0.000     8.695339    20.09143
------------------------------------------------------------------------------
```

**statistical significance at $p \geq 0.05$.
*statistical significance at $p = 0.10$.

Table 4
Panel Data Analysis

```
Cross-sectional time-series FGLS regression

Coefficients:  generalized least squares
Panels:        heteroskedastic
Correlation:   panel-specific AR(1)

Estimated covariances      =         4          Number of obs      =       108
Estimated autocorrelations =         4          Number of groups   =         4
Estimated coefficients     =        30          Time periods       =        27
                                                Wald chi2(29)      =    124.79
                                                Prob > chi2        =    0.0000

------------------------------------------------------------------------------
   lnarsenic |      Coef.   Std. Err.      z     P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
reg_Superf~d |
         L1. |   .0904926   .2175806     0.42    0.677    -.3359574    .5169427
     reg_TRI |
         L1. |   .2818846    .266142     1.06    0.290    -.2397441    .8035133
     reg_CAA |
         L1. |  -.0216899   .1677028    -0.13    0.897    -.3503814    .3070017
      reg_As |
         L1. |  -.2399962   .2671559    -0.90    0.369    -.7636122    .2836198
reg_AsAcid~n |
         L1. |  -.3114707   .1388671    -2.24**  0.025    -.5836452   -.0392963
indstructure |
         L1. |   .0044739   .0105112     0.43    0.670    -.0161277    .0250755
   recession |
         L1. |    .020368   .1011626     0.20    0.840    -.1779071    .2186431
        GaAs |
         L1. |  -.1266638   .3061567    -0.41    0.679      -.72672    .4733923
voluntaryban |
         L1. |  -1.298732   .2540721    -5.11**  0.000    -1.796704   -.8007593
        yr76 |  -.0155762   .1415288    -0.11    0.912    -.2929675    .2618152
        yr77 |   .1717786   .0992479     1.73    0.083    -.0227438     .366301
        yr79 |   .3848405   .1464603     2.63**  0.009     .0977836    .6718975
        yr80 |    .160479   .1053584     1.52    0.128    -.0460197    .3669777
        yr82 |   .3042688    .148518     2.05**  0.040     .0131788    .5953588
        yr83 |   .0620947   .1874856     0.33    0.740    -.3053703    .4295596
        yr84 |   .1789981   .2116684     0.85    0.398    -.2358643    .5938606
        yr85 |  -.0485355   .1420527    -0.34    0.733    -.3269537    .2298827
        yr86 |   -.018779   .0990518    -0.19    0.850     -.212917     .175359
        yr88 |  -.1072471   .0992398    -1.08    0.280    -.3017537    .0872594
        yr17 |  -.0779536   .1003908    -0.78    0.437    -.2747159    .1188086
        yr91 |  -.1067359   .1028654    -1.04    0.299    -.3083485    .0948766
        yr92 |  -.2094837   .1411325    -1.48    0.138    -.4860983    .0671308
        yr93 |  -.0354807   .1700458    -0.21    0.835    -.3687643    .2978029
        yr94 |   .1463467   .1970182     0.74    0.458     -.239802    .5324953
        yr97 |  -.2335817   .2161073    -1.08    0.280    -.6571442    .1899808
        yr98 |  -.1329099    .233115    -0.57    0.569     -.589807    .3239871
        yr99 |   -.056777   .2473091    -0.23    0.818     -.541494      .42794
        yr01 |   .0286249   .1416227     0.20    0.840    -.2489505    .3062003
        yr02 |  -.0148103   .1781535    -0.08    0.934    -.3639848    .3343641
       _cons |   6.759555   1.490845     4.53**  0.000     3.837551    9.681558
------------------------------------------------------------------------------
```

**statistical significance at $p \geq 0.05$.

Table 5
Difference-in-Difference Analysis

Panel A: 2001 EPA Ruling

```
Linear regression                               Number of obs =      20
                                                F(  3,    16) =   27.57
                                                Prob > F      =  0.0000
                                                R-squared     =  0.6223
                                                Root MSE      =  .36804

------------------------------------------------------------------------------
             |               Robust
 lnemissions |      Coef.   Std. Err.      t      P>|t|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
 postEPA2001 |   .3545315   .0468382     7.57      0.000     .2552389    .4538241
     EPA2001 |   .5579777   .0938147     5.95      0.000     .3590994     .756856
EPA2001treat |  -1.416408   .2499436    -5.67***   0.000    -1.946265   -.8865514
       _cons |   19.58851   .0358029   547.12      0.000     19.51262    19.66441
------------------------------------------------------------------------------
```

