The 1991 Lead/Copper Drinking Water Rule & the 1995 Decision Not to Revise the Arsenic Drinking Water Rule: Two Case Studies in EPA's Use of Science

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Abstract

This paper discusses EPA's acquisition and use of science in two decisions under the Safe Drinking Water Act: the 1991 revision of the lead drinking water regulations and the 1995 decision to pursue additional research instead of revising the arsenic in drinking water standard. In the first case, a committed band of policy entrepreneurs within EPA mobilized and supplemented scientific information which had accumulated in the agency's air program to force lead in drinking water up the agency's regulatory agenda. In the minds of senior EPA decisionmakers, there was adequate science to justify making the lead in drinking water regulation more stringent; the critical question was "how far to go" in terms of regulatory compliance expenditures. To the extent that the agency's use of science increased the regulatory benefits estimate, it could rationalize more stringent and costly regulations. In the case of arsenic in drinking water, not only the scientific uncertainties in estimating the health risks but also the regulatory compliance costs, the distribution of those costs, and the presumed public health impacts of delay were important in the decision to pursue additional research on the health risks of arsenic. However, because EPA decisionmakers have failed to articulate what they consider to be compelling scientific evidence to justify departing from default risk assessment procedures in this case, it seems less likely that future research will facilitate future decisionmaking. Both cases illustrate impediments to the generation of scientific data needed for regulatory decisionmaking, the potential for scientific information to be distorted in or omitted from the regulatory decisionmaking process, and the key roles played by intermediaries between scientists and decisionmakers within EPA.

Abstract

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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 97-05 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

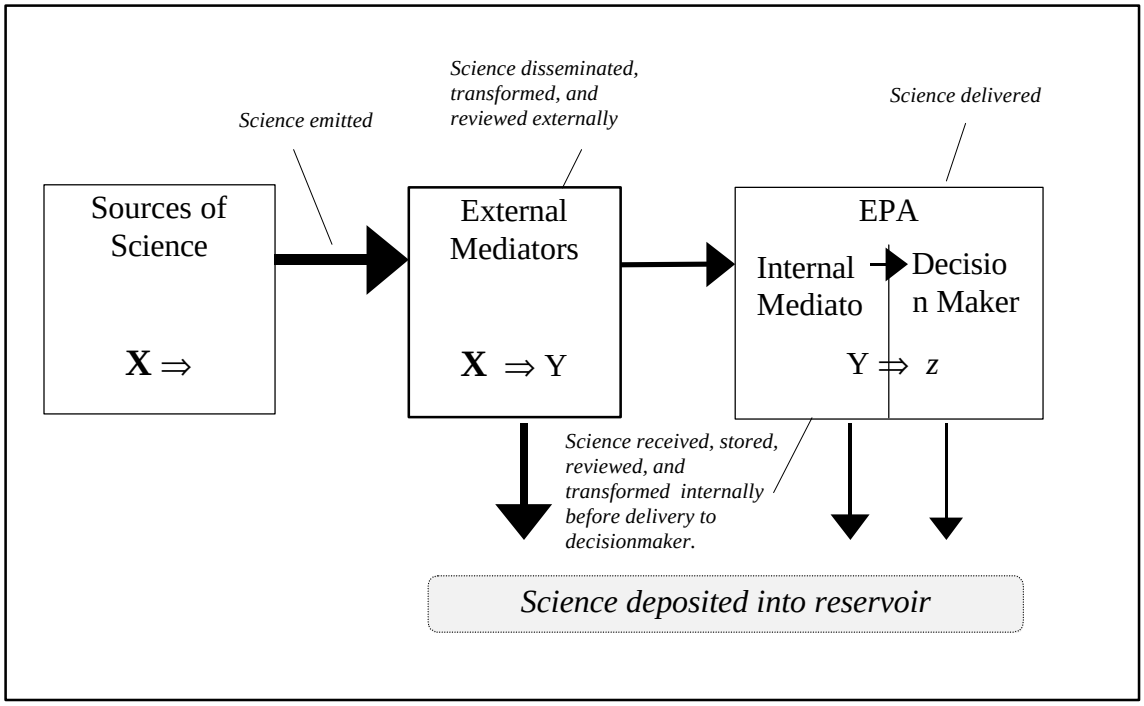
Development of the case studies was based on literature review and interviews with persons inside and outside EPA. The number of interviewees per case study varied roughly from a half dozen to a dozen. There was an effort to ensure balance in the group of respondents for any particular case study, but 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 1991 Lead/Copper Drinking Water Rule

1. Background

In response to the 1974 Safe Drinking Water Act (SDWA), in 1975 EPA promulgated an Interim Drinking Water Regulation for lead which set a Maximum Contaminant Level (MCL) of 50 ppb (parts per billion). Like rules for other drinking water contaminants, the lead rule was not specific as to where or how the water should be sampled to ensure compliance. For most contaminants it did not seem to matter where drinking water samples were taken, since roughly the same concentration was measured at the tap as at the drinking water treatment plant. However, the primary source of the metal is not lead in the source water but lead leached from the delivery system. Therefore, removal of lead from water at the treatment plant would not prevent drinking water contamination by plumbing.

In a June 1984 case which dramatized the exposure of children to lead through drinking water, a routine blood test run on a 24-month-old Massachusetts girl revealed blood-lead (PbB) level (i.e., concentration of lead in the bloodstream) of 42 μg/dL (micrograms per deciliter). This level was considerably higher than the Centers for Disease Control's screening level of concern. After eliminating paint, furniture, food, yard soil, and toys as possible sources of the lead exposure, public health officials discovered that the family's tap water contained up to 390 ppb lead and was being contaminated by lead solder in plumbing in the newly constructed home (Stapleton 1994, pp. 89-90).[1] By November 1985, EPA had proposed to lower the Maximum Contaminant Level Goal (MCLG) for lead to 20 ppb (*Fed. Reg.*, Vol. 50, p. 46936).

