The 1983-84 Suspensions of EDB under FIFRA and the 1989 Asbestos Ban and Phaseout Rule under TSCA: Two Case Studies in EPA's Use of Science

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Discussion Paper 97-06

March 1997 (Revised)

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Abstract

This paper discusses EPA's acquisition and use of science in a decision under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA): the 1983-84 suspensions of ethylene dibromide (EDB); and in a decision under the Toxic Substances Control Act (TSCA): the 1989 Asbestos Ban and Phaseout Rule. By requiring EPA to balance the risks and benefits of the commercial use of toxic substances, both statutes place considerable analytical burdens on the agency, though TSCA places a more substantial burden on EPA for acquiring science and demonstrating unreasonable risks. In the case of EDB, data produced outside EPA over which the agency had no control incited a public alarm. Because a senior EPA official had contaminated the agency's reservoir of public trust by cooking the scientific data to provide regulatory relief, EPA had no credibility to portray the health risks of EDB in an objective manner. In the case of asbestos, the reviewing court, despite its limited scientific capability and lack of political accountability, substituted its own science policy judgment for that of politically accountable decisionmakers of the more expert administrative agency. The court was arguably invited to do so, however, by the substantial evidentiary judicial review standard specified for TSCA by the legislature. Both cases illustrate the need for and difficulty of generating and considering scientific information regarding tradeoffs among risks in environmental regulatory decisionmaking.

Abstract

Table of Contents

INTRODUCTION

The case studies included in this discussion paper are part of a project that Resources for the Future (RFF) is conducting under a cooperative agreement with the U.S. Environmental Protection Agency (EPA) and with general support from RFF. The case studies were originally vetted as RFF Discussion Paper 97-06 in 1996, and this revised version of the discussion paper reflects many useful comments and corrections supplied by reviewers.

The overall study is broadly concerned with the acquisition and use of scientific information by the Environmental Protection Agency in regulatory decisionmaking. The overall study focuses chiefly on national rulemaking (e.g., setting National Ambient Air Quality Standards and banning pesticides or toxic substances), as opposed to site-specific decisionmaking (e.g., Superfund remedy selection). For the purposes of this study, environmental "science" refers to information that can be used in assessing risks to human health, welfare, and the environment. (Therefore, economic and engineering information are not a chief focus of this study.) The project aims to help policymakers and others better understand the factors and processes that influence EPA's acquisition and use of science in national rulemaking so that they can better evaluate recommendations for improving environmental regulatory institutions, policies, and practices.

In all, eight case studies will be included as appendices to the full report:

- 1987 Revision of the National Ambient Air Quality Standard for Particulates (NAAQS)
- 1993 Decision Not to Revise the NAAQS for Ozone
- 1991 Lead/Copper Rule under the Safe Drinking Water Act (SDWA)
- 1995 Decision to Pursue Additional Research Prior to Revising the Arsenic Standard under SDWA
- 1983/4 Suspensions of Ethylene Dibromide under the Federal Insecticide, Fungicide, and Rodenticide Act
- 1989 Asbestos Ban & Phaseout Rule under the Toxic Substances Control Act
- Control of Dioxins (and other Organochlorines) from Pulp & Paper effluents under the Clean Water Act (as part of the combined air/water "cluster rule" proposed in 1993)
- Lead in Soil at Superfund Mining Sites

The case studies were selected in consultation with informal advisors to the project and are not intended as a random or representative sample of EPA regulatory decisions. None of the case studies could be fairly characterized as routine or pedestrian. As a group, the cases tend toward the "high-profile" end of the distribution of EPA decisions. Nevertheless, among the case studies, there is some variability in the political and economic stakes involved and in the level of development of the underlying science. The cases selected involve each of the "national" environmental regulatory statutes (Clean Air Act; Safe Drinking Water Act; Toxic Substances Control Act; Federal Insecticide,

Fungicide, and Rodenticide Act; and Clean Water Act), and two cases involve decisions to maintain the status quo (ozone and arsenic), as opposed to the remainder of the cases which involve decisions to change from the status quo.

Methodology

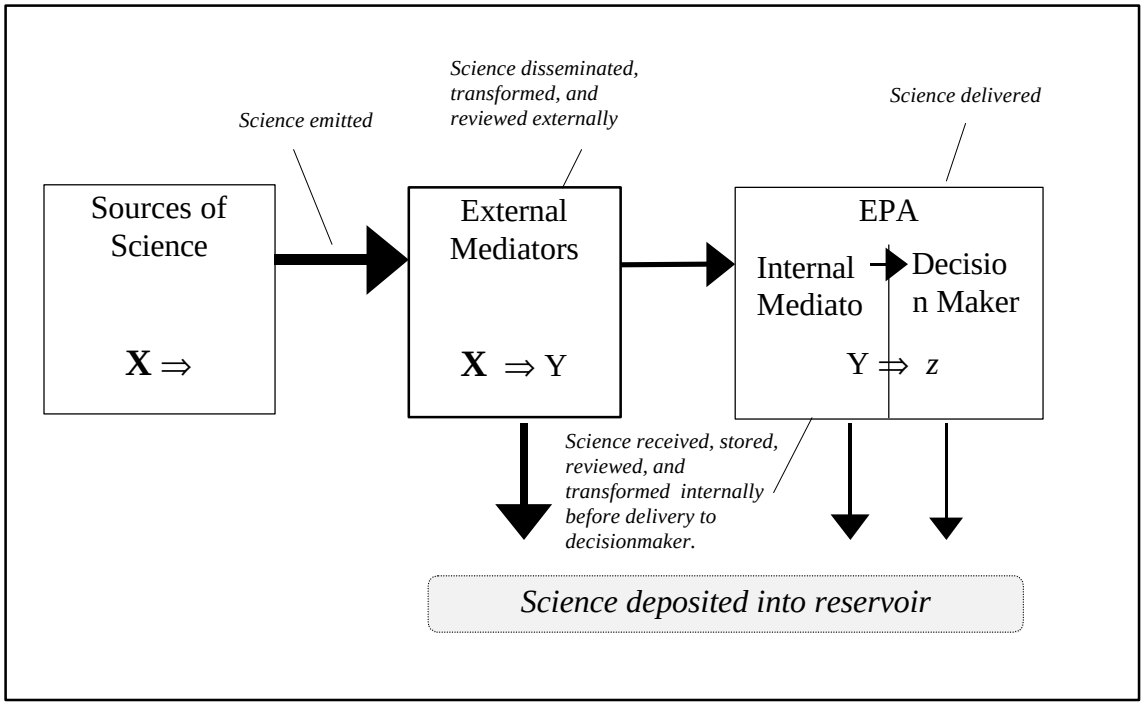
Development of the case studies was based on literature review and interviews with persons inside and outside EPA. The number of interviewees per case study varied roughly from a half dozen to a dozen. There was an effort to ensure balance in the group of respondents for any particular case study, but because of the relatively small number of respondents and the non-random nature of the selection process, *extreme* caution should be taken in interpreting the numerical response summaries that are reported. Interviews were conducted primarily using a structured questionnaire format, but in some cases, comments were sought from specific individuals regarding particular issues instead of the case as a whole. In addition to interviews specific to particular case studies, interviews were also conducted for the overall study to elicit the views of current and former policymakers, senior scientists, specialists in regulatory science issues, and others regarding EPA's acquisition and use of science. The case studies also incorporate many comments and insights from these interviewees.

In all instances, interviewees were given the option of speaking for attribution or off-the-record, and almost all respondents elected to speak off-the-record. A complete listing of the more than 100 interviewees for the overall study will be included as an appendix to the final report. The selection of interviewees considered that individuals from the bench scientist through the agency staff analyst to the politically appointed decisionmaker, as well as advocates from outside the agency, would provide informative perspectives. Among the wide range of interviewees were: 5 of 6 former EPA Administrators, 4 current or former Deputy Administrators, and 5 current or former Assistant Administrators; 4 current or former congressional staff; several current and former EPA Science Advisory Board members; various representatives of industry and environmental advocacy groups; environmental journalists; and academics from the diverse fields of biology, public health, economics, political science, psychology, and philosophy. But to better understand the processes occurring *within* the agency, interviewees were disproportionately selected from among current and former EPA officials.

A prominent feature of the case studies consists of an effort to map the origins, flow, and effect of scientific information relating to a particular decision. To accomplish this, the case studies make use of an extended analogy to fate and transport modeling. As used in risk assessment, this modeling procedure predicts the movement and transformation of pollutants from their point of origin to their ultimate destination. Thus, to extend the analogy, one can imagine universities and research institutes "emitting" scientific findings, which are disseminated and "transformed" by the media and consultants outside the agency. (An alternative pattern is when scientific findings are generated within EPA by agency scientists.) Science can enter EPA through multiple "exposure routes," which assimilate information differently; once inside the agency, information is

"metabolized" prior to its "delivery" to the "target organ" (the decision-maker). This fate and transport terminology is adopted because it is part of the vernacular of many of those providing the information and of many of the ultimate users of the study results. Figure A presents a simplified model of the fate and transport of science in environmental regulation for illustrative purposes.

Figure A. Fate and Transport of Science in Environmental Regulation



Making use of these conceptual models, we attempt to address questions specifically about the *scientific information* in each of the case studies, such as: what are the sources and their relative contributions? where are the points-of-entry? who are the gatekeepers? what is the internal transport mechanism? how is the information transformed as it flows through the agency? what does and doesn't get communicated to the decisionmaker? and where and how is the information ultimately applied?

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A. The 1983-84 Suspensions of EDB under FIFRA

1. Background

Ethylene dibromide (EDB) once was widely used as an agricultural pesticide and as an additive to leaded gasoline to remove lead deposits from engines.[1] As a highly toxic pesticide detected in wells and consumer grain products, EDB became front-page news in the winter of 1983-84. However, the EDB story began in fact many years earlier, and it continues today as substitutes for EDB are themselves being phased out. In the case of one substitute, methyl bromide, the source of concern is an environmental effect that was completely off EPA's radar screen when it announced emergency suspensions of EDB in 1983-84--stratospheric ozone depletion.

EDB was an economically important pesticide used on food and nonfood crops. More than 90% of EDB's pesticide usage was as a soil fumigant. The pesticide was injected directly into the soil before planting crops to destroy nematodes, small parasitic worms that attack the roots of plants (EPA 1983). EDB-treated fields grew more than thirty fruit and vegetable crops in California, Hawaii, and the southern states (Jasanoff 1990). EDB was also used as a fumigant to control insects and molds on grain milling machinery and flour mills and on stored grain products. An estimated 75% of the nation's grain mills used EDB as a fumigant. Another important use of EDB was as a postharvest fumigant for grains, fruits and vegetables (*Fed. Reg.*, Vol. 42, pp. 63134-63161; *Fed. Reg.*, Vol. 49, pp. 4452-4457).[2] Agricultural imports and exports to and from the U.S. were commonly required to be fumigated with EDB to prevent the international spread of agricultural pests and pathogens.

Farmers began using EDB in the 1920s, and the first reports of adverse effects of EDB on test animals surfaced in 1927 (Jasanoff 1990). EDB was registered under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) in 1948 when the Department of Agriculture was responsible for administering pesticide registrations. Under FIFRA, pesticides are registered separately for particular uses, and between 1949 and 1956, EDB was registered as a fumigant for soil, fruits and vegetables, and stored grain. At the same time, the Food and Drug Administration (FDA) was responsible for setting tolerance levels for pesticide residues and food additives under the Federal Food, Drug, and Cosmetics Act (FFDCA). Based on data originally submitted by pesticide manufacturers, FDA set tolerances in 1955 for organic bromine compounds and inorganic bromine residues in food resulting from the use of EDB. In 1956, FDA granted Dow Chemical an exemption from a tolerance for inorganic bromine residues when EDB was used as postharvest fumigant for raw grain (Krimsky and Plough 1988).

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[1] Of over 300 million pounds of EDB produced each year by 1984, around 20 million pounds were used as pesticide and 230 million pounds went into leaded gasoline (Jasanoff, 1990, p. 130).

[2] Additional uses, described by EPA as minor, included control of mountain pine bark beetles in the western states by federal and state forestry agencies; control of dry-wood and subterranean termites in structural pest operations; control of wax moth in honey combs (*Fed. Reg.*, Vol. 42, pp. 63134-63161).

The federal approach to regulating pesticides began to change slowly in response to public concern about pesticides and the environment sparked by Rachel Carson's landmark 1962 book, *Silent Spring*. In 1970, the administrative reorganization establishing EPA shifted the pesticides registration and tolerance setting divisions from USDA and FDA to the new agency. Congress amended FIFRA two years later, propelling government into the active regulation of the harmful environmental and health effects arising from pesticide use. FIFRA requires the registration and labeling of all pesticide products (Sec. 3) and authorizes EPA to suspend, cancel, or restrict an existing pesticide registration if it present "any unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide" (Sec. 2). The suspension provisions of FIFRA (Sec. 6) authorize the Administrator, upon a finding of "imminent hazard," to take regulatory action immediately (announce a suspension) to avoid the lengthy time required for cancellation proceedings, but do allow registrants 5 days to request an expedited hearing. The Administrator is authorized to issue an "emergency suspension," which does not permit a hearing, upon findings of an "imminent hazard" and an "emergency."