**statistical significance at least at p = 0.001.

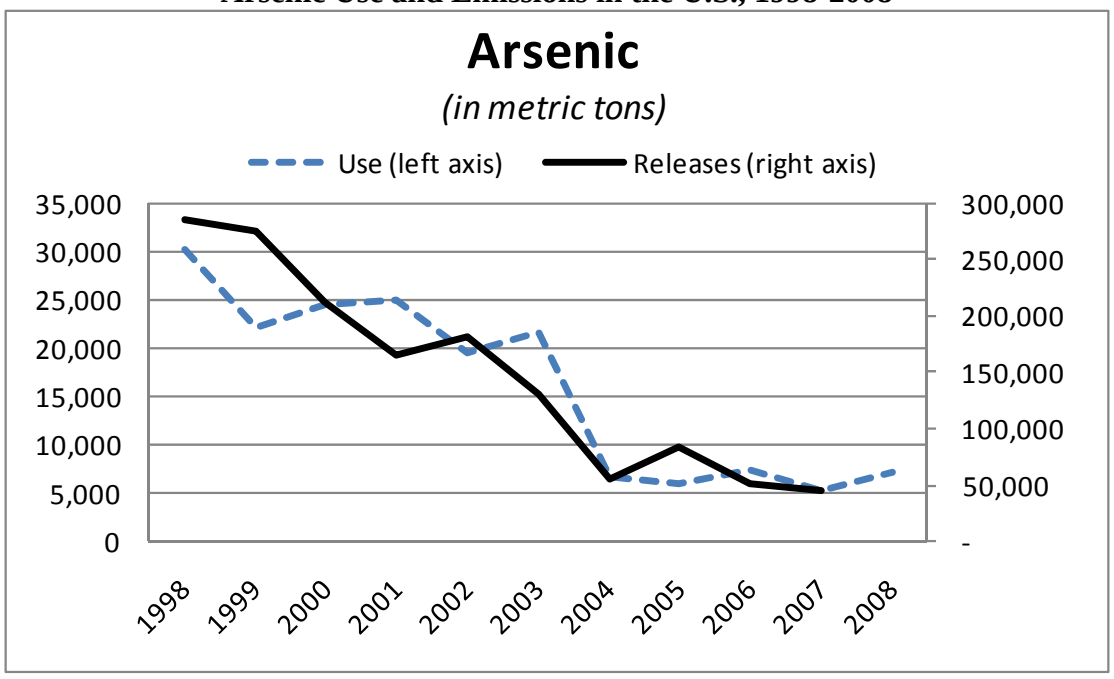
Panel B: Wood Products Voluntary Ban

```
Linear regression                               Number of obs =      20
                                                F(  3,    16) =   36.69
                                                Prob > F      =  0.0000
                                                R-squared     =  0.8574
                                                Root MSE      =  .22617

------------------------------------------------------------------------------