However, the agency's proposed revision was overtaken by events. In January 1986, Massachusetts instituted the nation's first ban on the use of lead solder in lines carrying drinking water (Stapleton 1994, pp. 89-90). In May 1986, the U.S. Congress weighed in through the SDWA Amendments, which banned future use of materials containing lead (e.g., solder, flux, and pipes) in public drinking water systems and residences connected to them and limited the lead content of brass used for plumbing (e.g., in fittings). The 1986 Amendments also listed 83 contaminants, including lead, for which EPA was to simultaneously develop MCLGs and National Primary Drinking Water Regulations (NPDWRs). MCLGs are non-enforceable health-based goals, while NPDWRs (which include either MCLs or treatment and/or monitoring requirements) set enforceable standards. The SDWA directs that MCLGs be set at a level at which "no known or anticipated effects on the health of persons occur and which allows an adequate margin of safety." The House Report on the 1974 bill stated that if there is no safe threshold for a contaminant, the recommended MCL (the health-based goal) should be set

[1] A reviewer notes that documented cases of lead poisoning from tapwater date back to the 1920s and that this particular case was not reported in the scientific literature until 1989. This episode is not cited to imply that it motivated EPA to propose to revise the MCLG in 1985; however, Stapleton (1994) suggests that this particular episode dramatized the danger of lead solder in Massachusetts, which instituted the nation's first ban on lead solder in lines carrying drinking water shortly thereafter.

at zero. Prior to the 1996 SDWA amendments, the SDWA directed the agency to set the MCL for a contaminant as close to the MCLG as is feasible (*Fed. Reg.*, Vol. 56, p. 26460).[2]

In August 1988, the agency proposed to revise the lead in drinking water MCLG, MCL, and NPDWR (*Fed. Reg.*, Vol. 53, p. 31516), and the rule was finalized in June 1991 (*Fed. Reg.*, Vol. 56, p. 26460). The final rule established some precedents, including setting an MCLG of zero for a non-carcinogen and establishing an action level triggering specified treatment, as opposed to an MCL. In the proposed rulemaking, EPA had proposed an MCL of 5 ppb measured at the drinking water plant, but the agency adopted the action level of 15 ppb measured at the tap in order to capture all sources of lead in drinking water. Some members of Congress, notably Rep. Henry Waxman (D-CA), and environmental groups protested EPA's decision to forego setting a numerical MCL because they believed an action level would be more difficult to enforce. The rule was also one of the most expensive drinking water rules ever adopted by EPA, with compliance costs estimated at $500-$790 million per year (*Fed. Reg.*, Vol. 56, p. 26539).

The 1991 Lead/Copper[3] drinking water rulemaking can be separated into three parts: 1) the decision to adopt a multi-media strategy for controlling lead; 2) the determination of the MCLG; and 3) the formulation of the NPDWR. Science played a substantial role in each of the three components of the rulemaking. First, the control of lead in drinking water is appropriately viewed as part of one of EPA's first concerted attempts to regulate a chemical as a multi-media pollutant. The agency's multi-media strategy for controlling lead was based on the recognition that lead levels measured in blood and bone were the sum result of exposures and uptake from various sources (industrial and auto emissions, lead paint, lead plumbing) and media (air, water, food, soil, and dust). Second, although EPA would typically consider its classification of lead as a probable human carcinogen as sufficient to warrant an MCLG of zero, lead's carcinogenicity was rendered subordinate in the drinking water decision. It was overtaken by a coalescence of scientific opinion that there is no discernible threshold for lead's non-cancer effects and that the non-cancer effects, in particular impaired learning ability and delays in normal mental and physical development in children, were more serious than the

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[2] Prior to the 1996 amendments, the SDWA did not explicitly direct EPA to consider costs, however, on the basis of 1974 House Report language and 1986 statements in the Congressional Record, EPA interpreted feasibility of MCLGs to mean that "the technology is reasonably affordable by regional and large metropolitan public water systems" (*Fed. Reg.*, Vol. 54, pp. 22093-22094). While the EPA followed this interpretation of feasibility in the case of lead in drinking water despite concerns voiced by some about the costs of regulatory compliance for small systems, the agency's decisionmaking regarding arsenic in drinking water has been greatly influenced by the projected costs that would be borne by small suppliers if the MCL for arsenic were substantially lowered. (See the accompanying arsenic in drinking water case study.) The 1996 SDWA Amendments direct EPA to consider a feasible MCL and define feasible as affordable to large systems, but permit the agency to adopt another standard if the benefits of the feasible standard do not warrant the costs.

[3] EPA promulgated the rules for lead and copper concurrently because both occur in drinking water as corrosion by-products; however, the rulemaking for copper was relatively non-controversial, and this case study focuses exclusively on lead issues.

potential cancer effects. Third, the realization that the major source of lead in drinking water was lead corroded from materials in household plumbing and the distribution system, in combination with the observation that measurements of lead in drinking water display great variability at the same tap over time, persuaded EPA to adopt an NPDWR designed as an action level requiring water treatment to reduce the corrosiveness of water in the distribution system, replacement of water service lines containing lead, and/or source water treatment.

The science of water chemistry made important contributions in formulating the treatment requirements, but there remained considerable uncertainties about the efficacy of corrosion controls and the contribution of lead service lines to lead levels at the tap. Science also played an indirect--but crucial--role in the development of the NPDWR, because to the extent that EPA could use scientific information to quantify and monetize greater regulatory health benefits, the agency could justify a more stringent regime of treatment and monitoring requirements.

Table A-1 provides background for the development of national lead regulation and policy. Table A-2 provides a timeline of the lead in drinking water rulemaking. In terms of establishing health-based goals, the lead drinking water regulatory development essentially "piggy-backed" on the scientific analysis performed by and for the EPA air quality program. In fact, although the venue shifted from air to drinking water, there was considerable continuity in the "cast of characters" that took part in the debate over the agency's lead strategy. Ultimately, a committed band of policy entrepreneurs within the agency effectively mobilized the scientific data, analysis, and legitimacy accumulated in one compartment of the agency (the air program) and transported it to another (the drinking water program).[4] Due to prior exposure to the science of lead's health effects in relation the air quality program, senior agency officials were familiar with and sensitized to much of the relevant scientific information when the drinking water decision arose. This pre-existing sensitivity enabled the science--and those responsible for transporting and supplementing it--to have considerable access and impact in the lead in drinking water rulemaking.

The agency's multi-media lead strategy, formally articulated in 1991, evolved over many years during which a potent group of EPA staff, acting as policy entrepreneurs, formed what became known, both disparagingly and admiringly, as the "Lead Mafia." Although one component of the agency's lead strategy--the phaseout of lead in gasoline--has resulted in what many analysts regard as perhaps the most cost-effective environmental regulation on the books,[5] the scientific basis for, and implementation of, the strategy continue to be controversial. A primary focus of the current controversy

[4] As used in the public policy literature (e.g., Kingdon 1984), the concept of policy entrepreneurs refers to members of the policy community who blend the qualities of the expert analyst and the advocate to effect policy change, particularly through advancing ideas onto the policy agenda.