As a consequence of the "unreasonable risk" standard, FIFRA is considered a "balancing" statute requiring the Administrator to weight the costs and benefits of pesticide use. Because FIFRA places the burden of proving that a pesticide product satisfies the criteria for registration on the registrant, FIFRA is a licensing program that permits EPA to require pesticide manufacturers to provide chemical testing and other data needed for regulatory decisionmaking. FIFRA also requires external review of EPA proposals to suspend or cancel pesticide registrations by the USDA and an official Scientific Advisory Panel (SAP).

In order to expedite the risk-benefit review of some 50,000 pesticide products that had been approved during the previous 30 years, EPA in 1975 instituted a modified review process called Rebuttable Presumption Against Registration (RPAR). Pesticides flagged as posing a substantial question of safety by a screening process using a series of risk criteria entered the RPAR process, which began with an informal comment period during which stakeholders could either rebut the presumption of unreasonable risk or seek to document the hazard. Despite EPA's hopes that RPAR would streamline the review of existing pesticide registrations, three to seven years were ordinarily required for a full RPAR review (Jasanoff 1990).

By 1973, a number of scientific studies had reported that EDB caused mutations and reproductive damage in animals. In 1975, the National Cancer Institute (NCI) reported that EDB should be considered a human carcinogen. The findings of cancer and noncancer effects in animals, the discovery of EDB traces in food, and a petition by the Environmental Defense Fund (EDF) led EPA to issue an RPAR notice for EDB in 1977 (*Fed. Reg.*, Vol. 42, pp. 63134-63161). Following the notice, EPA was peppered with rebuttal comments from industry. In December 1980, the outgoing Carter administration issued a preliminary notice of its proposal to cancel EDB's uses as a fumigant for grain

and grain milling machinery and as a citrus fumigant (*Fed. Reg.*, Vol. 45, pp. 81516-81524).

During its first years, the Reagan administration sat on the proposal, and EDB became "a symbol of congressional concern" about the delay in pesticide reviews under FIFRA in general and, in particular, EPA pesticide policy under Administrator Anne Gorsuch-Burford (Krimsky and Plough 1988). The new administration had sharply reduced the pesticides program staff, and in particular, the staff responsible for reviewing registered pesticides. Meanwhile, EDB took on complicated interstate and international dimensions. In 1981, California agricultural authorities embarked on a massive fumigation program using EDB in an effort to control the Mediterranean fruit fly (Medfly). In response, the state's Occupational Safety and Health Administration (Cal OSHA) proposed a stringent workplace standard for EDB, and Japanese dockworkers refused to unload fruit arriving in their country until the Cal OSHA exposure standard was adopted. (Grapefruit exports to Japan were worth $65 million in 1982.) The Japanese government, on the other hand, was more concerned with preventing the introduction of the Medfly and insisted on fumigation of all produce arriving from California. California supermarkets, in the meantime, started boycotting EDB-fumigated fruit from Texas and Florida to meet Cal OSHA standards (Jasanoff 1990).

The process within EPA was given a prod when EDB groundwater contamination was detected between 1982 and 1983 in California, Florida, Georgia, and Hawaii. While EPA had expressed concerns about EDB's use in soil fumigation in the 1980 notice, groundwater contamination was not thought to be a problem due to EDB's volatility. It was, after all, a *fumigant*. According to Rick Johnson, the EPA EDB review team leader, the discovery of EDB in groundwater "was the straw that broke the camel's back" (Sun 1984). Shortly after Administrator Anne Gorsuch-Burford, John Todhunter, Assistant Administrator for the Office of Pesticides and Toxic Substances (OPTS), and most other senior officials left EPA in a 1983 housecleaning, Administrator William Ruckelshaus and OPTS Acting Assistant Administrator Don Clay acted quickly, issuing an emergency suspension of EDB's soil fumigant registration in October 1983 (*Fed. Reg.* Vol. 48, pp. 46228-46248).

Shortly thereafter, public alarm about EDB in grain products mushroomed. The FDA had begun monitoring EDB residues in food in 1978 after EPA first placed EDB in the RPAR process. In December 1983, the Florida Agricultural Commission reported that EDB residues had been detected in numerous grain products and started a product embargo. According to a former senior EPA official, when the State of Florida started pulling bread off grocery shelves because of EDB, it created a public panic. Other states responded with similar emergency measures against grain products. An EPA official relates a story revealing the level of hysteria: "I had a mother call me on the phone, and she would not let her children eat bread, orange juice, drinking water. She said, 'I have not fed my babies in two days because I don't know what to feed them.'" What seemed to scare the public most was that EDB was reported to be potentially capable of causing infertility and genetic mutations. Such dreaded effects that could damage future

generations were perceived as being "riskier" than cancer. Shortly after being reinstated at the helm of EPA, Administrator Ruckelshaus gave a major press conference at which he announced that the agency was conducting an accelerated study to determine the risks of EDB in an attempt to calm public fears.

According to a former senior EPA official, a Florida state official sought to divert attention to the use of EDB as a grain fumigant to cover the state's negligence in permitting Florida citrus growers to use EDB as a soil fumigant greatly in excess of standards, which had lead to high levels of groundwater contamination. Added to the alarm of domestic consumers were international concerns about the safety of U.S. grain exports. To allay public fears about the nation's food supply, EPA issued an emergency suspension of EDB as a fumigant for grain and grain milling machinery in February 1984 (*Fed. Reg.*, Vol. 49, pp. 4452-4457).

Since the emergency suspensions of EDB in 1983-84, EPA has announced the phaseout of methyl bromide, a substitute for some EDB uses, due to its contribution to stratospheric ozone depletion, and California suspended, then restricted the use of Telone, another EDB substitute. The suspensions and restrictions of EDB susbstitutes is particularly ironic since EDB itself was an approved replacement for another suspended fumigant, DBCP (dibromochloropropane). Table A-1 provides a summary chronology on EDB regulation.

Table A-1. Summary of background on EDB regulation.

1925	EDB is first used as a pesticide.
1927	First reports of EDB's toxicity in exposed animals.
1947	Congress passes FIFRA.
1948	Dow Chemical Co. registers EDB as a pesticide with USDA.
1949	EDB is registered for use as a soil fumigant.
1956	EDB is registered as a fumigant for stored grain, fruits and vegetables.
1956-58	FDA sets tolerances and exemptions from tolerances for inorganic bromide residues resulting from use of EDB on a variety of fruits, vegetables, and grains.
1970	EPA is created.
1972	FIFRA amended.
1973	Reports show EDB causing mutations and reproductive damage in animals.
1974	NCI issues a "Memorandum of Alert", identifying EDB as a potential carcinogen.
1975	July. EDF petitions EPA under FIFRA to investigate the carcinogenic potential of EDB pesticides and to either suspend or cancel their registrations.
	NCI reports that EDB should be considered a human carcinogen based on animal study results.
1977	EPA issues Position Document (PD) 1, stating the agency's rationale for the Rebuttable Presumption Against Registration of EDB.
	NIOSH (National Institute of Occupational Safety and Health) study concludes EDB is mutagenic.
	OSHA (Occupational Safety and Health Administration) issues guidelines to limit occupational exposures to EDB.

Table A-1. Summary of background on EDB regulation (cont'd).

1978	FDA begins to monitor EDB residues in food.
1979	NIOSH recommends that OSHA reduce permissible workplace exposures from 20 ppm to 130 ppb.
	EPA suspends another fumigant, DBCP, for all uses except Hawaiian pineapple.
1980	To replace DBCP, EPA grants emergency approval for soil fumigation with EDB for soybeans.
	Carter Administration EPA issues a PD 2/3, a proposal to end EDB's uses as a fumigant for grain and grain milling machinery. Use as a citrus fumigant would end on July 1, 1983. Other uses would be continued but on a restricted basis.
1981	Levels in some USDA/EPA wheat samples reached 4200 ppb.
	FIFRA SAP concurs with EPA's proposal to cancel EDB uses.
	Medfly (Mediterranean Fruit Fly) crisis.
	Reagan White House Office of Science and Technology Policy (OSTP) chairs interagency task force to review EDB problem.
1982	Reagan appointee John Todhunter, Assistant Administrator for OPTS sits on EDB proposal.
	Groundwater residues of EDB, from soil injection in citrus fields, detected in Georgia, California, and Hawaii.
1983	March. Administrator Anne Gorsuch-Burford, Todhunter, and other high-ranking EPA officials resign.
	Florida and California suspend use of EDB as soil fumigant.
	September. Congressional hearing on EDB.
	September. EPA releases PD4, declaring an emergency suspension of EDB as a soil fumigant for agricultural crops, and a cancellation proceeding against all other pesticide uses of EDB.
	October. EPA issues *Federal Register* notice revealing its intent to cancel registrations of pesticide products containing EDB, except use as a postharvest citrus fumigant which may continue until September 1, 1984, to allow time for alternatives to be found.
	November. SAP meeting with OPP representatives to discuss alternatives to EDB.
	December. Florida Agricultural Commission reports that residues of EDB are in numerous grain-based products and starts a product embargo.
1984	February. EPA announces emergency suspension of EDB as a fumigant for grain and grain milling machinery.
	June. SAP meets to review the emergency suspensions of grain fumigation uses of EDB, discusses EPA's lack of attention to the hazards of probable alternatives to EDB.
1990	Stratospheric ozone depletion potential of methyl bromide recognized.
	California suspends Telone, a soil fumigant containing 1,3-dichloropropane (1,3-D) after the state's Air Resources Board detects 1,3-D 'levels of concern' in ambient air.
1993	EPA announces phaseout of methyl bromide under the Clean Air Act.
1994	California allows restricted use of Telone.

2. Scientific Issues

EDB is a synthetic halogenated organic compound (1,2-dibromoethane). (Halogens include chlorine, bromine, fluorine, and iodine.) While chlorinated organic pesticides (e.g., DDT) show a strong tendency to accumulate in the environment and food chain, EDB was originally believed not to accumulate because of its relatively high volatility. Improvements in analytical measurement techniques (capable of detecting EDB levels at 1 ppb), however, revealed traces of EDB in food and animal feed in the 1960s

and 1970s, with EDB levels in some wheat flour samples reaching 4 parts per million (ppm) (*Fed. Reg.*, Vol. 42, pp. 63138-63139). During 1982-83, EDB was also detected in ground water in several states as a result of soil fumigation, with levels varying from 0.02 parts per billion (ppb) to more than 100 ppb (*Fed. Reg.*, Vol. 48, p. 46230). According to an EPA official, "the biggest leap in scientific understanding" regarding the risks of soil fumigants was their discovery in groundwater, a discovery that started with DBCP.

Between 1972 and 1974, an NCI-sponsored cancer study was conducted that concluded that EDB was very carcinogenic in rats and mice. Moreover, the tumors were observed to begin after a short time compared to the results of tests of other chemical carcinogens. Critics of the study, however, pointed out what they viewed as its inadequacies. First, the cancers were formed in the forestomach of the rodents, and though humans have an esophagus, they do not have a forestomach. Second, the EDB was administered to the rodents by intubation into the stomach. To apply the results to exposures to farm workers via inhalation and dermal absorption required extrapolating from one exposure route to another. However, EDB was also found to be a carcinogen in a variety of species of laboratory animals when the chemical is administered by inhalation, skin painting, and intubation into the stomach. A number of studies, including a 1977 NIOSH study, also found EDB to be mutagenic, reinforcing the evidence of EDB's carcinogenic potential across species. Evidence showing that EDB is mutagenic to germ cells (e.g., sperm), also suggested the possibility of human genetic damage. Animal husbandry researchers in the 1960s and 1970s found that ingested or injected EDB caused spermicidal and other infertility effects in rams and bulls, and in 1978, an EPA contractor produced a report demonstrating that inhaled EDB resulted in reduced sperm counts in rats (EPA 1983).

There were no strong epidemiological studies of any adverse health effects of EDB in humans. But to give the reader an indication of EDB's cancer potency estimated by EPA from the animal studies, EPA calculated that the workers most highly exposed to the pesticide (warehouse workers who over 40 year working lives handle agricultural commodities that are fumigated postharvest for pest quarantine) could have a lifetime risk of cancer as high as 2 in 10 (2×10^{-1}) (*Fed. Reg.*, Vol. 48, p. 46237).

3. Process within EPA

Setting the Agenda

The NCI animal study was sufficient to push EDB into the RPAR process, and the EDF petitions may have forced EPA to issue its regulatory proposals in 1980. But the suspension of DBCP (for which EDB was a substitute), the Medfly infestation in California, and federal pesticides policy during the first years of the Reagan administration combined to prevent further action on EDB. According to current and former EPA officials, the detection of EDB groundwater contamination in 1982-83 provided the impetus for proceeding with the suspension of soil fumigation uses, and Todhunter's

resignation in March 1983 removed the principal internal obstacle. The scope of the problems associated with soil fumigation were geographically limited, however. The safety of the nation's food supply was placed onto the regulatory agenda by Florida's actions regarding EDB residues in grain products which ignited a public panic.