             |               Robust
 lnemissions |      Coef.   Std. Err.      t      P>|t|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
postvolunt~n |   .2660384   .0717137     3.71     0.002     .1140121    .4180647
voluntaryban |   .1769466   .1426448     1.24     0.233    -.1254467      .47934
voluntaryb~t |  -1.526137   .1962291    -7.78***  0.000    -1.942124    -1.11015
       _cons |   19.73027   .0676578   291.62     0.000     19.58684     19.8737
------------------------------------------------------------------------------
```

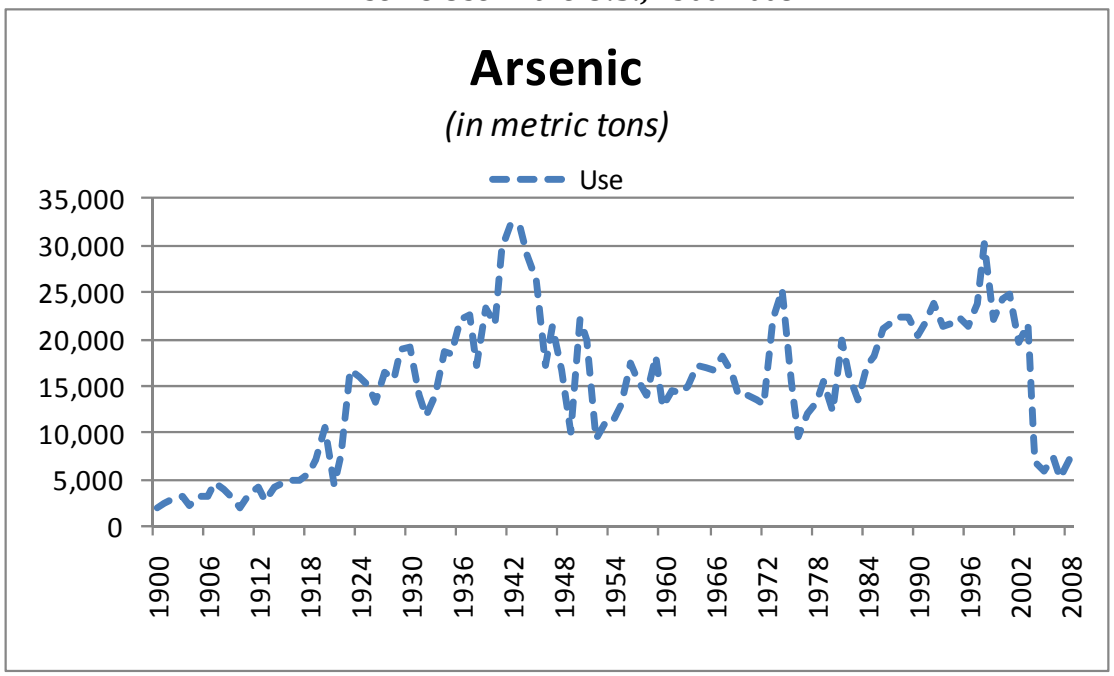
**statistical significance at least at p = 0.001.

Figure 1
Arsenic Use and Emissions in the U.S., 1998-2008



Source: USGS Mineral Year Book (http://minerals.usgs.gov/ds/2005/140/) and the EPA's Toxic Release inventory (http://www.epa.gov/triexplorer/). Data from the TRI include information on both toxic releases on-site and off-site.

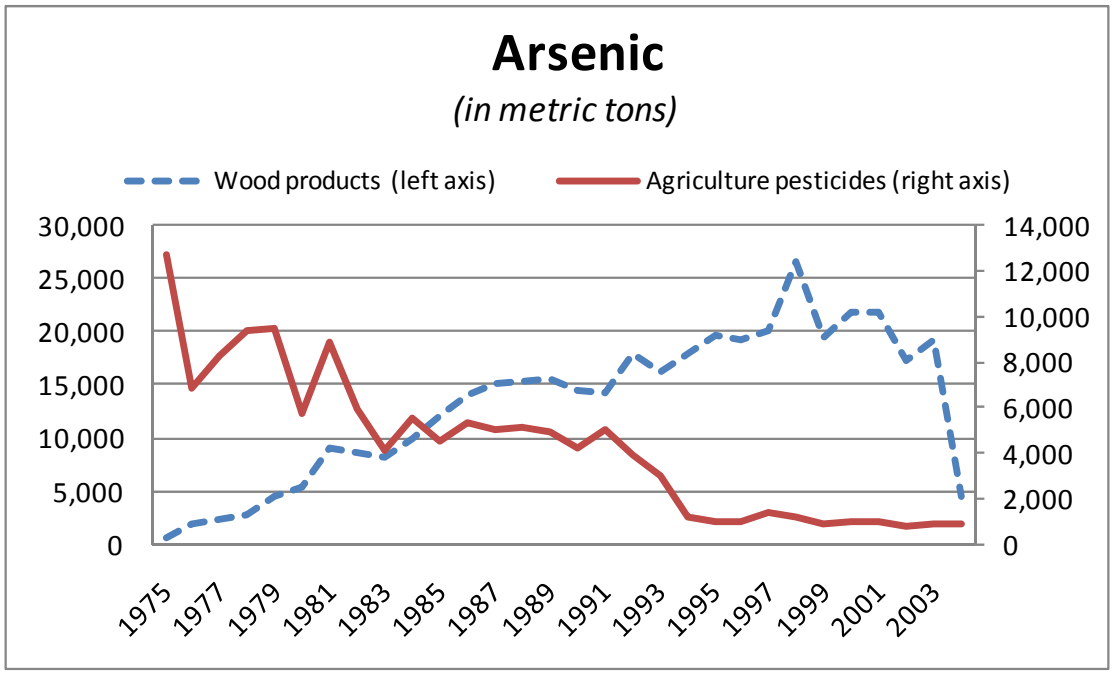
Figure 2
Arsenic Use in the U.S., 1900-2008



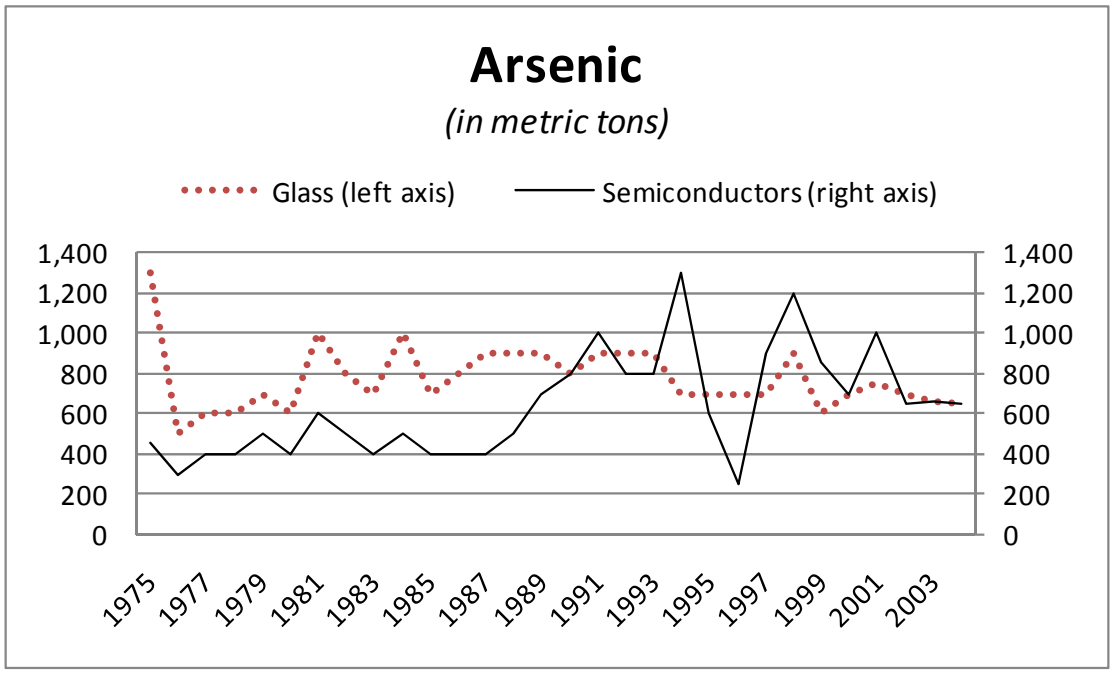
Source: USGS Mineral Year Book (http://minerals.usgs.gov/ds/2005/140/).

Figure 3