[5] See, for example, Portney (1990), pp. 62-63.

regarding EPA's lead strategy is the cleanup of sites with lead contaminated soils (*Science*, 10/15/93, p. 323).[6]

Table A-1. Background on U.S. Lead Regulation and Policy.

1946	Episode in England where workers cleaning gasoline tanks suffered neurologic effects from high level exposure to tetra-ethyl-lead.
1960's	Public health officials consider blood lead (PbB) levels up to 60µg/dL in children and 80 µg/dL in adults acceptable, based on acute clinical effects from occupational exposure studies.
1971	Lead-based Paint Poisoning Prevention Act directs the Consumer Product Safety Commission (CPSC) to establish a level of safety for lead in paint. The Department of Housing and Urban Development (HUD) is made responsible for removing lead-based paint from public housing.
1972	EPA inherits the Lead Liaison Committee from the Public Health Service.
1973	Clean Air Act initiates phase-out of leaded gasoline. (EPA negotiations with industry over lead standards in gasoline are the first opened to public scrutiny under the 1972 Federal Advisory Committee Act.)
1975	Lead-intolerant catalytic converters required on new automobiles. EPA sets an "Interim" MCL for lead in drinking water. Centers for Disease Control (CDC) guidelines set screening level for children's PbB at 30µg/dL.
1977	Clean Air Act Amendments add lead to criteria air pollutants.
1978	EPA sets lead National Ambient Air Quality Standard (NAAQS) of 1.5µg/m^3 (designed to consider children's multi-media exposures to lead).
1979	Needleman *et al*. report study of non-acutely toxic lead levels on children's IQ.
1980's	FDA encourages domestic food industry to decrease use of lead-soldered cans.
1981	Ernhart *et al*. dispute Needleman's results.
1982	EPA begins review of lead NAAQS, convenes a CASAC (Clean Air Science Advisory Council) subcommittee to review dispute between Needleman and Ernhart. Review panel concludes that Needleman's study neither supports nor refutes the hypothesis that low or moderate levels of lead exposure lead to cognitive or behavioral impairments in children.
1983	National Health and Nutrition Examination Survey (NHANES II) shows decline in population PbB levels resulting largely from reductions in leaded gasoline.
1985	EPA accelerates phase-out of leaded gasoline, reducing the limit from 1.0 grams/gallon to 0.5 grams/gallon, based on assessment of non-cancer health effects. EPA proposes revised lead in drinking water standard. CDC screening level for children's PbB reduced from 30µg/dL to 25µg/dL.
1986	Safe Drinking Water Act Amendments ban future use of materials containing lead in public drinking water distribution systems and residences connected to them. Superfund Amendments and Reauthorization Act (SARA) requires the Agency for Toxic Substances and Disease Registry (ATSDR) to prepare a study of lead poisoning in children. Lead in gasoline limit further reduced to 0.1 grams/gallon. EPA revises Air Quality Criteria for Lead. CASAC accepts inclusion of Needleman's reanalysis supporting his earlier conclusions.
1987	Reports of studies in Boston (Bellinger *et al.*) and Cincinnati (Dietrich *et al.*) confirm effects of low-level lead exposure on children's cognitive development.
1988	Lead Contamination Control Act requires monitoring and recall of lead-lined tanks from drinking water coolers in schools. Lead-Based Paint Poisoning Prevention Act authorizes federal funds for community PbB screening. ATSDR report estimates that about 17% of U.S. children in 1984 were exposed to lead at levels that pose the risk of adverse health effects; suggests a potential risk of developmental toxicity from lead exposure at PbB levels of 10-15 µg/dL or lower; and identifies paint and contaminated soil as the principal sources of lead for children most at risk. EPA proposes Lead/Copper Drinking Water Rule.
1990	Clean Air Act Amendments prohibits leaded gasoline by 1996. EPA updates Air Quality Criteria and Staff Paper for lead, but NAAQS for lead remains unchanged. CASAC recommends a maximum safe PbB level for children of 10 µg/dL and concludes that there is no discernible threshold for lead effects. Superfund suit pits Needleman and federal government v. Ernhart and industry.
1991	EPA formalizes its multi-media *Strategy for Reducing Lead Exposures*. EPA finalizes the Lead/Copper Drinking Water Rule. U.S. Centers for Disease Control and Prevention (CDC) lowers its PbB screening level to 10 µg/dL and its guideline for medical intervention to 20 µg/dL.
1992	Residential Lead-Based Paint Hazard Reduction Act requires EPA to develop standards defining hazardous levels of lead in lead-based paint, household dust, and soil.

[6]See Powell (1996a) for a discussion of lead in soil at Superfund mining sites. Another issue of contention is the tradeoffs between promoting recycling and limiting emissions from lead battery recycling.

Table A-1. Background on U.S. Lead Regulation and Policy (cont'd)

1994	Based on a finding that abatement of lead in soil above 500 ppm can achieve "measurable" reductions in children's PbB, EPA issues Revised Interim Soil Lead Guidance for CERCLA sites and RCRA Corrective Action Facilities.
1995	EPA proposes Maximum Achievable Control Technology for secondary lead smelters under the Clean Air Act Amendments of 1990.

Table A-2. Timeline of the 1991 Lead/Copper Drinking Water Rule.