Assessing the Science

Six out of seven interviewees responded that there was adequate science to inform EPA's decision to suspend EDB registrations in 1983-84. The data on human exposure to EDB were considered particularly abundant. According to a former SAP member, it was one of the more "cut and dried" pesticides reviews in terms of the availability of scientific information. Respondents characterized the scientific uncertainty at the time of the 1983-84 suspensions as moderate-to-very small. Areas of scientific uncertainty identified by respondents were an incomplete understanding of noncancer effects and of the risks of substitutes for EDB. Interviewees agreed that EPA's final treatment of the available science was good-to-very good.

Under the Rebuttable Presumption Against Registration process, EPA laid out its scientific and economic analyses in a series of Position Documents (PDs) prepared by a working group managed by the Office of Pesticide Programs' Office of Special Pesticide Reviews (OPP/OSPR). PD 1, issued in December 1977, stated the agency's rationale for placing EDB in the RPAR process. PD 2/3, issued in December 1980, responded to information submitted in rebuttal, reported the review's findings, and proposed regulatory action. PD 4, issued in September 1983, summarized the data, public commentary, and scientific review and announced a final regulatory decision.

PD1: The primary basis for the EDB RPAR review was the NCI rodent study, although it was only available in 1977 in manuscript form. OPP was also able to take advantage of a 1977 NIOSH criteria document (produced by a contractor) assessing the available EDB health effects information. The EPA Office of Research and Development Cancer Assessment Group (ORD/CAG) conducted a quick review of summary data from the NCI study and concluded that EDB caused a significant increase in the incidence of stomach cancer in rodents and that the tumor rates appeared high. A review of the EDB mutagenicity studies was conducted by an OPP contractor and supplemented by the OPP Criteria and Evaluation Division (OPP/CED). The EDB working group performed a review of the EDB reproductive effects studies, and the EPA Office of Toxic Substances (OTS)[3] contracted for a 1976 study of the developmental toxicity of EDB inhaled by rodents. OPP/CED conducted some preliminary EDB exposure assessment based on available literature, which included studies by the National Institutes of Health (NIH) and industry contractors. The exposure assessment looked at exposures to EDB applicators, residues on raw agricultural commodities, and residues in grain products, but did not consider the possibility of groundwater contamination.

[3] OPP and OTS are both under the Assistant Administrator for Pesticides and Toxic Substances.

PD 2/3: In the 1980 PD 2/3, EPA responded to an accumulation of rebuttal comments questioning the validity of the NCI study, the mutagencity studies, and EPA's exposure analyses. In addition to the information provided by the rebuttals, EPA was authorized under FIFRA to require registrants to provide much of the data used in the analysis. The CAG analyzed the rebuttals regarding the carcinogenicity of EDB. Two studies issued since the PD 1, one of which was by the NCI, showed that inhaled EDB produced a significant increase in rodent tumors. The agency reviewed a number of new studies that supported the earlier findings of EDB mutagenicity. The agency rejected as technically flawed two human epidemiological studies submitted by the Ethyl Corporation in rebuttal to the PD 1 analysis of reproductive risks. The agency also sponsored an epidemiological reproductive study that found no effects but considered it of limited significance due to its small size. Using data submitted to the agency, OPP conducted a more extensive assessment of human exposures to EDB. Residues of EDB were detected in flour and bread, on fruits and vegetables, in air inhaled by exposed workers and from skin contact with treated commodities.

EPA estimated that the range of cancer risks to people exposed occupationally or through dietary exposures was from moderate (one in one hundred thousand, 10^{-5}) for the general population to very high (one in ten, 10^{-1}) for some agricultural workers. The agency also concluded qualitatively that EDB posed mutagenic and reproductive risks to exposed humans. Weighing these health risks against the benefits of continued registered uses of EDB and the availability of alternative pesticides, the agency proposed to cancel immediately use of EDB on stored grain, flour mill machinery, and felled logs; to cancel use of EDB for postharvest fumigation of citrus and tropical fruits as of July 1983; and to allow continued but restricted use for soil fumigation and other uses (*Fed. Reg.*, Vol. 45, p. 81516).

SAP Review of PD 2/3: The FIFRA SAP reviewed the EDB PD 2/3 in 1981. In addition to briefings from OPP/SPRD staff and an EPA reproductive biologist, the panel received statements from representatives of the USDA; the State of Florida, Department of Citrus; the Hawaii papaya industry; Ethyl Corporation; Dow Chemical Corporation; corporations recommending irradiation as an alternative to EDB for citrus fumigation; National Association of Wheat Growers; National Pest Control Association; academics; and others. The SAP concurred with the agency's decision to cancel the registration for stored grain, flour mill machinery, and felled logs. However, the SAP considered the phaseout of citrus fumigation to be unnecessary, as well as impracticable, due to the uncertain feasibility of irradiation to control fruit flies on citrus. Instead, the panel recommended additional restrictions on this registered use, including increased protection for exposed workers. That panel also recommended that prior to final rulemaking, EPA require additional data and analysis of exposures resulting from the use of EDB for flour mill fumigation and EDB residues in grain and grain products, monitoring of highly exposed workers in the grain, citrus, and minor use areas, and an additional rodent reproductive study. Finally, the panel also indicated its concern about the occurrence of EDB in groundwater and urged that groundwater be monitored closely in high use areas (*Fed. Reg.*, Vol. 48, pp. 46242-46244.)

Todhunter's "Reassessment" of EDB: John Todhunter served as Assistant Administrator of OPTS from 1981 to March 1983. Disinclined to proceed with the EDB regulatory actions proposed by the outgoing Carter administration, Todhunter produced his own "alternative" EDB risk assessment which downplayed the risks of the pesticide. According to an EPA official who served on the EDB working group, Todhunter's analysis consisted of "back of the envelope calculations." Edwin Johnson, Director of OPP, testified to Congress that Todhunter's reassessment of EDB was "an example of Todhunter playing scientist...[and he] used poor methods" (cited in Lash 1984). According to Lash (1984), Todhunter had also asked EPA statistician Anne Barton to alter her risk estimates for EDB using an obscure scientific theory she had never seen before (or after). Todhunter's EDB reassessment conveniently presumed that risk levels declined exponentially (instead of linearly) with decreasing exposure time (Walsh 1982, p. 1595). An environmentalist reports that Todhunter compared the risk of EDB to that of smoking one cigarette in a lifetime. In 1983, prior to his resignation, Todhunter requested the SAP to re-review the EDB regulatory package (Jasanoff 1990).

Todhunter's penchant for conducting his own re-analysis was not limited to EDB. His highly controversial analysis of formaldehyde, which the OTS identified in 1981 as a candidate for priority attention under TSCA, deviated from carcinogen risk analyses previously performed at EPA and rested on a number of questionable assumptions (Jasanoff 1990, pp. 197-198). It was in the wake of these episodes that the National Research Council recommended that EPA develop formal risk assessment guidelines to ensure methodological consistency (NRC 1983).

PD 4: Citrus growing states began reporting detection of EDB in groundwater in 1982, and the policy vacuum at EPA was filled by the states until Todhunter resigned in March 1983. During the spring and summer of 1983, under the new Ruckelshaus administration, the EDB working group worked feverishly to update the EDB regulatory package in light of the new data and to respond to comments on the EDB PD 2/3. The final EDB team, lead by SPRD's Rick Johnson, included 19 EPA staff with training in statistics, toxicology, chemistry, entomology, plant pathology, reproductive biology, law and economics (EPA 1983).

Communicating the Science to Agency Leadership

Three respondents characterized the communication of the scientific information to agency decisionmakers as very good. Due to the high stakes and public visibility, the decisionmakers were "remarkably and unusually interested in the details," says one EPA official. According to this source, Administrator Ruckelshaus was briefed on EDB by the review team manager two to three times a month during 1983 and 1984.

A former senior EPA official says that the issue was first raised to the new EPA leadership when Don Clay, the Acting Assistant Administrator of OPTS, requested a meeting with the new Deputy Administrator, Alvin Alm. At this 1983 meeting, OPP

presented the toxicological evidence on EDB, saying that "they had never seen a substance that caused so many tumors so quickly" in laboratory animals. The leadership was also impressed by the hard data that the agency had assembled on exposure to EDB. The staff recommended that on the basis of the new data on EDB groundwater contamination, the agency should immediately suspend the use EDB as a soil fumigant to control nematodes. Alm questioned whether the agency should also suspend EDB use as a grain fumigant, but Clay convinced Ruckelshaus and Alm not to do so due to the lack of data on dietary exposure from grain products.

4. Science in the Final Decisions

The suspension of EDB's use as a soil fumigant was driven by scientific information. But the suspension of EDB for use to treat stored grain and milling equipment was dictated "purely by economics and public hysteria."

According to a former senior EPA official, the 1983 emergency suspension of EDB as a soil fumigant was driven by scientific information. "The science was adequate for the suspension--it was not a hard call," says this source. But the 1984 emergency suspension of EDB for use to treat stored grain and for treating grain milling equipment was dictated "purely by economics and public hysteria." The grain product bans and inconsistent EDB residue tolerance levels being enacted by the states were disrupting the market, causing economic losses, and creating concerns about the safety of U.S. grain exports. A former EPA political appointee recalls that the EDB grain suspension was " unlike any regulatory action [he] ever dealt with in that the people who normally opposed regulation were pushing hardest for regulation.... USDA, the Grocery Manufacturers Association, and the food industry all demanded EPA action. They felt that grain and other food supplies were threatened by public panic if EPA didn't take charge and calm people's fears. [There was] something like $3 billion worth of stored grain. The Russians threatened not to buy U.S. grain because of EDB" and the Japanese needed high-level assurances that U.S. grain was safe. Most of the states, according to this source "were glad to be bailed-out" by EPA's decision.

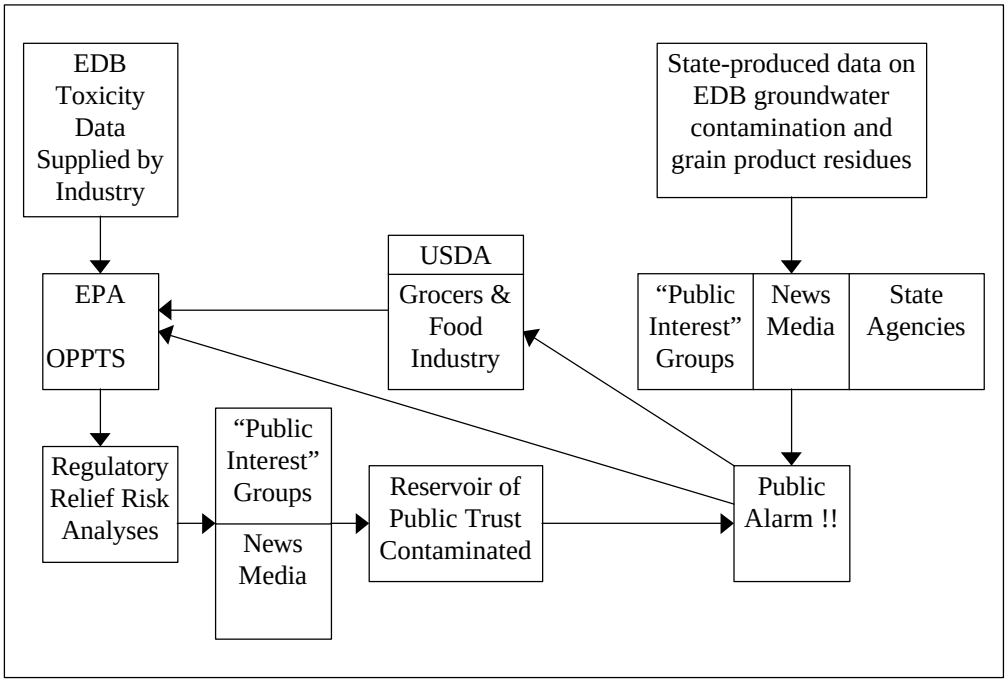
5. Concluding Observations

The story of EDB suggests the power of new scientific data--the occurrence of EDB in groundwater--to break through a logjam erected by a reluctant regulator--Assistant Administrator Todhunter. In terms of a fate and transport analogy, the data produced outside EPA on the occurrence of EDB in groundwater and consumer grain products were released into the public domain beginning in 1982 prior to Todhunter's resignation. EPA had no control over the data and lost control over interpretation of the data. The fact that the states got out ahead of EPA on the issue in reaction to the new data makes it seem likely that the agency would have been forced to take regulatory action even if there had been no turnover in EPA's political leadership. It should be underscored that the new findings could not have been completely unexpected, since the SAP strongly urged EPA to require groundwater monitoring in 1981. EDB also illustrates the downside

of EPA losing control over the regulatory agenda. Cross (1989) concluded that while EPA eventually regulated the carcinogenic hazard from EDB, agency action was unnecessarily slow and final EPA restrictions may have been unnecessarily stringent.