Arsenic Use Across Sectors, 1975-2004

Panel A

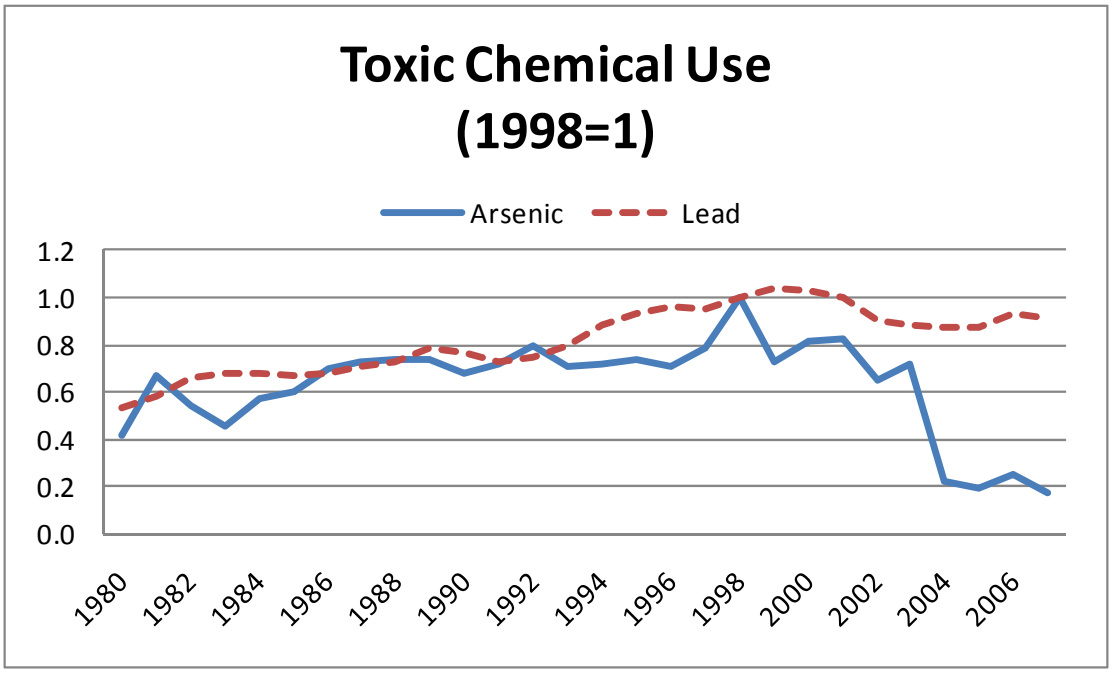


Panel B



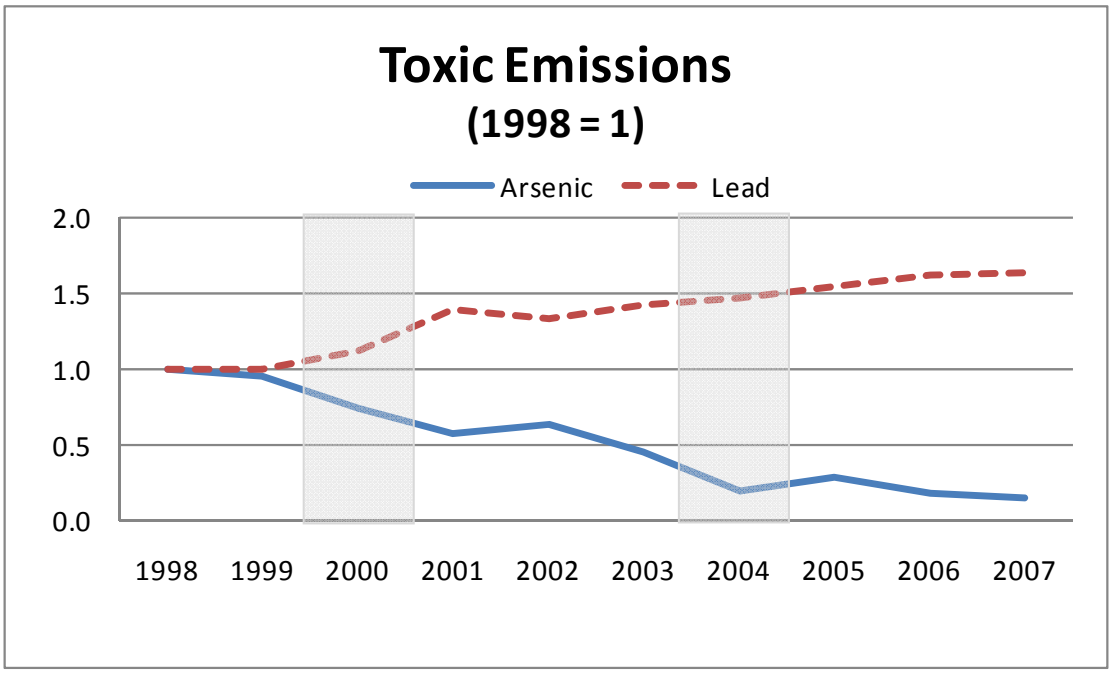
Source: USGS Mineral Year Book (http://minerals.usgs.gov/ds/2005/140/).

Figure 4
Toxic Chemical Use, 1980-2007



Source: USGS Mineral Year Book (http://minerals.usgs.gov/ds/2005/140/).

Figure 5



Source: The EPA's Toxic Release Inventory (TRI).
*The two gray bars represent the 2001 EPA ruling on arsenic in drinking water and the 2004 voluntary ban of the use of CAA for residential construction in the wood products industry.