1974	Safe Drinking Water Act requires EPA to establish health-based goals and enforceable regulations to protect public health with an adequate margin of safety.
1975	EPA sets an "Interim" lead in drinking water MCL of 50 ppb.
1977	National Research Council's Safe Drinking Water Committee suggests EPA's interim MCL for lead may not provide a sufficient margin of safety. OAR develops first Air Quality Criteria for lead.
1982	OAR begins review of lead NAAQS.
1984	ORD/ECAO/Cincinnati's Health Effects Assessment for Lead, prepared at request of OSWER, classifies lead as a probable human carcinogen (group B2). (Note that OSWER has interpreted Superfund ARARs (applicable or relevant and appropriate requirements) for groundwater contamination to include drinking water MCLs.)
1985	ODW draft Water Criteria Document developed (never finalized). ODW proposes revised lead in drinking water standard, an MCLG of 20 ppb.
1986	Safe Drinking Water Act Amendments list lead among 83 contaminants for which EPA required to develop MCLGs and NPDWRs. OAR's revised Air Quality Criteria for Lead suggesting no discernible threshold for indicators of lead exposure endorsed by CASAC. OPPE issues *Reducing Lead in Drinking Water: A Benefits Analysis* (revised in Spring 1987). SAB Subcommittee on Metals suggests that EPA's Drinking Water Health Advisory for PbB of 15 µg/dL is too high.
1988	Draft provision in Lead Poisoning Prevention Act that would have contained a drinking water MCL for lead measured at the tap is deleted. Lead Contamination Control Act calls for monitoring, recall of water coolers with lead-lined tanks, especially from schools. ORD's Cancer Assessment Group (CAG) issues *Review of the Carcinogenic Potential of Lead Associated with Oral Exposure*, prepared at request of ODW and OSWER, characterizing lead as a probable human carcinogen (group B2). EPA proposes an MCLG for lead of zero with an MCL of 5 ppb measured at the source and corrosion control triggered at 10 ppb based on tap samples. Debate within EPA over contribution of lead in water to blood lead levels. SAB Drinking Water Subcommittee concludes that neither a zero MCLG or an MCL of 5 ppb had been justified on a public health basis. SAB Executive Committee forms ad-hoc Joint Study Group, including members of Environmental Health Committee and CASAC, to review agencywide lead issues.
1989	CASAC reviews lead NAAQS Staff Paper and Criteria Document Addendum. SAB Joint Study Group on Lead agrees with B2 carcinogen classification for lead; finds inconsistencies among research, air, and drinking water programs regarding threshold PbB of concern and sensitive population; and recommends that EPA develop an agencywide lead strategy based on preventing adverse neurological effects in children.

Table A-2. Timeline of the 1991 Lead/Copper Drinking Water Rule (cont'd)

1990	With disagreement over health-based goal largely put to rest by CASAC's finding of no discernible threshold for effects, debate centers on costs and benefits of alternative regulations.
1991	Rep. Henry Waxman (D-CA) fails to pass legislation mandating an at-the-tap MCL for lead. EPA uses unpublished CDC report to estimate a relationship that drives a substantial portion of the estimated regulatory benefits. A later version of the report reduces the estimated relationship by a factor of 3. The CDC report is never published.
	EPA issues final rule. MCLG for lead set at 0 and NPDWR requires corrosion treatment, lead service line replacement, source water treatment, monitoring, and education triggered at 15 ppb based on tap sampling scheme.

2. Scientific Issues

In general, virtually all of the respondents (90%) believed that there was adequate scientific information available in 1991 to inform the decision to revise the lead drinking water standard. Most (80%) responded that the *quantity* of scientific information was abundant-to-very abundant, though the health effects information was consistently viewed as more abundant than the exposure information. A less robust majority (70%) responded that the *quality* of the scientific information was good-to-very good. A slim majority (60%) responded that the level of scientific uncertainty was small-to-very small. Information regarding lead exposure from drinking water (as opposed to health effects at given PbB levels) was perceived as contributing most to the uncertainty.

Early public health PbB screening levels were based (with safety factors added) on the levels associated with clinically observable lead intoxication, primarily of occupationally exposed adults. Over the past 20 years, health researchers have become aware of a suite of toxic effects occurring with lead exposures considerably below those associated with acute poisoning. NRC (1993) reports that the health effects noted at PbB of approximately 10 μg/dL include:

- impaired cognitive function and behavior in young children;[7]
- increases in blood pressure in adults, including pregnant women;
- impaired fetal development; and
- impaired calcium function and homeostasis[8] in sensitive populations.

NRC (1993) also concludes that somewhat higher PbB concentrations are associated with impaired biosynthesis of heme (a substance required for blood formation, oxygen

[7] More recent studies by Needleman and others suggest an association between cumulative low-level lead exposures and delinquent behavior in youths (e.g., Needleman, *et al*. 1996).
[8] This refers to maintaining calcium levels at the appropriate dynamic equilibrium.

transport, and energy metabolism) and cautions that some cognitive and behavioral effects may be irreversible.[9]

Because EPA conventionally assumes that the dose-response relationship for suspected carcinogens contains no threshold, the health-based goals (MCLGs) for suspected carcinogens in drinking water have been set at zero (in accordance with the 1974 House report language). In a position endorsed in 1989 by the SAB, EPA has classified lead as a probable human carcinogen since 1984. However, lead's carcinogenicity was rendered subordinate in the drinking water rulemaking by a consensus of opinion that lead's neurological effects in children and other non-cancer effects were more detrimental to public welfare than its carcinogenic effects. But until the lead rulemaking, the goals for drinking water contaminants regulated on the basis of non-carcinogenic effects had been set using the reference dose (RfD) approach. (The RfD presumes a threshold below which adverse effects would not be anticipated.) In some cases, however, the appearance of a threshold for non-cancer effects, if one exists, may be limited only by our technical detection capabilities. Acknowledging this, EPA's 1986 Air Quality Criteria Document for lead found that there was no apparent threshold for biomarkers of lead exposure (EPA 1986), and in 1990, CASAC concluded that there is no discernible PbB threshold for some lead health effects (CASAC 1990).

The prevailing scientific consensus regarding both the PbB level at which particular adverse effects can be expected to occur and the severity of those effects at particular PbB levels was bitterly fought over in scientific journals and courtrooms, and some lingering dissent remains within the scientific community. However, many scientists and analysts who were previously skeptical or uncertain about the validity of reports of adverse health effects from low-level exposure to lead have since come to conclude that the weight of evidence supports the earlier conclusions, particularly regarding neurological effects in children. Nevertheless, the lead in drinking water case needs to be considered in the context of the scientific controversy that coincided with the regulatory development.[10]

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[9] In an unprecedented decision, acting National Research Council chairman Robert White threatened to withhold the Academy's endorsement and distribution of the report of the Committee on Measuring Lead in Critical Populations (chaired by University of Maryland toxicologist Bruce Fowler) because the draft report addressed lead abatement, economic, regulatory, and policy issues outside the agreed scope of the ATSDR-sponsored study (*Science*, 7/30/93, p. 539). According to a member of the committee, the members took a "broad view" of how to address the ATSDR charge to the committee "and had the expertise to do so." This presumes, of course, that scientific expertise qualifies one to make judgments regarding regulatory policy issues. Ultimately, the NRC study was issued with slight modifications after ATSDR intervened (*Science*, 7/30/93, p. 539).