A former senior EPA official believes that although Todhunter deserves a fair amount of criticism for his role in the EDB decision, the culpability extends beyond the former Assistant Administrator. "There was dawdling within the agency. The [EDB toxicity] data that the agency acted on was in the agency before [Todhunter took office]. They're giving one individual an awful lot of credit." Whether or not agency staff contributed to the slow pace of the EDB review, however, Todhunter was the politically accountable decisionmaker. Furthermore, to continue the fate and transport analogy, EPA's reservoir of public trust was contaminated by Todhunter's track record for cooking the data to provide regulatory relief. The agency had no public credibility to portray the dietary risks of EDB in an objective manner or call for more time to evaluate the public health risks (as Clay had counseled Alm and Ruckelshaus). The news media were handed a juicy storyline that included unseemly official conduct, bumbling bureaucrats, and horrific public health threats contained in something as familiar as a bread wrapper. Those with competing agendas filled the vacuum, whipping the public into a frenzy and further eroding trust and confidence in public institutions. These dynamics are illustrated in Figure A-1.

Figure A-1. Fate and Transport of Science in the EDB Decisions



Graham (1991) cites EDB as an example of the credibility problem exacerbated by political appointees in the Reagan administration who pursued a policy of "regulatory relief." Because early Reagan-era EPA policymakers could not be trusted to resist

adjusting the science to meet their policy preferences, the agency spent the next decade developing formal guidelines for risk assessment that have now come to be viewed by some EPA critics as inflexible and overly simplistic. These episodes also fed the perception that there should be a firewall between risk assessors and risk managers within the agency. With the growing recognition of the value of participation by decisionmakers and stakeholders to help guide judgments made in the risk analysis process (e.g., regarding the scope and intended use of the analysis and what environmental or health effects are of concern), the virtue of a rigid distinction between risk assessment and risk management is increasingly being called into question. (See, for example, NRC 1996.)

The case of EDB also indicates the unintended consequences of considering a pesticide and its alternatives independently.

The case of EDB also indicates the unintended consequences of considering a pesticide and its alternatives independently and the need in regulating pesticides to thoroughly consider the risks of substitutes as well as other risks that may be created unintentionally by regulatory action. Because the 1983-84 EDB suspensions were issued on an emergency basis, the SAP did not review the decisions until after they were final. When EPA presented its decisions to the panel in 1983 and 1984, SAP members cautioned the agency about the lack of knowledge concerning the health effects of alternative nematocides (principally Telone) and grain fumigants (primarily methyl bromide). EDB, the panel noted, had itself replaced the canceled pesticide dibromochloropropane (DBCP) as a soil fumigant for soybeans in 1980. Would Telone be next? Would methyl bromide, which a Dutch study had found to be an animal carcinogen, cause the next spasm of public hysteria? (Jasanoff 1990). An EPA official explains what the SAP member recognized--that nematocides are simply "a nasty group" of pesticides.[4] "To be efficacious, they [nematocides] need to leach down into the soil, and they are very toxic as a group," says this official. After suspending permitted uses of Telone in 1990, California in 1994 allowed the use of Telone as a soil fumigant to continue on a restricted basis. According to a statement by James Wells, Director of the California EPA Department of Pesticide Regulation, restricted uses of Telone were reinstated because "farmers are trying to cope with a diminishing number of pest management tools...this problem is especially critical with soil fumigants...[and] limited use under strict conditions does not pose a significant risk and can be of benefit to the state's agricultural economy" (http://www.cdpr.ca.gov/docs/archives/pressrls/1994/94-42.arc).

However, the types of risk issues that the SAP raised in 1983-84 were the familiar risks (e.g., cancer) of alternative pesticides. A substitute risk that nobody seems to have considered at the time regarding EDB's use as a grain fumigant was that of aflatoxin, a very potent animal carcinogen produced by a fungus on corn and peanuts that was controlled by EDB (Gray and Graham 1995). EPA also failed to foresee the unconventional environmental effects that environmental releases of EDB substitutes could have. The agency, for example, responded to the SAP's concerns about methyl bromide by indicating that since it was more volatile than EDB, it could be expected to

[4] Methyl bromide has also been used as a soil fumigant, but its greater volatility requires covering fields with tarps to ensure penetration sufficient to control nematodes.

pose fewer risks of contaminating the food chain or the environment (Jasanoff 1990). However, an independent risk analyst notes that the higher volatility of methyl bromide results in greater transport of the halogen bromine into the upper atmosphere, or stratosphere, than would result from use of EDB. Therefore, methyl bromide poses a greater threat to stratospheric ozone than does EDB, resulting in a greater risk of environmental and health effects from unfiltered ultraviolet radiation.

The "Molina and Rowland" hypothesis of stratospheric ozone depletion by the halogen chlorine was first published in 1974 (Molina and Rowland 1974). After the Antarctic ozone hole was discovered in 1985 and grew unexpectedly in 1987, the Montreal Protocol was signed in September 1987. According to an EPA official, within three years, researchers had a good understanding of the contribution of methyl bromide to stratospheric ozone depletion. In December 1993, EPA announced that the production and importation of methyl bromide would be phased out by 2001.

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LIST OF ABBREVIATIONS

CAG	Cancer Assessment Group
CED	Criteria and Evaluation Division
DBCP	Dibromochloropropane
DDT	Dichlorodiphenyltrichloroethane
EDF	Environmental Defense Fund
EPA	Environmental Protection Agency
FDA	Food and Drug Administration
FFDCA	Federal Food Drug and Cosmetic Act of 1938
FIFRA	Federal Insecticide Fungicide and Rodenticide Act of 1972
NCI	National Cancer Institute
NIH	National Institute of Health
NIOSH	National Institute of Occupational Safety and Health
OPP	Office of Pesticide Programs
OPTS	Office of Pesticides and Toxic Substances
ORD	Office of Research and Development
OSHA	Occupational Safety and Health Administration
OSPR	Office of Special Pesticide Reviews
OTS	Office of Toxic Substances
PD	Position document
RPAR	Rebuttable Presumption Against Registration
SAP	Scientific Advisory Panel
SPRD	Special Pesticide Review Division
TSCA	Toxic Substances Control Act of 1976

B. The 1989 Asbestos Ban and Phaseout Rule under TSCA

1. Background

Asbestos is a commercial term applied to a class of naturally occurring fibrous minerals. Asbestos has been called a "miracle fiber" due to its many useful properties (Peck 1989). Products containing asbestos are naturally flexible, heat resistant, non-combustible, chemically inert, and possess high tensile strength. Asbestos-containing products include: asbestos cement pipe, flooring products (e.g., tiles), paper products (e.g., padding), friction materials (e.g., brake linings and clutch facing), roofing products (e.g., tiles), fire retardant/fireproof insulation, and coating or patching compounds (NRC 1984). About 30 million tons of asbestos were used in the U.S. between 1900 and 1980, and as of 1986, when EPA proposed an asbestos ban, hundreds of products were still being made with asbestos (EESI 1996).

Health effects from exposure to asbestos have been recognized in the U.S. since the turn of the century, and about 65,000 Americans are now estimated to suffer from asbestosis, a chronic lung disease that makes breathing progressively more difficult (EESI 1996).[5] The majority of asbestos-related disease incidence has been traced back to occupational exposures to miners, shipbuilders, insulators and factory workers (NRC 1984). Asbestos first gained broad notoriety in the U.S. in 1964 when Dr. Irving Selikoff of Mt. Sinai Hospital and colleagues issued a landmark paper on high rates of mesothelioma and other cancers in New York City insulation workers exposed to asbestos. The study, which was presented at the annual meeting of the New York Academy of Science, "blew the asbestos issue out of the quiet circles of scholarly study and into the public consciousness," according to the *Boston Globe* (1/18/83). During the 1970s, concern mounted among the general public about potential health effects resulting from non-occupational exposures to asbestos. Production and use of asbestos in the U.S. has fallen dramatically since it peaked in the late 1960s and early 1970s.[6] Product liability ("toxic tort") litigation against domestic asbestos product manufacturers has played a significant role in the industry's decline since 1973.[7]

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[5] Asbestosis was recognized as long ago as 1907(Hodgson *et al*. 1988).

[6] Peak U.S. production reached over 136,000 metric tons annually, with imports at approximately 680,000 metric tons per year. By 1993, domestic production had dropped to an estimated 15,000 metric tons, and imports were done to 33,000 metric tons. Historically, asbestos was produced in the U.S. in California, Arizona, North Carolina and Vermont. During the 1970s, many companies in these areas discontinued mining operations. By 1991, only 2 U.S. firms produced asbestos in Vermont and California. Ninety-eight percent of U.S. asbestos imports have come from Canada. Largely as a result of the decline in U.S. asbestos consumption, the Canadian asbestos mining industry has declined considerably (ATSDR 1995).

[7] According to Cross (1989), until 1973, plaintiffs had little success in recovering damages for occupational asbestos-induced diseases. A modest success in that year, however, marked a key turning point that "opened the floodgates of litigation." In 1982, due to the large number of outstanding injury claimed filed against it, Johns-Manville, the major producer of asbestos in the U.S. was forced into bankruptcy (Bates 1994 and Cross 1989). An academic mineralogist suggests that EPA's 1989 asbestos ban and phaseout rule was intended to halt much of the forecasted litigation and compensation claims regarding asbestos-related cancers and disease.

After five years of development and debate, Congress passed the Toxic Substances Control Act (TSCA) in 1976 to address non-pesticidal toxic chemicals in commerce.[8] According to a source involved in negotiations over the bill, TSCA evolved specifically with substances like asbestos in mind.[9] TSCA was designed to provide a "life-cycle" framework for management of chemical risks, taking into account risks from product manufacturing, use, and disposal and exposures associated with the air, water, and land (Shapiro 1990). TSCA requires pre-manufacture evaluation of most new chemicals (under Sec. 5)[10] and allows EPA to regulate existing chemical hazards (under Sec. 6). The risk management tools available to EPA for existing chemicals range from labeling standards to outright bans.

TSCA is unlike most other pollution control statutes (with the exception of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA)) in that it primarily regulates commercial products, not wastes, and therefore acknowledges the many benefits of commercial chemicals. Most environmental statutes require decisions to be based upon precautionary margins of safety or the availability of pollution control technology and seek to limit explicit consideration of economic factors. In contrast, to enact regulatory controls under TSCA, EPA must have a "reasonable basis" for finding that a toxic substance poses an "unreasonable risk" to health or the environment and must adopt the "least burdensome" control option which the agency is authorized to require to limit the risk to an acceptable level. This "unreasonable risk" provision has been interpreted as requiring EPA to balance the costs and benefits of proposed regulatory decisions, taking into account the availability of substitutes and other adverse effects (e.g., the risks posed by substitutes) which the regulation may have (Shapiro 1990).[11] TSCA is also unlike other environmental statutes in that EPA is subject to the "substantial evidence" standard of judicial review (under Sec. 19). By comparison, under other statutes, EPA must meet the less demanding "arbitrary and capricious" judicial review standard. Therefore, TSCA is formulated to require EPA to provide a considerable weight of evidence and regulatory analysis to document its regulatory decisions and defend them under judicial review.

In 1979, EPA formally announced its intent to pursue a complete ban of all uses, manufacturing, mining and importation of asbestos under TSCA Section 6 (*Fed. Reg.*, Vol. 44, p. 60061).[12] The international asbestos industry and, according to a former

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[8] In 1971, the President's Council on Environmental Quality issued a report on toxic substances and recommended comprehensive toxic substances control legislation (TSCA Research Project 1982).

[9] Peck (1989) corroborates that asbestos was salient in TSCA's development. Congress also directly banned the manufacture, processing, and distribution of polychlorinated biphenyls (PCBs) when it wrote TSCA (Shapiro 1990).

[10] Food, food additives, drugs, pesticides, alcohol, and tobacco are exempted from TSCA.

[11] The House Report on TSCA specifically states that a quantitative cost-benefit analysis is not required to show unreasonable risk (Shapiro 1990).

[12] In 1979, EPA granted a public petition under TSCA (Sec. 21 allows individuals to "petition EPA to initiate a proceeding for the issuance, amendment, or repeal of a rule) to prohibit the future use of asbestos cement-pipe in water systems (*Fed. Reg.*, Vol. 44, p.60155).

senior EPA official, the Canadian government lobbied vigorously against the proposal,[13] and there were years of conflict between EPA and the Office of Management and Budget (OMB) over the rule (EESI 1996). Considerable controversy was generated in 1985 when EPA tentatively referred asbestos to the Occupational Safety and Health Administration (OSHA) and the Consumer Product Safety Commission (CPSC) and later reversed its decision. (According to a former senior EPA official, OMB raised objections that the rule had not been coordinated with OSHA and then-EPA Administrator Lee Thomas pulled the rule back from OMB.[14]) In 1986, seven years after EPA announced its initial intent, the agency proposed a rule to ban asbestos (*Fed. Reg.*, Vol. 51, p. 3738). It took another three years (during which EPA held informal rulemaking hearings and developed or updated several of the major supporting documents) before the rule was finalized in 1989 (*Fed. Reg.*, Vol. 54, p. 29467). The rulemaking prohibited (in three staged intervals) the future manufacture, importation, processing, and distribution in commerce of virtually all asbestos products by 1997.

EPA's 1989 asbestos ban and phaseout rule was based on one of the longest (dating back to the 1940s) and largest substance-specific human health databases ever amassed.[15] When OSHA established its occupational standard for asbestos in 1986, it stated, "OSHA is aware of no instance in which exposure to a toxic substance has more clearly demonstrated detrimental health effects on humans than has asbestos exposure" (*Fed. Reg.*, Vol. 51, p. 22612). EPA's rulemaking procedure was also one of the most extensive in the agency's history (Carnegie Commission 1993). According to an EPA official, the agency spent approximately $10 million on the regulation's cost-benefit analysis alone. In the judgment of EPA Administrators Lee Thomas and William Reilly and Assistant Administrator for Pesticides and Toxics John Moore, the health database and the agency's analysis provided substantial evidence that asbestos posed an unreasonable public health risk. In 1991, in deciding the case of *Corrosion-Proof Fittings v. EPA* (947 F.2d 1201 (5th Cir. 1991)), the U.S. Fifth Circuit Court of Appeals ruled otherwise, concluding that EPA had not presented sufficient evidence to justify banning, for all practical purposes, all commercial uses of asbestos.

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[13] The Asbestos Information Administration in Washington, DC was involved in major lobbying efforts against the ban (Peck 1989). Canada also strongly opposed proposals to ban asbestos in the U.S. It financed lobbying efforts of the Asbestos Institute (based in Montreal). In 1989, the Institute was receiving two-thirds of its $5 million budget from the federal government in Ottawa and the Province of Quebec (the center of Canada's asbestos mining industry).

[14] TSCA Sec. 9 requires EPA to refer chemicals to another agency with regulatory authority, if that agency can manage the associated risks. Most non-occupational asbestos exposure, however, resulted from the normal use of auto brakepads and was beyond the purview of OSHA and CPSC. (The 1991 court decision which overturned EPA's 1989 asbestos ban and phaseout rule did find that it was appropriate for EPA to regulate asbestos under TSCA.) According to press reports, the OMB blocked EPA's proposed asbestos ban in 1984, but congressional pressure later overrode OMB in 1985 (*Inside the EPA* 2/8/85; *Inside the EPA* 3/15/85).

[15] Long-term databases are often needed to accurately assess chronic health effects which may take decades to manifest.

The court justified its decision by finding that EPA had failed (1) to give public notice of its intended use of "analogous exposure" data to calculate expected regulatory benefits and (2) to promulgate the least burdensome, reasonable regulation required to provide adequate protection. Arguing that the agency bears a heavier burden when it seeks a ban of a substance than when it merely seeks to regulate that product, the court held that the rule was not promulgated on the basis of substantial evidence as required under TSCA. The court delved into substantive issues in which administrative agencies are traditionally granted judicial deference, discussing at considerable length what it regarded as shortcomings in EPA's cost-benefit analysis and in the agency's analysis of the health and safety risks posed by asbestos substitutes. The court also considered *amicus curiae* briefs relating to scientific details such as toxicological differences in asbestos fiber types and sizes. The court held that EPA's failure to more thoroughly consider the risk of asbestos substitutes "deprives its order of a reasonable basis" because "EPA cannot say with any assurance that its regulation will increase workplace safety when it refuses to evaluate the harm that will result from the increased use of substitute products." The court concluded that "eager to douse the dangers of asbestos, the agency inadvertently actually may increase the risk of injury Americans face." But what ultimately seemed to drive the decision was that the judges felt that the estimated cost of $30-$40 million per life saved by the asbestos ban was unreasonable. The court stated, "the EPA, in its zeal to ban any and all asbestos products, basically ignored the cost side of the TSCA equation."

The Department of Justice elected not to appeal the decision. The following year, a *Federal Register* notice stated that, in a subsequent clarification of its decision, the court had allowed EPA's 1989 rule to continue to govern "new" uses of asbestos in products that were not being manufactured, imported or processed on July 12, 1989 (*Fed. Reg*., Vol. 57, p. 11364). In 1994, EPA issued an amended rule that bans "new uses" of asbestos. With a few exceptions, products containing asbestos originally manufactured before July 12, 1989 can still be made today, but any asbestos products made thereafter are now banned (EESI 1996). Prior to the *Corrosion-Proof Fittings* decision, Shapiro (1990) observed that TSCA had resulted in less regulatory activity than was expected when the law passed. Many observers now interpret the 1991 decision as paralyzing EPA's use of TSCA Section 6 regulatory powers (e.g., Mazurek *et al*. 1995).[16]

Over the past 50 years, industry guidelines for "safe levels" of exposure to asbestos in the workplace have given way to federal regulation of asbestos in a variety of settings. In 1946, the American Council of Governmental and Industrial Hygienists (ACGIH) established a non-regulatory occupational standard of 5 million particles/ft^3 (approximately 177 fibers per milliliter (f/ml)). In its first attempt to regulate a substance as a carcinogen, OSHA set the federal regulatory workplace standard for asbestos at 5 f/ml in 1972. The agency subsequently lowered the standard, most recently to 0.1 f/ml in

[16] During the first fifteen years of the TSCA program, production, manufacture, importation and use of only 5 chemical substances have been restricted: PCB's (directly by Congress), CFC's in aerosols, nitrates in metal-working fluids, dioxin (disposal requirements), and asbestos (inspection and removal from schools) (Mazurek *et al*. 1995).

1994 (Bates 1994 and ATSDR 1995).[17] One year later, in the first regulatory action addressing non-occupational asbestos exposure, EPA listed asbestos as the first hazardous air pollutant under the Clean Air Act (Sec. 112). At the time, EPA rejected a proposed asbestos ban primarily for economic reasons.[18] In 1978, EPA banned all spray applications of asbestos (especially popular as a fire retardant/fireproof insulation in the 1950s during the post-war building boom) (Bates 1994).

During the years leading up to *Corrosion Proof Fittings*, the public debate over EPA's ban and phaseout was overshadowed by the more politically visible controversy over the occurrence and removal of asbestos from schools and other public buildings. Of all forms of indoor air pollution, asbestos has probably received the greatest publicity. During the 1980s, a flurry of federal statutes and regulations were enacted to address the occurrence and removal of asbestos in schools and other public buildings (Bates 1994).[19]

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[17] The OSHA standard applies to fibers longer than 5 micrometers and is averaged over 8 hours (gopher://ecosys.drdr.Virginia.EDU:70/00/library/gen/toxics/Asbestos). According to Cross (1989), OSHA's occupational standard was based on technical feasibility, the agency conducted no quantitative risk assessment for asbestos, and the rule was accompanied by very little explanation. Industry successfully contested the 1972 rule in *Industrial Union Department, AFL-CIO v. Hodgson* (D.C. Cir. 1974). OSHA amended the 1972 standard in 1986 (*Fed. Reg.*, Vol. 51, p. 22612, cited in OTA (1995)).

[18] In 1973, the EPA set a "no visible emissions" standard for asbestos air emissions from five major sources. At the time, EPA declared it impossible to prepare a quantitative risk assessment for airborne asbestos exposures. EPA gave no basis for its use of cost considerations under Section 112, nor did the agency explain how continued emissions of carcinogenic asbestos could be squared with the mandate to provide an ample margin of safety to protect public health (Cross 1989).

[19] In 1981, the US Department of Education promulgated a rule under the Asbestos School Hazard Detection and Control Act, but no funds were appropriated. In 1982, EPA promulgated a rule under TSCA requiring identification and public notification of asbestos in schools. The agency issued its first guidance for controlling asbestos in school buildings (the "Blue Book") the following year. In 1984, Congress enacted the Asbestos School Hazard Abatement Act ,establishing grants and interest-free loans for school asbestos projects. The same year, EPA established regulations under the Clean Air Act governing the removal of asbestos from buildings and disposal of wastes generated from removal (*Fed. Reg.*, Vol. 49, p. 13658). In 1985, EPA promulgated a worker protection rule for municipal and state workers not covered by OSHA regulations (*Fed. Reg.*, Vol. 50, p. 28530). In 1985, EPA issued new guidance (the "Purple Book") for controlling asbestos in school buildings which emphasized that asbestos can often be safely managed in place instead of being removed. This created the perception that EPA was giving mixed signals about the need for regulation (Bernard 1990, p. 274). In 1986, prompted by stories of botched asbestos cleanups and the lack of action by many schools, Congress codified the asbestos in schools notification rule and required all asbestos inspection, testing, planning, and response actions be performed by accredited personnel and labs with the Asbestos Hazard Emergency Response Act (Title II of TSCA). In response to the 1985 "Purple Book," the AHERA required EPA to specify measures to reduce asbestos hazards in schools. TSCA Sec. 201(a) states that EPA's guidance "is insufficient in detail to ensure adequate responses." EPA promulgated the asbestos-containing materials in schools rule under TSCA section 203 in 1987 (*Fed Reg*., Vol. 52, p. 41826). The 1987 rule required local school systems to inspect facilities for asbestos and "select and implement in a timely manner the appropriate response actions." In adopting this regulation, EPA did not rely on a quantitative risk assessment and deflected judgments about asbestos containment and removal to local school districts. EPA's rules on asbestos in schools were upheld on judicial review (Cross 1989). In 1989, Congress approved a hotly debated extension of AHERA deadlines. The following year Congress reauthorized the Asbestos School Hazard Abatement Act (EESI 1996).

While there was initially much public outrage about children's exposure to asbestos from school building materials, the controversy soon expanded to include the question of local government financing of costly inspection and abatement procedures and the health risks posed by asbestos removal itself. Some skeptics viewed the political support for asbestos abatement as an effort to create work for construction firms who discovered a sideline during the building slump that began with the 1982-83 economic recession. After gaining some experience with asbestos abatement, it became apparent that removal was only warranted when asbestos is exposed and friable (crumbly), causing fibers to be released into the air. In many cases, it seems, asbestos abatement was doing more harm than good and existing asbestos materials could be encapsulated at lower cost *and* risk. The high cost and unintended consequences of asbestos abatement in schools and public buildings may have cast doubts--warranted or not--on the trustworthiness of EPA's judgment concerning asbestos regulation under TSCA Section 6.[20] Table B-1 provides a summarized chronology of asbestos regulation.

Table B-1. Summary chronology of asbestos regulation.

1907	Asbestosis recognized in asbestos workers.
1940s	WW II shipbuilders and pipefitters exposed to high levels of asbestos used for insulation.
1946	ACGIH recommends occupational standard of 177 f/ml.
1955	Doll links asbestos exposure with lung cancer in *British Journal of Industrial Medicine*.
1964	Selikoff *et al*. report strong link between occupational asbestos exposure and lung cancer in insulators in *Journal of American Medical Association*. New York Academy of Sciences meeting produces "the first signal event," pronouncing asbestos as serious public health issue.
1971	Council on Environmental Quality calls for toxic substance legislation.
1972	OSHA sets asbestos occupational standard of 5 f/ml.
1973	EPA lists asbestos as Hazardous Air Pollutant under Clean Air Act. First damages awarded in court for asbestos-related disease.
1974	Court overturns OSHA occupational standard for asbestos.
1976	Congress passes TSCA.
1977	CPSC bans asbestos in patching compounds and artificial fireplace logs.
1978	Dept. of Health, Education and Welfare (HEW) releases a report (the "Estimates Document") projecting 2 million deaths over the next three decades from asbestos exposure. EPA bans spray applications of asbestos.
1979	February. EPA releases its preliminary evaluation, *Exposure to Asbestos*. October. EPA announces intent to ban most remaining uses of asbestos.

[20] According to a former senior EPA official, the asbestos in schools abatement program is a classic example of legalistic decisionmaking that ignores science. In their rush to remove asbestos from the classrooms, policymakers ignored warnings that removal was likely to cause more problems, in part because there were few people who knew how to remove asbestos safely. The response from policymakers, according to this source, was "No problem. We'll fix it later. If they do it wrong, we'll sue them." An EPA official singled out former U.S. Representative Florio (D-NJ) for championing the issue of asbestos in schools. An academic suggests that the mishandling of asbestos in schools contributed to the growth in anti-regulatory public sentiments.

Table B-1. Summary chronology of asbestos regulation (cont'd).