[10] Some may feel that the discussion of scientific issues gives too much weight to the arguments of a few skeptical scientists. As indicated above, many scientists who were previously skeptical about the health effects from low-level exposure to lead have since come to conclude that the weight of evidence supports the earlier conclusions of other scientists. My intent here is, to the extent possible, to avoid making substantive scientific judgments that I am unqualified to render, but as a disinterested party, to present the scientific issues and debates that are germane to the case. Even if one takes the view that the charges were trumped-up by parties with financial or personal interests (an argument that I am not making), it would be wishful thinking to believe that the controversy over the science was irrelevant to the 1991 lead

The Controversy over Neurological Effects in Children

In a 1979 issue of *The New England Journal of Medicine*, lead researcher Herbert Needleman of the University of Pittsburgh and his colleagues reported a drop in IQ of 3-4 points associated with "high" but non-acutely-toxic lead levels measured in children's teeth (Needleman *et al*. 1979).[11] In 1981, Claire Ernhart, a psychologist at Case Western Reserve University in Cleveland, and her colleagues argued in *Pediatrics* that Needleman had not done an adequate job of controlling for confounding variables that might explain the differences in cognitive performance (e.g., poor schools, parental neglect) and had performed so many comparisons that he was bound to come up with a few that were statistically significant merely by chance.[12] Ernhart and colleagues were also concerned that a large number of subjects had been eliminated from the analysis without a well-described exclusion procedure. Ernhart and colleagues suggested the lead effects were too small to be detected by a crude measure like IQ, except at some of the highest levels of exposure, just below acutely toxic levels.

When EPA began a review of the NAAQS for lead in 1982, Lester Grant, director of ORD's Environmental Criteria and Assessment Office (ECAO) convened an *ad hoc* subcommittee of CASAC to review Needleman's and Ernhart's work. Needleman claims that his refusal to share his data with industry precipitated the formation of the review panel. At the same time, according to Needleman, Grant was under pressure in a similar case because a carbon monoxide[13] researcher would not, or could not, produce his data. The review panel concluded that Needleman had used questionable measures to categorize lead exposure and had not provided sufficient justification for excluding particular subjects from the study. They also expressed concern about missing data, and some of the statistical analyses Needleman employed, all of which led them to conclude that the results neither support nor refute the hypothesis that low or moderate levels of lead exposure lead to cognitive or behavioral impairments in children.[14] The panel reached the same

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in drinking water decision while one of the principal scientist/advocates in the field was under investigation for scientific misconduct by the Department of Health and Human Services Office of Scientific Integrity.

[11] According to Needleman, the bulk of his epidemiological studies were supported by NIEHS, but his relationship with EPA began early. EPA first became aware of his work in the early 1970s via a "group of MDs" working in the ORD Health Effects Research Lab in Research Triangle Park, NC. Needleman's first paper on tooth lead levels was published in 1972, and EPA sent Needleman to Amsterdam to present it. EPA later provided additional research support.

[12] To maintain the global Type I error rate (probability of a false positive), only a limited number of significance tests can be run. Consequently, if a researcher were to perform a large number of statistical tests, some positive results are expected to occur by chance. Ernhart *et al*. (1981) was supported by a grant from the National Institute of Environmental Health Sciences.

[13] Carbon monoxide is a criteria air pollutant for which ECAO would prepare criteria documents.

[14] Ernhart *et al*. (1993) cite the panel's conclusions from p. 38 of EPA report 600/8-83-028A. A member of the expert panel states that the panel's report remained interim and was not intended for citation. It is not uncommon for some agency reports never to be finalized, either because they are controversial, superseded by more current reports, or simply not worth the time and effort to finalize.

conclusions about two of Ernhart's papers, which they also criticized for methodological flaws (*Science*, 8/23/91, pp. 842-844).[15]

According to Needleman, EPA then provided funding for him to reanalyze his original data based on more appropriate statistical methods (regression rather than analysis of covariance). By the time the review panel's report was presented to CASAC, both Grant and CASAC were convinced that Needleman's original conclusions were accurate, and they became part of the 1986 lead Air Quality Criteria Document (*Science*, 8/23/91, pp. 842-844). Studies in Boston (Bellinger *et al*. 1987) and Cincinnati (Dietrich *et al.* 1987),[16] as well as others, independently reported a relationship between low-level lead exposures and cognitive function in children. Needleman contends that EPA responded to the controversy "timidly" by stating, "even if we disregard Needleman's study, you get the same take home message." Thus, publicly at least, Needleman's work only contributed to the "weight of evidence" underlying EPA's lead policy. Notwithstanding EPA's public position, according to EPA officials, Needleman remained active and vocal in the process and was invited to brief drinking water program management. Two former senior EPA political appointees specifically recalled Needleman's work as being influential in the lead in drinking water decisionmaking.

Ernhart, however, continued to criticize Needleman's work and argue that the link between low-level lead exposure and neurological problems was being overstated. She also testified in favor of industry positions on phasing out leaded gasoline. In 1990 (in the interim between proposal and finalization of the lead in drinking water rule), the Department of Justice brought a Superfund suit against Sharon Steel, UV Industries and the Atlantic Richfield Company (ARCO). Each company had had a financial interest in a closed lead smelter in Midvale, Utah. The government intended to show that mine tailings on the site posed a health risk to children living in the area and hired Needleman as a lead expert. The corporations' lawyers brought in Ernhart as an expert witness and U of VA psychologist Sandra Wood Scarr, who had served on EPA's special review panel that had examined Needleman and Ernhart's research.[17] After briefly reviewing Needleman's original data analysis printouts, Scarr concluded that Needleman's first set of analyses failed to show any relationship between lead level and IQ, and that only by rerunning the analyses, eliminating important variables that might also cause changes in IQ scores, did the statistically significant relationship show up (*Science*, 8/23/91, pp. 842-844).

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[15] According to Ernhart *et al*. (1993), Ernhart submitted all of her data to the panel. Ernhart's Cleveland study, which was primarily funded by the National Institute of Alcohol and Drug Abuse, hypothesized that prenatal alcohol and lead exposures had an interacting effect on children's intellectual development, but the analysis did not confirm the effect. Although the inclusion of pregnant women with histories of alcoholism might have been regarded as a strength of the Cleveland study if it had detected a synergistic effect between lead and alcohol, according to an independent researcher, any *independent* effects of lead on children's neurological development may have been masked or swamped by effects of alcohol on fetal development, parental neglect, etc.

[16] According to an independent researcher, primary support for the Boston and Cincinnati studies was provided by NIEHS.

[17] Scarr has retired from U VA and is currently CEO of Kindercare Learning Centers.