1981	Reagan administration Executive Order 12291 requires OMB review of new regulations.
	Dept. of Education promulgates rule under the Asbestos School Hazard Detection and Control Act, no funding appropriated.
	Doll and Peto denounce HEW's asbestos "Estimates Document" as "clearly inflated" and a "confidence trick" in *Science*.
1982	EPA promulgates asbestos in schools identification and notification rule under TSCA.
	International Agency for Research on Cancer (IARC) classifies asbestos as a human carcinogen.
	Largest U.S. asbestos producer declares bankruptcy due to toxic tort liability.
1983	EPA issues initial guidance for controlling asbestos in school buildings (the "Blue Book").
1984	Congress passes Asbestos School Hazard Abatement Act.
	National Academy of Science issues *Asbestiform Fibers: Non-occupational Health Risks*.
	March. Asbestos Industry Association (AIA) argues EPA should refer asbestos to OSHA.
	July. EPA Science Advisory Board reviews *Airborne Asbestos Health Assessment Update*.
1984	December. OMB finds EPA's proposed asbestos ban not cost-effective, calls for EPA to refer asbestos to OSHA.
1985	February. EPA refers asbestos to OSHA and CPSC.
	April. Rep. John Dingell (D-MI) holds oversight hearings on EPA's asbestos referral.
	June. EPA issues new guidance for controlling asbestos in school buildings (the "Purple Book") emphasizing that asbestos can often be safely managed in place instead of being removed.
	August. EPA releases its first Regulatory Impact Analysis (RIA) for asbestos ban.
1986	Congress passes Asbestos Hazard Emergency Response Act.
	January. OMB signs-off, EPA proposes asbestos ban and phaseout.
	July and October. EPA holds informal administrative hearings on the proposed asbestos ban.
	OSHA reduces its Permissible Exposure Level (PEL) to 0.2 f/Ml.
1988	March. EPA releases updated RIA, Asbestos Exposure Assessment.
	August. Congress earmarks $2 million of EPA's appropriations for Health Effects Institute research on asbestos in buildings.
	September. EPA conducts informal hearings on proposed asbestos ban.
	December. Symposium on Health Aspects of Exposure to Asbestos in Buildings held at Harvard's Energy and Environmental Policy Center. Findings reported suggest that children in schools with asbestos containing materials are exposed to a 1 in 100,000 risk of death. This risk is characterized as far less than other indoor air health risks (EEPC 1989).
1989	Congress reauthorizes Asbestos School Hazard Abatement Act.
	EPA finalizes asbestos ban and phaseout rule.
1991	September. Health Effects Institute-Asbestos Research releases study suggesting low risk from buildings with asbestos containing materials.
	October. Fifth Circuit Court of Appeals overturns EPA's asbestos ban.
1992	Court clarifies its decision
1993	EPA announces 6 asbestos products remain subject to the 1989 ban.
1994	OSHA tightens asbestos occupational standard to 0.1 f/ml.

2. Scientific Issues

Asbestos is released into the environment from the weathering of natural deposits and the degradation and/or destruction of man-made asbestos products. Because asbestos

fibers do not evaporate, dissolve, or break down into other compounds, they accumulate in the environment over time. Small asbestos fibers can remain suspended in the air for long periods, can be carried long distances by wind or water currents before settling, and can be resuspended in the air after settling out. Inhalation of fibers suspended in the air is the most common source of human exposure to asbestos. Asbestos fibers also may be present in drinking water from erosion of natural deposits or piles of waste asbestos, from asbestos-containing cement pipes, or from filtering through asbestos-containing filters. However, in this country, concentrations of asbestos in drinking water are generally less than 1 million fibers per liter (ATSDR 1995).[21] Asbestos most commonly affects the lungs, and the diseases primarily associated with asbestos are asbestosis, lung cancer, and mesothelioma.[22]

There is an unusual amount of epidemiological evidence (both domestic and international) on occupationally-related cancers and disease arising from asbestos exposure. According to an EPA official, the link between asbestos and human health effects "was among the strongest ever seen." Evidence from animal studies confirms the carcinogenicity of asbestos. The International Agency for Research on Cancer (IARC) classifies asbestos as a Group 1 carcinogen, meaning that there is sufficient evidence of carcinogenicity in humans and animals. EPA classifies asbestos as a Group A (known) human carcinogen. In 1986, EPA estimated that about 2,560 lung cancers and mesotheliomas and an undetermined number of mortalities from asbestosis and other cancers in the U.S. would result from production of asbestos products over 15 years without EPA action under TSCA (*Fed. Reg.*, Vol. 51, pp. 3738-3759).

There was virtually no doubt in the scientific community of the hazard posed by asbestos exposure at the time of EPA's ban and phaseout rulemaking. According to an EPA official, there were four principal scientific issues remaining: the differential toxicity of different asbestos fiber types; EPA's choice of a single, no-threshold dose-response model for quantitative cancer risk assessment; the accuracy and precision of EPA's exposure analysis; and the risk of asbestos substitutes.

Fiber type

There are two major groups of asbestos minerals: serpentine (chrysotile) and amphibole (crocidolite, amosite, and tremolite). Chrysotile represents the majority (90-95%) of asbestos used commercially in the U.S., but products usually contain a mixture of chrysotile and other types of asbestos. According to an EPA official, there was considerable scientific uncertainty regarding the relationship between the length and type

[21] In some locations, however, water samples may contain 10-300+ million fibers per liter (ATSDR 1995).
[22] Asbestosis (or pulmonary fibrosis) is a respiratory disorder resulting from inhaling asbestos, characterized by scarring (fibrosis), calcification, and tumors of the lungs. Asbestosis is synergistic with smoking-related cancers (i.e., smoking and asbestos are more toxic in combination than individually). Mesothelioma is a tumor, either benign or malignant, arising from the stomach lining or membranous sacs enveloping the heart (pericardium) or lungs (pleura). Pleural mesotheliomas are most common (Hodgson *et al*. 1988).

of asbestos fibers and their potency, with some evidence suggesting that chrysotile asbestos would be less potent than amphibole forms. In what has become known as the "Amphibole Hypothesis," various commenters have suggested that chrysotile is far less carcinogenic than amphiboles, and that this distinction should be included in any asbestos risk assessment.[23]

A former senior EPA official, however, notes that epidemiological evidence from "North Carolina, where the overwhelming exposure of the population was almost exclusively chrysotile," supported the agency's position that all forms of asbestos increased relative health risks. Whereas previous British epidemiological studies had failed to find an effect from chrysotile, the Carolina study was larger and provided more power to detect effects of exposure.[24] On that basis, says this source, "you can't say asbestos is not a risk," despite differences that may be associated with fiber type. An EPA official says, "There are different forms of asbestos--nasty stuff and not-so-nasty stuff. Most of the studies were on the nasty stuff; there were fewer studies on the not-so-nasty stuff. The agency made a policy call to treat them the same." Another EPA official explains that the agency did not want to run the risk of waiting 5 or 10 more years for scientific consensus on the relative risk of different fiber types saying, "We won't play with public health as an experimental group."

Five years prior to the ban, a committee of the National Academy of Sciences concluded that "difficulties in interpreting studies of various groups of workers do not allow for reliable assessments of the role of fiber type (chrysotile or crocidolite) in determining risk for developing either lung cancer or mesothelioma" (NRC 1984). According to a former senior EPA official, "the linchpin to treating all fibers equally was the Academy report." In a more recent review of the scientific evidence, Stayner *et al*.

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[23] The link between fiber type and risk of cancer and disease is said to arise from different rates of fiber clearance from the lung (Bates 1994). Due to greater durability of amphibole fibers in the lung compared with the rapid dissolution of chrysotile (Doll and Peto 1985), it has been argued that amphibole fibers are the sole cause of mesothelioma, due to the presence of traces of tremolite fibers which contaminate commercial chrysotile and cause the small number of mesothelioma cases observed in male workers exposed to chrysotile (Doll 1989). According to Doll, studies strongly suggest that pure chrysotile does not cause mesothelioma. Doll does note, however, that a low occurrence of the disease has been observed in groups of men and women occupationally exposed only to chrysotile (with or without tremolite contamination). With regard to lung cancer, Doll notes firm evidence to prove a lower risk of lung cancer incidence clearly attributed to chrysotile is lacking. Nevertheless, there is a prominent group of scientists defending an "amphibole hypothesis," a term coined by Mossman *et al*. (1990). Mossman and colleagues argue that exposure to chrysotile at the level of current occupational standards does not increase the risk of asbestos-associated diseases. They criticize U.S. federal agencies for not differentiating between different types of asbestos like the European Community, which has more stringent rules for amphiboles. According to Stayner *et al*. (1996), opponents of the amphibole hypothesis contend that it is not a valid explanation for unexplained variances in effect from asbestos exposure because a mixture of fibers is usually present in commercial asbestos (i.e., chrysotile contaminated with tremolite).

[24] In its report on the health effects of asbestos, the Health Effects Institute, which is a public-private sector partnership, also hypothesized that chrysotile asbestos was the cause of most mesothelioma deaths of miners in Quebec (HEI 1991).

(1996) conclude, "Given the evidence of a significant lung cancer risk, the lack of conclusive evidence for the amphibole hypothesis, and the fact that workers are generally exposed to a mixture of fibers, we conclude that it is prudent to treat chrysotile with virtually the same level of concern as the amphibole forms of asbestos." A former senior EPA official reports being "skeptical" about treating all fibers as toxicologically equivalent but recognized that if the agency had proceeded down the "slippery slope" of assigning different toxicity values to different forms of asbestos, any specific toxic equivalency factor (a ratio of the toxicity of one substance to that of a reference substance) would have been construed as "arbitrary" and subject to attack on that basis.[25] In the face of uncertainty and the lack of scientific consensus, therefore, EPA viewed equal treatment not as arbitrary (as some have argued) but rather as precautionary.

Dose-Response Analysis

Epidemiological evidence suggests that asbestos is a potent carcinogen at relatively high occupational exposure levels. (EPA estimates that the 1986 OSHA standard of 0.2 f/ml represented a lifetime cancer risk on the order of 1 in 1,000 (10^{-3}) (*Fed. Reg.*, Vol. 54, p. 29467).) There remains, however, more uncertainty about the potency of asbestos at lower-level ambient exposures. (As discussed above, the potency may differ by fiber type.) NRC (1984) observed that few epidemiological studies had established quantitative dose-response relationships between levels of asbestos and health effects and that there was considerable variability observed among that studies that had. A factor that complicates interpretation of the epidemiological studies is that the risk of lung cancer from asbestos exposure is greatly increased by smoking (i.e., a synergistic effect), and a large proportion of the populations first identified as suffering from the effects of asbestos (e.g., World War II shipyard workers) were smokers. Some scientists held that evidence of DNA repair and asbestos fiber elimination would suggest a sub-linear dose-response form, an argument advanced by the Asbestos Industry Association (Gaheen 1995). (A sub-linear curve would estimate lower risks at low exposure levels than would a linear dose-response form.) Two years after EPA finalized the ban and phaseout rule, however, Health Effects Institute concluded that there was no experimental evidence for establishing the precise dose-response relationship and observed that occupational exposure levels have never been recorded with much accuracy, contributing to the uncertainty in the dose-response analysis (HEI-AR 1991).[26]

Consistent with its 1986 cancer risk assessment guidelines, EPA estimated dose-response relationships for asbestos-related lung cancer and mesothelioma using a linear,

[25] Note that EPA is confronting precisely this challenge in its current reassessment of dioxin. The agency has assigned different "toxic equivalent factors " (TEFs) to different types of dioxin, and commenters have criticized the lack of empirical basis for the different TEF values. Although assigning different uncertainty distributions to the toxicity of various dioxin congeners seems to be a reasonable alternative to TEFs, it is not hard to imagine similar challenges being raised about the lack of empirical basis for the selection of distributions.

[26] HEI is a non-profit research organization established in 1980 by EPA and the automotive industry. It created HEI-AR in response to the congressionally requested asbestos study to restrict HEI's civil liability and protect it against too close an involvement in the regulatory politics of asbestos (Jasanoff 1990).

no-threshold model. The agency estimated the dose-response constants as the geometric means of the "best estimates" (maximum likelihood estimates) from a number of epidemiological studies of effects associated with occupational exposures.[27] Thus, EPA directly extrapolated from the observed effects at relatively high, imprecisely measured occupational exposure levels to predict effects that would occur at low, ambient exposure levels to which most of the general public would be exposed. An academic mineralogist observes that there is no direct data or scientific evidence to validate the choice of model for extrapolation and concludes that the matter "could not be resolved on scientific grounds."

Exposure Analysis

TSCA's life-cycle perspective called for EPA to use an innovative approach to exposure analysis. Traditionally, EPA evaluates emissions or releases of toxic substances at the "end of the pipe" (i.e., point source emissions from production facilities).[28] EPA estimated that the majority of ambient asbestos exposures resulted from the use--not the manufacture--of automobile drum brake linings and disc brake pads (*Fed. Reg.*, Vol. 54, p. 29478). By banning asbestos, EPA sought to reduce or eliminate exposures from "cradle-to-grave" in mining, product fabrication, distribution, end use, and disposal.