The Superfund case was settled out of court before Scarr and Ernhart had presented their conclusions (for more on the story of lead in soil at Superfund mining sites, see Appendix H). Before the settlement agreement was announced, however, Department of Justice lawyers asked the court to force Scarr and Ernhart to return their notes on the Needleman data and refrain from speaking about what they had found. Contending that there was no good cause to suppress data gathered with public funds and that the government's request was an abridgment of First Amendment rights, Scarr and Ernhart fought the gag order and won. In turn, Scarr and Ernhart submitted their report to the NIH Office of Scientific Integrity (OSI) (*Science*, 8/23/91, pp. 842-844), leading to a University of Pittsburgh inquiry. Ultimately, Needleman was found guilty of sloppy statistics but cleared of scientific misconduct charges.[18]

A former SAB member remarked, "There was controversy about the Needleman data because industry-sponsored scientists charged that Needleman falsified the analysis. But there were ample international data that supported Needleman's conclusions. His statistics may not have been ideal, but you can't fault the conclusions that he drew." Responding to accusations that she was merely an industry mouthpiece, Ernhart claims she objected to Needleman's work before she began accepting research support from the International Lead Zinc Research Organization (ILZRO).[19] Scarr reportedly had no ties to the lead industry (*Science*, 8/23/91, pp. 842-844). A meta-analysis of lead studies reported by Needleman in 1990 (*JAMA* 263:673-678) is sometimes regarded as confirming his earlier work. However, meta-analysis is controversial, in large part because assigning weights to different studies is notoriously subjective (see: Mann 1990). An academic concludes that "Needleman's meta-analysis was subjective, with no explicit rationale for the weighting of various studies."[20] According to Sue Binder, formerly chief of CDC's Lead Poisoning Prevention Branch, it is extremely hard to find unaligned lead experts: "They will all go to their graves thinking the other side is made up of total idiots" (*Science*, 8/23/91, pp. 842-844).

Though perhaps not entirely responsible for their differences, disagreements over values contribute to the Needleman/Ernhart feud. While Needleman's career has centered

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[18] The Office of Research (formerly Scientific) Integrity of the Department of Health and Human Services (DHHS) accepted a University of Pittsburgh panel of inquiry's finding of no scientific misconduct on the part of Dr. Needleman. The ORI concluded that Needleman *et al*. (1979) and Needleman *et al*. (1990) inaccurately reported the methods and criteria for selection and exclusion of subjects, but found no resultant bias in the analytical results (ORI 1994).

[19] Ernhart's lead research also had support from the March of Dimes Birth Defects Foundation (see, e.g., Ernhart *et al*. (1986)).

[20] In a more recent review of 26 epidemiological studies since 1979, Pocock *et al*. (1994) determined that there is a statistically significant association between children's blood lead and IQ, with a doubling of PbB from 10 μg/dL to 20 μg/dL associated with a mean reduction in IQ of around 1-2 points. The finding is perhaps most noteworthy because Pocock has been a noted skeptic of a causal relation between lead and IQ. Pocock *et al*. (1994) conclude that while low level lead exposure may cause a small IQ deficit, "the degree of public health priority that should be devoted to detecting and reducing moderate increases in children's blood lead, compared with other important social detriments that impede children's development, needs careful consideration."

almost exclusively on lead (he chairs the U. of Pitt. Medical Center's Lead Research Department), Ernhart's research has addressed other risk factors in early childhood development such as fetal alcohol exposure.[21] Although many environmental advocates, researchers, and officials are quick to dismiss or vilify Ernhart and her associates for fraternizing with industry, perhaps they should not be so quick to dismiss the *sentiment*--based on judgment, not science--that concern over low-level lead exposures may not warrant the same attention and resources as more manifest risk factors such as poor nutrition, abuse, and random violence. As one academic commented, "there are plenty of risks that we know about that are certain and are certainly large. These are the things we should go after, rather than small, uncertain risks." Viewed from this perspective, the fight is over tradeoffs and about priorities.

Effects Related to Blood Pressure

In the 1980s, EPA researcher Joel Schwartz (see discussion below) determined a relationship between blood lead and blood pressure among adult males in the U.S. on the basis of data from NHANES II, the second National Health and Nutrition Examination Survey (e.g., Schwartz 1988).[22] An independent researcher reported, "There's a general consensus now among the non-occupational hygiene folks that there's a blood pressure effect" due to lead.[23] However, the specific relationship between increased blood pressure and more serious cardiovascular outcomes (such as hypertension, stroke, heart attack, and mortality) remains highly uncertain. One reason that there is less confidence in the outcomes for effects related to blood pressure than there is for the neurological effects in children, according to the researcher, is that whereas the epidemiological studies of cognitive impairment in children measured the same children over time (longitudinal), the studies relating blood pressure to other effects compare different groups in a given period (cross-sectional). The effects of lead on blood pressure may also be hard to detect given the comparatively large effects of other variables such as diet and smoking on blood pressure.

The 1991 rulemaking did not attempt to quantify the effects of lead on fetal development and other health endpoints due to the uncertain state of the science. (See further discussion below concerning unquantified health effects.)

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[21] In general, the community of lead researchers has been shaped to a great extent by a self-selection process in the academic community. An environmental lawyer suggests that as a result of sustained federal support for research into lead's health effects, it is one of the few areas of pollution control for which there is a sizable number of scientists not affiliated with industry.

[22] NHANES is conducted by CDC's National Center for Health Statistics. NHANES II covered 1976-80. Schwartz first reported the analysis in a 1986 memorandum to ECAO's Lester Grant in relation to the lead air quality criteria.

[23] The field of occupational hygiene is viewed by some as being industry-oriented.

Exposure Analysis

In assessing the magnitude of risks associated with lead in drinking water, EPA had to evaluate the various sources of lead exposure, the occurrence of lead in drinking water, drinking water consumption patterns, and the water lead to blood lead relationship. Overlapping the debate over lead's health effects and the PbB threshold of concern was another regarding the relationship between PbB levels and lead exposures from various media, including air, food, paint, dust and soil, and water. As an EPA official formerly in ODW recalls, "background levels of lead were lowered due to the phase-out of lead in gas, and population blood lead levels were declining measurably. As a result, drinking water was a larger portion of a smaller total." While the lead in drinking water rule was being formulated, a multi-media uptake model developed by EPA as a tool for the lead NAAQS was available, though unvalidated.[24] Using this model, the agency estimated in the proposal that, on average, the typical drinking water contribution to total lead exposure for a 2-year-old child is about 20 percent (EPA 1988). At the same time, ATSDR (1988) concluded that lead paint and contaminated urban soils were the main sources of lead exposure for children with PbB above the screening level (10 μg/dL).