However, as is the norm for any particular toxic substance (even one as intensely scrutinized as asbestos), national data were critically lacking on the number of people exposed to various levels of asbestos as a result of non-occupational exposures. EPA's asbestos exposure modeling relied heavily on extrapolations from occupational settings to non-occupational exposures. What EPA termed "analogous" exposure data would be extremely difficult and costly to validate but easy (as the Fifth Circuit Court demonstrated) to find fault with. The agency used, for example, data on asbestos levels measured at garages where brakes were repaired and at highway toll booths to estimate exposure levels for the general public. Another complicating factor is that data on workplace asbestos fiber concentrations are generally given as number of fibers longer than 5 μm per unit volume, whereas data on ambient concentrations have been expressed as mass per unit volume, and accurate conversions between mass measurements and fiber concentrations depend on a variety of factors for which data are generally unavailable (NAS 1984).

Although EPA did conduct sensitivity analyses on many of its exposure assumptions to examine if different assumptions would substantially change the projected health benefits, according to an EPA official, in retrospect, the agency "should have had real measurements" from the field to strengthen the non-occupational exposure

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[27] This statistical estimation procedure is similar to what EPA used in conducting the quantitative risk assessment for arsenic in drinking water using epidemiological data. This procedure differs somewhat from the standard procedure EPA uses in analyzing animal study data. It is somewhat less conservative than, but generally consistent with, agency cancer risk assessment guidelines (see Appendix B).

[28] EPA has used a similar multi-media approach to evaluating lead exposures, but lead pollution control has been implemented under a variety of statutes, and at times, there have been inconsistencies among EPA programs in addressing lead pollution. See Appendices A and H.

assessment. Another EPA official, however, points to the extremely high level of resources the agency devoted to asbestos ($10 million for the cost-benefit analysis alone) and concludes that the agency cannot afford to spend such sums of money on substance-specific analyses given the tens of thousands of chemicals in commerce.[29]

Combining its dose-response and exposure analyses, EPA estimated that thousands of asbestos workers and members of the general population were exposed to risks on the order of 1-in-1,000 (10^{-3}) from asbestos released from products subject to rule and that millions of people were exposed to risks on the order of 1-in-1 million (10^{-6}). It should be noted, however, as an EPA official suggests, public asbestos exposure resulting from the end use of products was estimated to be "tiny relative to occupational exposure and product fabrication." As the figures suggest, EPA estimated that the relatively low health risk from asbestos exposure to any particular member of the general public aggregated over the entire population roughly corresponded to the total risk of the smaller population exposed to relatively high individual risks (i.e., $10^{6} \times 10^{-6} = 10^{3} \times 10^{-3}$). Altogether, the agency projected that the ban and phaseout rule would avoid approximately 160-200 cancer cases over 13 years, as well as yielding other health benefits for which quantitative estimates were not available (e.g., cases of asbestosis) (*Fed. Reg.*, Vol. 54, p. 29460).[30]

Note that the final estimated health benefits of the 1989 asbestos ban and phaseout were considerably lower than those included in the proposal. Additional scientific analysis in combination with changes in the market place were responsible for the change. EPA's 1986 proposal estimated approximately 2,560 avoided cancer cases versus 160-200 avoided cases in the final rule. The two primary reasons for the difference were: 1) some of the products (e.g., vinyl-asbestos floor tile) included in the proposal were no longer manufactured or imported in the U.S. by 1989 (accounting for about 475 cases); and 2) modifications were made to the health effects model (e.g., lower dose-response constant for mesothelioma resulting from using the average of many studies rather than the estimate from one large study) that resulted in an estimate of health benefits approximately 20% lower for the final rule than for the proposal (accounting for about 200 cases). Thus the estimated health benefits of the proposed ban were nearly halved as a result of projected asbestos exposures being "preempted" by the market's reaction to asbestos liability and in anticipation of the ban.[31]

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[29] Dahl (1995) reported that the TSCA inventory included more than 72,000 chemicals and that EPA receives more than 2,000 PMNs per year. Shapiro (1990) estimates that a modest battery of physical/chemical properties tests would cost $250 thousand per substance. The costs of exposure analysis would be additional.

[30] EPA did not quantify avoided asbestosis cases because there was no evidence of "disabling" asbestosis occurring at low occupational exposure levels (*Fed. Reg.*, Vol. 54, p. 29470). Asbestosis that is not so severe as to disable, however, might significantly affect quality of life.

[31] The remainder of the difference in estimated health benefits between the proposed and final rule was accounted for by revised final exposure assessments for some asbestos products that were lower than those used in the proposal and a two year reduction in the time frame for the regulatory analysis (15 years for the proposal versus 13 years for the final rule) (*Fed. Reg.*, Vol. 54, p. 29486).

Substitutes: Risk-Risk Tradeoffs

Some critics of the proposed asbestos ban suggested that the poorly understood toxicity of substitute fibrous materials might be comparable to or worse than that of asbestos.[32] EPA concluded on the basis of available toxicological and epidemiological data that fibrous substitutes are generally less biologically active and pathogenic than asbestos. The agency also concluded that occupational exposures due to synthetic fibrous substitutes would probably be lower than that from naturally-occurring substitute fibers because the mining and milling of the latter tend to be "dusty" operations. Another aspect of synthetic substitutes that EPA believed made them less risky was that their diameter size could be controlled to reduce the presence of fibers in the respirable range (under a few microns) (*Fed. Reg.*, Vol. 54, p. 29460).

EPA did not, however, explicitly estimate safety and some non-cancer risks due to the reduced performance or higher costs of products containing asbestos substitutes. According to an EPA official, the first risk substitution issue raised was brakepads. There was concern that banning the use of asbestos in brake linings might increase traffic accidents and fatalities due to longer breaking distances.[33] (EPA did convene meetings with the National Highway Traffic Safety Administration (NHTSA) to consider this question, and NHTSA did not object to EPA's final rulemaking (*Fed. Reg.*, Vol. 54, pp. 29494-20495).) Others raised concerns that the asbestos ban might limit access to safe drinking water in less developed countries because asbestos-cement pipe is the cheapest form of building structurally-sound water mains.[34]

3. The Process within EPA

Setting the Agenda

Prompted by a public petition to ban asbestos cement pipe (under the provisions of TSCA Sec. 12), the asbestos ban officially got onto EPA's regulatory agenda in 1979 when the agency published its advance notice of proposed rulemaking. As witnessed by its prominence in the earliest regulatory actions of EPA and OSHA, however, asbestos was on the public agenda long before the Carter administration.

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[32] For example, Interior Department scientist Malcom Ross suggested that replacement substances such as rock wool and fiberglass could be more harmful than asbestos and accused EPA's plan to prohibit the asbestos of creating a public "crisis", only because a small number of people had hypothesized non-occupational exposures to asbestos as harmful (*Inside the EPA* 3/22/85).

[33] According to a DuPont official cited by (Peck 1989), there were very few uses of asbestos for which there were no substitutes available with the exception of brakes for heavy machinery. DuPont is a manufacturer of asbestos substitutes. The observation that substitutes were unavailable for heavy-duty brakes tends to support the idea that substitutes were not as effective for lighter vehicles.

[34] One of the legal challenges to the asbestos ban was EPA's failure to consider risks borne outside the country, but the court held that this was a proper construction of TSCA. As a former senior EPA official remarked, "TSCA doesn't regulate Guatemala."

Assessing the Science: Asbestos

Three out of five interviewees responded that there was adequate science to inform EPA's decision to ban or phaseout asbestos in 1989. Respondents generally agreed that the areas of greatest uncertainty were non-occupational exposure levels and the risks posed by substitutes for some of the lower volume asbestos uses. One respondent felt that the scientific information available to assess non-occupational risks was sparse, poor in quality, and highly uncertain and characterized EPA's treatment of the science as poor. Otherwise, EPA's scientific assessment got good marks.

As has been indicated, EPA's assessment of the health risks posed by asbestos took place over many years and was a large enterprise, so large in fact, that the Office of Pesticides and Toxic Substances (OPTS) employed a complicated matrix-management tasking scheme to conduct components and sub-components of the regulatory analysis and to organize staff for particular working groups and assignments. Although the practice was very much in keeping with the latest business management trend, the result was (perhaps unavoidably) fragmented. According to an EPA official "top people didn't necessarily know what was going on, nor did the staff scientists."

The so-called "matrix management" approach was necessitated by OPTS's need to be flexibly staffed with analysts having adaptable functional skills (e.g., exposure assessment) necessary to conduct or oversee components of any substance-specific assessment. As one EPA official laments, EPA does not have nearly enough resources to house experts for every suspected toxic substance. Thus, OPTS could not afford to amass (and then disband) a team of asbestos experts to work closely as an interdisciplinary team in a comprehensive scientific assessment. The agency, in fact, had no in-house asbestos expertise. Instead, it contracted for expertise as needed. In short, then, the agency traded-off expertise for flexibility, and the result was a fragmentary analytical process. Fortunately for EPA, riding herd over the asbestos assessment from 1983-89 was Assistant Administrator John Moore, a toxicologist by profession, and a former Deputy Director of the National Toxicology Program.

EPA's initial assessment of asbestos (*Exposure to Asbestos*) was prepared in 1979.[35] After meeting with asbestos industry representatives to discuss the report's methods and conclusions, EPA informed the Asbestos Information Association (AIA) that the agency did not plan to use the report as a reference document in support of future rulemaking (Gaheen 1995). In 1980-82, EPA issued a series of 3 draft documents on asbestos health effects and the magnitude of indoor air exposures in support of rulemaking on asbestos in school buildings. Hughes and Weill (1989) suggest that the changing risk estimates from each subsequent draft added to public confusion over the risks posed by

[35] IIT Research Institute (of Chicago, IL) prepared the assessment under contract to EPA.

exposure to asbestos in schools.[36] With no real in-house asbestos expertise, EPA turned to external scientists and blue ribbon panels.

In 1982, during the Gorsuch administration, EPA requested that the National Academy of Sciences undertake a study to evaluate non-occupational exposure to asbestos and to determine whether differences among fiber types should be incorporated into the agency's risk assessment. The committee convened in 1983 and issued its final report in 1984 (NRC 1984). As discussed above, this report served as the basis for EPA's decision to treat different types of asbestos fibers as toxicologically equivalent. In addition, the committee estimated lung cancer and mesothelioma risks resulting from given ambient asbestos levels for smokers and non-smokers, males and females. Of 13 members, however, the NRC panel included only one expert on quantitative risk assessment methods. Several calculation errors were discovered in the committee's original report, which was withdrawn, and other errors remained undetected in the revised version.[37] As a result, using the committee's own underlying assumptions, the report was later found to have underestimated risks for mesothelioma by a factor of 17.4 and for lung cancer by a factor of 4.5 (Breslow *et al*. 1986). As a former senior EPA official allows, "The report was not an example of the Academy at its finest." Needless to say, the report did not provide EPA with a recognized authoritative basis for its quantitative conclusions.[38]

In 1984, the EPA's Office of Research and Development/Environmental Criteria and Assessment Office (ORD/ECAO) contracted out the *Airborne Asbestos Health Assessment Update* to William Nicholson, an epidemiologist and colleague of Irving Selikoff at Mt. Sinai Hospital.[39] EPA's critics suggested that Nicholson's assessment was unbalanced and overstated the health risks from asbestos, largely due to the lack of

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[36] The 1980 draft estimated 100-8000 premature deaths attributable to school exposures, while this was reduced to 40-400 deaths in the 1981 draft, without any changes in the underlying exposure assumptions. The 1982 draft contained no quantitative risk estimate.

[37] Hughes and Weill 1989 note that the NRC report failed to use lifetable methods (taking into account age at onset of exposure and disease) for estimating mesothelioma risk or to adjust lung cancer risk to continuous (24 hr./day) rather than work-place exposure (8 hr./day)

[38] In contrast to the approach adopted by EPA and the NRC, in 1982 the CPSC appointed a panel of 7 scientists, including 3 experts in quantitative methods, from a list of scientists nominated by the National Academy of Sciences to prepare a report on asbestos (CPSC 1983). The estimates of mesothelioma risks provided by the CPSC panel were about an order of magnitude higher than those in NRC (1984) (see EPA 1986, Table 7-1). Also, unlike EPA, which released of a series of widely circulated draft reports during 1980-82, it was CPSC policy not to publicly release preliminary drafts. Only the final draft was released for public comment (Hughes and Weill 1989). As a result, the broad uncertainty in CPSC's estimates was revealed to the public in one fell swoop, whereas EPA's sequence of draft estimates changed over time.

[39] Although this assessment document was developed nominally for the purposes of updating the scientific criteria for the national emission standard for asbestos as a hazardous air pollutant under the Clean Air Act (asbestos was on of the few substances regulated by EPA as a hazardous air pollutant prior to the 1990 Clean Air Act Amendments), it was a principal supporting document for the asbestos ban and phaseout rule under TSCA. Nicholson also served as the consultant to OSHA for a 1986 asbestos health assessment (Hughes and Weill 1989). See Stone (1991) for a discussion of the two staunchly opposing camps of asbestos health effects scientists.

distinction made between types of asbestos.[40] An academic mineralogist insinuated that the choice of Nicholson to conduct the asbestos health assessment was due to then-ORD Assistant Administrator Bernard Goldstein being a like-minded "Selikoff guy." Nicholson's report was, however, released in draft in 1984 and 1985 for public comment and was peer reviewed by the Environmental Health Committee of the EPA Science Advisory Board in 1984 prior to revisions and release in 1986.[41] According to an EPA official, the agency moderated and significantly changed the early draft of the asbestos health assessment "in response to external reviewers, primarily from industry, who objected wildly."

All of these externally prepared reports, however, fell short of generating the type of scientific information and analysis EPA needed under TSCA--an exposure assessment. TSCA requires EPA to balance regulatory costs and benefits. Therefore, it was not sufficient to demonstrate conclusively, as one EPA official pointed out "that the stuff [asbestos] kills people," or to provide, as did Nicholson and the NRC panel, disease risk estimates for *given* low levels of ambient asbestos. The agency needed a projected "bodycount" to compare with the costs of regulatory compliance, and an asbestos exposure assessment estimating the *actual* levels of asbestos in the environment was the missing piece to link available health effects information to regulatory benefits (i.e., avoided cases of disease). As an EPA official explains, the agency treated the hazard the same across products but conducted a separate exposure analysis for each product. "It was a tremendous amount of work to identify each product category, and a lot of work done to figure out the exposure for each product category. The economic model needed exposure work from all parts of the product lifecycle." EPA thus confronted the challenge of estimating the occupational and non-occupational asbestos exposures resulting from the entire life-cycle, from the mine to the landfill and every point in between, of hundreds of asbestos products.

Much of the original occupational exposure data EPA used during formulation of the 1986 proposal came as a result of a 1982 reporting rule issued under TSCA (Sec. 8). This data was supplemented by inspection reports from OSHA and the Mine Safety and Health Administration (MSHA) and studies by the National Institute of Occupational Safety and Health (NIOSH). Predictably, the asbestos industry faulted the quality of the data for regulatory decisionmaking and EPA's use of the data to extrapolate to non-occupational exposures. In preparation for the final rulemaking in 1989, EPA conducted an Asbestos Exposure Survey in 1986-87. OPTS's final asbestos exposure assessment was like a garment stitched together from three bolts of cloth: an analysis of occupational exposure to asbestos and asbestos releases from manufacturing and commercial operations; a modeling study estimating ambient exposure levels resulting from the

[40] The EPA (1986) risk estimate for lung cancer is also approximately 50% higher (across all groups) than that of the CPSC (1983). The difference is primarily due to different assumptions concerning the background risk of lung cancer in the absence of asbestos exposure (Hughes and Weill 1989.

[41] Nicholson later criticized the 1991 HEI-AR report for downplaying the health risks of asbestos fibers less than 5 micrometers in length. An HEI-AR panel member, Nicholson refused to sign the report.

releases from industrial and commercial sources; and an analysis of consumer and ambient exposures resulting from asbestos products in use.[42]

Assessing the Science: Asbestos Substitutes

According to a former senior EPA official, EPA spent "a fair amount of time trying to develop reasonable estimates" of exposure to and health effects from asbestos substitutes and was "very sensitive to the need to look at friction product substitute risks." This source states that the agency's major focus in this area was on friction products; however, for other uses of asbestos, "particularly where the quantity of asbestos used compared to friction products dropped off by orders of magnitude, there was lower scrutiny and less intellectual input" into the risk analysis of substitutes. OPTS's 1988 *Health Hazard Assessment of Non-Asbestiform Fibers* was produced by its Health and Environmental Review Division. Its evaluation was based, in part, on a prior review of epidemiological studies on populations exposed to non-asbestos fibers prepared by the OPTS Economics and Exposure Division. The examples of safety and non-cancer risks posed by asbestos substitutes discussed above (i.e., from brakepads and cement pipe) suggest the great difficulties inherent in comparing qualitatively different health risks (e.g., cancer vs. trauma) and underscore the dilemma of establishing the appropriate scope of analysis for substitute risks.

Scientific Review Procedures

TSCA requires that EPA conduct quasi-judicial hearings as part of its rulemaking process. These administrative proceedings included scientific testimony from agency staff and permitted cross-examination of witnesses, and according to an EPA official, forced EPA to pay more attention to particular analytical issues. However, EPA's scientific review process for the asbestos ban and phaseout seems to have revolved principally around its internal workgroup process. An EPA official described the work group review process as a series of staff briefings for EPA managers in which staff not directly responsible for the analysis would participate and in which staff recommendations either "would be ratified or shot down." As suggested above, EPA also engaged in an informal external scientific review process by widely circulating a series of draft asbestos health assessments and reacting to subsequent feedback (Hughes and Weill 1989). The fact that much of the scientific review of staff analysis was internal and informal, however, does not necessarily mean that it was not rigorous. Regarding the asbestos ban and phaseout rule, an EPA official remarked that Assistant Administrator John Moore insisted on independent peer review and held staff to a high standard.

The appeals court found, however, that EPA was deficient in one part of the formal scientific review process, public notice and comment. EPA argued that it gave "constructive" notice by notifying the public of the available exposure information that

[42] The analysis of occupational exposures was conducted by ICF under contract to OPTS. The OPTS Economics, Exposure, and Technology Division would presumably have been responsible for the exposure assessments.

could be manipulated to estimate exposures. According to the court, however, this did not constitute adequate public notice of the agency's intended use of exposure data. In addition, according to an EPA official, the EPA Science Advisory Board (SAB) was troubled that it was not more involved in the asbestos ban and phaseout decisionmaking process. Since the SAB's Environmental Health Committee was involved in reviewing the agency's Asbestos Health Assessment Update (EPA 1986), one can infer that the Board's concern in this case also related to the asbestos exposure analysis and their lack of opportunity to review and comment on it.

Communicating the Science to Decisionmakers

According to a former senior EPA official, the proposed ban and phaseout rule eventually went forward in 1986, largely on the basis of a review of the epidemiological information provided by Assistant Administrators John Moore (OPTS) and Bernard Goldstein (ORD) to Administrator Lee Thomas. Linda Fischer became Assistant Administrator for OPTS under the Reilly administration in 1989 prior to the final rulemaking, and, according to an EPA official Reilly administration officials were briefed on the science and requested additional analysis. Moore, however, was a key decisionmaker. Of the asbestos ban and phaseout, an EPA official simply states, "Jack Moore ran that show." As an experienced scientist and science administrator, Moore was effective in creating demand for rigorous scientific analysis by having an expert's grasp on the substance and by appealing to the pride and professionalism of his staff. "If the staff briefed Moore on lousy science, they were embarrassed, and this permeated" throughout the pesticides and toxic substances program, recalls an EPA official.

4. Science in the Final Decision

Three out of five interviewees believed that the scientific information had a high impact on the agency's final decision to ban or phaseout nearly all uses of asbestos, and there appear to have been very few, if any, impediments to the consideration of the science by decisionmakers.[43] In fact, one EPA official suggests that the role of science in the decision was excessive. "People were too concerned with the science," while too little attention was paid to the "balancing of costs required under the law [TSCA]. Asbestos was so clearly a danger to humans that it short-circuited thinking about other things people should think about in making regulatory decisions, [such as] cost-benefit analysis and substitutes." In the judgment of a former senior EPA official, the science supporting the asbestos ban and phaseout was, on the one hand, "very strong" because of the data on observed health effects in humans, but on the other hand, it was "not strong in relation to exposure and particular uses." Although OMB had officially signed-off on EPA's regulatory analysis when it acceded to the proposal in 1986, the budget office's involvement in the process apparently did not end there. According to an EPA official,

[43] An academic mineralogist suggested that EPA ignored scientific information generated by the asbestos industry that did not support its decision. An EPA official suggests that the principal impediment to considering science in this case was that the agency underestimated the opposition and failed to effectively engage asbestos exporters from Canada and Australia.

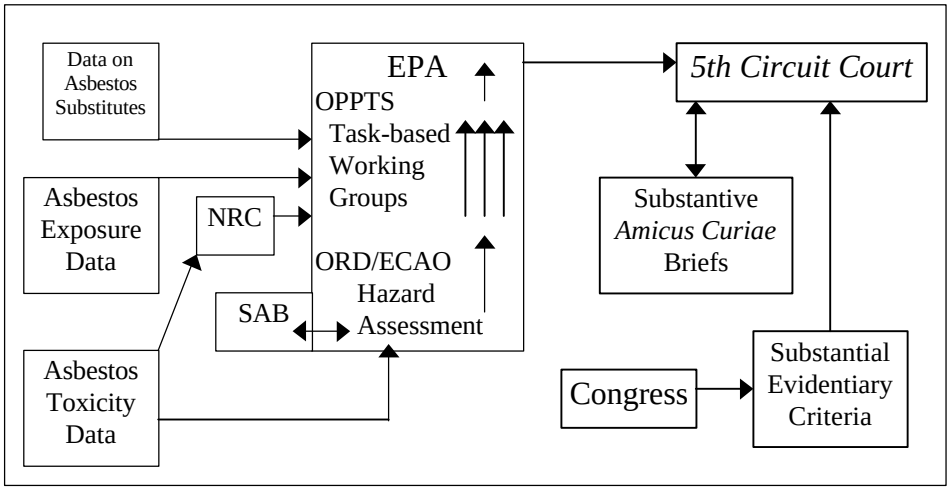
"At the end we were arguing with OMB." Apparently what was decisive in coming to closure on the rule was that "there was a body count from shipyard workers exposure." The fact that EPA was going to regulate asbestos on the basis of occupational (human) data rather than bioassays (animal studies) "gave more weight" to the agency's argument to proceed.

5. Concluding Observations

Regarding the appellate court decision to overturn the asbestos ban, an EPA official speculates, "Once the court thought it was dumb, they were going to find a reason to overturn EPA's decision." A number of commenters have similarly characterized the court's ruling as replacing the administration's policy judgment about what constitutes an unreasonable risk and sufficient evidence with the court's own judgment. An academic defends the court's activism: "EPA's stance was precautionary to the extent of being financially untenable." The agency's analysis was "not founded on the most balanced and rational assessment of what was known, and what was socially responsible." The court's decision, in turn, was a "reasonable response" to EPA's "unacceptable decision." In this view, then, the judicial appeals process is an appropriate, and perhaps necessary, component of political regulatory decisionmaking. However, Davies *et al*. (1979) argue that "the determination of what constitutes an unreasonable risk is a societal value judgment." Under the constitutional separation of powers, such judgments are reserved for politically accountable decisionmakers.

In terms of a fate and transport analogy, the court can be compared to wildlife that is unintentionally exposed to pesticides in the field--it was not the intended "target" of the scientific information. (In the pesticides field, non-pest wildlife are referred to as non-target organisms.) The court received science both indirectly as mediated by EPA and directly through soliciting *amicus curiae* briefs on substantive matters. Despite the court's limited capability to comprehend and critically analyze, or "absorb and metabolize" the science, it substituted its own science policy judgment for that of politically accountable decisionmakers of the more expert administrative agency. As illustrated in Figure B-1, however, the court was arguably invited to do so by the substantial evidentiary judicial review standard specified under TSCA by Congress.

Figure B-1. Fate and Transport of Science in the Asbestos Ban and Phaseout Rule.



The Carnegie Commission (1993, p. 57) observes:

The asbestos decision has provoked considerable debate, and fingers have been pointed in several directions. Regardless of whether the statute, the courts, the agency, or others should be faulted in this case, it is unsettling that EPA could not satisfy TSCA's requirements for promulgating a single rule after a decade's effort. The case raises numerous questions, including whether the executive branch should encourage Congress to revise this legislation, and under what circumstances the agency should devote such a vast amount of time and resources to a single substance at the expense of many of pressing issues in its jurisdiction.

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LIST OF ABBREVIATIONS

ACGIH	American Council of Governmental and Industrial Hygienists
AHERA	Asbestos Hazard Emergency Response Act of 1986
AIA	Asbestos Industry Association
ATSDR	Agency for Toxic Substances and Disease Registry
CPSC	Consumer Product Safety Commission
ECAO	Environmental Criteria and Assessment Office, EPA
EPA	Environmental Protection Agency
f/ml	fibers per milliliter
HEI	Health Effects Institute
HEI-AR	Health Effects Institute-Asbestos Research
HEW	Department of Health, Education and Welfare
IARC	International Agency for Research on Cancer
µm	micrometer
NIOSH	National Institute of Occupational Safety and Health
NRC	National Research Council
OMB	Office of Management and Budget, The White House
OPTS	Office of Pesticides and Toxic Substances, EPA
ORD	Office of Research and Development, EPA
OSHA	Occupational Safety and Health Administration
PMN	premanufacture notice
RIA	Regulatory impact analysis
TEF	toxic equivalent factor
TSCA	Toxic Substances Control Act of 1976