Estimating exposure was complicated because survey data on the presence of lead in drinking water at the tap were crude and scarce, and data on drinking water consumption patterns were even more so. According to an EPA official, researchers that were key in the area of lead occurrence in drinking water included Peter Karalekas, formerly an EPA Region I (Boston) engineer, and Michael Schock, a chemist and metal corrosion expert then with the Illinois State Water Survey and now with EPA/ORD in Cincinnati (see, for example, Karalekas *et al*. (1976) and Schock and Wagner (1985)).[25] Given the complexity of water chemistry and its importance in assessing lead occurrence in drinking water, Schock's rare corrosion expertise was particularly valuable. According to an EPA official formerly in ODW, "water chemistry is among the hardest to do. Water is the universal solvent, so everything is a contaminant, many of which then affect the physical properties of the water. How aggressive the water is in attacking lead in pipes depends on the water composition."

Given these complexities, modeling estimates of lead occurrence were of limited value, and quality monitoring data were most needed to accurately and precisely assess lead exposures via drinking water. In 1981, the ODW reported a national study of lead levels in drinking water, but it was based on partially-flushed (30 sec.) convenience (i.e., nonprobabilistic) samples (Patterson 1981). Early in the analytical process, there were

[24] According to Needleman, the model has since been tested and does "a pretty good job in relation to empirical data." However, the validity of the model remains an issue under debate, says an EPA official. In large part, the question of model validation hinges on the extent to which the generic model has to be modified for site-specific applications. See Powell (1996a) for further discussion of EPA's Integrated Exposure Uptake Biokinetic Model for Lead in Children (or IEUBK Model).

[25] Schock had been with ORD Cincinnati prior to joining the Illinois State Water Survey. He later rejoined the EPA laboratory and was a member of the regulatory development working group on lead in drinking water after the 1988 proposal.

limited metropolitan survey data regarded as statistically more reliable, including studies in Boston and Chicago. After the proposal, individual drinking water utilities submitted survey data as public comments, and in 1988, the American Water Works Service Company (AWWSC) conducted a national survey. Although some respondents viewed the survey data as important, it did not yield a rock-solid foothold for the exposure assessment. A former ODW official commented, "the utilities conducted their own peer review, the agency hired external statisticians to evaluate the information, and there was further analysis by in-house staff. The agency spent a lot of time and money on it, and ultimately, different people had different takes on it."

Drinking water consumption patterns were an important consideration in the exposure assessment because lead measurements in tap water display considerable variability. For example, lead from plumbing tends to accumulate in the standing water over time, so that the first flush from a faucet in the morning contains overnight accumulations. There is also substantial variability even with repeated samples, e.g., same house, same tap, first flush. As a result, "any particular measurement should be taken very cautiously," concludes EPA biostatistician Allan Marcus, who contributed to the lead in drinking water analysis as an EPA contractor with Battelle.[26] The drinking water office, according to a former ODW official, contracted out for a "quick survey" on children's drinking water consumption patterns, but it was not used because it suggested that "children rarely drink the first flush from the tap in the morning." (On the other hand, the survey may have been disregarded because it was viewed as statistically unreliable.) In any event, the final rule did not specify the presumed underlying drinking water consumption pattern.

In addition to considering the drinking water consumption profile, another aspect of the exposure analysis is the relationship between the concentration of lead in drinking water and resultant blood lead levels in various sub-populations. In the proposed rule, EPA relied on a 1983 study supported by the FDA that correlated lead in milk (canned formula and breast fed) with infants' PbB (Ryu *et al.* 1983) and the "Glasgow Duplicate Diet Study" (Lacey *et al.* 1985) to develop a "correlation" factor (i.e., a non-threshold linear relationship) for predicting PbB from drinking water lead concentrations. EPA's water lead to blood lead relationship developed for the proposal was criticized by public commenters. The agency contracted Allan Marcus to reanalyze the Ryu and Lacey studies, along with a study of Edinburgh school-age children (Laxen *et al*. 1987). Marcus developed a non-linear relationship between water lead and blood lead levels in children that is more consistent with what is known about lead pharmacokinetics. In promulgating the final rule, EPA also concluded that it was better to rely on the Glasgow study for indicating responses among infants because it relied on exposure through drinking water (*Fed. Reg.*, Vol. 56, p. 26469). EPA relied on a British study (Pocock *et al*. 1983) for responses in adults. For responses in children older than 6 months, the final rule relied not on the reviewed Edinburgh study but on another study (Maes *et al*. 1991) which had been submitted for publication--and therefore not published in a peer-reviewed journal--by staff

[26] Marcus currently works in ORD/ECAO.

of CDC's Center for Environmental Health and Injury Control. The CDC study evaluated Hawaiians exposed to lead in drinking water. The Edinburgh and Hawaiian studies were noteworthy in that they both attempted to control for other sources of lead exposure (e.g., dust and soil) in addition to lead from drinking water, but they provided differing estimates of the relationship between water lead and blood lead in older children. (See discussion below regarding these studies.)

3. Process Within EPA

Setting the Agenda

Several factors contributed to getting the issue of lead in drinking water on the agency's agenda. Certainly, it was part of an overall strategy and an extension of the agency's program addressing lead in air. An academic expressed the view that EPA's entire lead strategy was developed in response to the blow to the agency's reputation in the wake of the scandals of the early 1980s. In this view, "lead was a convenient target," and lead in drinking water was simply a continuation of the agency's attempt to burnish its image with a formula that had proven effective in the lead in gasoline phaseout. On the other hand, an agency insider who was skeptical of the health benefits from reducing lead in drinking water found the agency's lead strategy to be "an honest attempt by the administration to find the intersection between high risk problems and things people care about."

The factors most frequently mentioned by respondents as keys to getting lead in drinking water on the agency's agenda were congressional pressure and a statutory deadline for review of the drinking regulations imposed by the SDWA. According to a former senior agency official, "my first meeting in [the agency] was with a congressional staffer who was concerned that we were very far behind our statutory obligation on drinking water standards. Of the 83 contaminants that Congress had set in '86 Safe Drinking Water Act Amendments, only 35 had been regulated. Too many things were in the pipeline."

However, congressional forces had allies among a core group of agency staffers. According to a former ODW official and others, the "Lead Mafia" was primarily responsible for pushing the lead in drinking water issue up the agency's agenda. Informally leading this group of policy entrepreneurs in the case of lead in drinking water was Ronnie Levin of OPPE. Levin's 1986 benefits analysis estimated substantial net economic benefits from reducing lead in drinking water (Levin 1986).[27] Joel Schwartz, who is Levin's husband and was also an OPPE official at the time, had been conducting statistical and economic analysis of lead epidemiological data for many years and,

[27] Levin (1986) estimated materials benefits from reduced corrosion damage ($525.3 million per year) alone exceeded estimated compliance costs ($239 million per year).

according to a former drinking water official, "did work that was so complex that it was unintelligible to most within EPA."[28]

According to press reports, an executive summary of Levin's analysis was made public in November 1986 to preempt another study being prepared by a group led by Ralph Nader (*Inside EPA*, 11/14/86, p. 13). The public release of the benefits analysis sparked citizen group criticism and media attention of then-Assistant Administrator for Water Larry Jensen's decision to conduct further analysis prior to promulgating revised lead drinking water standards. After an internal review of her first draft conducted by ORD, Levin produced a "Draft Final" benefits analysis that was released in December 1986 (and revised Spring 1987).[29] Internal allies allowed that Levin's regulatory analysis relied on limited exposure data[30] and her water chemistry analysis was "not as precise as some would have liked" (see further discussion below). Furthermore, approximately half of the monetized health benefits in Levin's analysis were associated with highly uncertain reductions in hypertension, heart attacks, strokes, and mortality in males aged 40 to 59.[31] One EPA official speculates that the studies linking exposure to blood pressure in middle-aged men were salient to policymakers. "It was no longer them, those poor, highly exposed urban children; it became 'our' problem." Whether or not linking lead to cardiovascular effects in fact made the issue more salient to policymakers, and despite the warts and uncertainties in Levin's estimates, her analysis secured a spot high on the agency's agenda for lead in drinking water.

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[28] Schwartz is currently an Associate Professor of Biostatistics at the Harvard School of Public Health. Schwartz was also involved as an analyst in the particulate matter case study. See Powell (1996b).

[29] The internal review resulted in increased exposure and cost estimates, reducing Levin's benefits-to-cost ratio from 7:1 to 4:1 but maintaining the net economic benefits at about $800 million (*Inside EPA*, 12/12/86, p. 3).

[30] According to a former EPA official, one reason that Levin had to "scrounge around" for lead in drinking water exposure data was that EPA/ODW vetoed a survey. Although the ODW previously had reported a crude national survey (Patterson 1981), during the mid-1980's while CDC's National Center for Health Statistics was planning the first phase of NHANES III (covering 1988-91), the EPA Administrator's Office was convinced to commit additional funds for a more rigorous lead drinking water survey. However, says the former EPA official, the administration acquiesced when an ODW representative insisted that the funds be allocated to other research. An EPA official commented that the drinking water program management felt that sufficient information was available on the occurrence of lead in drinking water and that waiting for new survey data from the NHANES III study could have lead to unnecessary delay in the rulemaking.

[31] The ODW Final Regulatory Impact Analysis (Wade Miller Associates and Abt Associates 1991) characterized the health benefits associated with cardiovascular effects as "conceivable," but not within the "estimated range" of benefits. According to an EPA official formerly in ODW, part of the reason that the office regarded the cardiovascular effects as "speculative" was that Joel Schwartz, who had conducted the analysis, would not permit access to the NHANES II data because Schwartz had accessed them in draft under agreement that they would not be distributed. Instead of sharing the data with other analysts, according to a colleague, Schwartz responded to methodological criticism of his analysis by rerunning the analysis until his critics "ran out of objections." EPA's Information Law Division is unaware of any current agencywide policy regarding the internal sharing of scientific data that is not designated confidential business information.

Assessing the Science

Apart from a 1985 ODW Water Criteria Document which was never finalized, the risk analysis specifically for lead in drinking water was conducted primarily as input to the economic analysis. "The scientific work on lead was very well known to Levin and Schwartz from the lead in gasoline debate," according to a senior EPA official, "but the drinking water office was largely ignorant of the scientific findings on lead due to the agency being compartmentalized." The "stove pipes" delineating people along programmatic lines also "extended beyond the agency to the environmental community whose drinking water experts were unaware of lead issues." As a result, comments this official, "the drinking water office and the environmental community jumped on the bandwagon late in the process." Richard Cothern, a former ODW scientist, recalled that the office spent a couple of years reassessing lead before it became aware of the air criteria documents. A former ODW official laments that Bill Marcus, the office scientist responsible for the lead review, was "was collecting rat data," mechanically following the protocol for developing an RfD from bioassay data. According to Cothern, once aware of the epidemiological data on neurological effects, and confident because "so much bright light had been shone on the studies by then," some of the ODW technical staff, including Marcus and himself, promoted lead on the drinking water agenda because they "felt that cancer got too much attention. We looked at lead...as a serious non-carcinogen."[32]

However, others in ODW were in no apparent hurry to tighten the standards for lead and other contaminants. A senior careerist stated that within EPA "the Drinking Water Office is regarded as captured" by the regulated community. As evidenced by the deadlines imposed by the 1986 SDWA Amendments, Congress grew impatient with the drinking water program's pace of issuing and revising regulatory standards. Lee Thomas, who took over the agency during President Reagan's second term, replaced then-Director of ODW Vic Kimm with Mike Cook, who had worked for Thomas previously in OSWER. An academic remarks, "EPA's drinking water office is a peculiar institution. I don't understand how they get through conflict of interest guidelines. Members [of ODW] held positions in the AWWARF [American Water Works Association Research Foundation]. If someone in the air office were a member of EPRI [the Electric Power Research Institute], that would raise some eyebrows." An EPA official formerly in ODW stated, "Cook got sick of his in-line staff not finishing any of the regulations they were working on." He assigned Jeanne Briskin, who came to ODW from OPPE as a special assistant, to lead the Lead Task Force charged with developing a proposed revision to the lead drinking water regulations. While these personnel changes enabled the reconsideration of the scientific issues, they also resulted in an interpretation and use of science by people more inclined to regulatory action. "Personnel," as the adage says, "is policy."

[32] Marcus and Cothern presented a paper in May 1985 (Marcus and Cothern 1985) discussing the various non-carcinogenic effects of lead and the blood lead levels at which they may occur. They concluded that the lowest blood lead levels at which adverse effects occur is "about 10 μg/dL," the same level to which CASAC referred 5 years later.

An *ad hoc* organizational arrangement that coincided with the personnel shakeup also led to conflicting interpretations within EPA. Levin gained approval from Joe Cotruvo, then Director of the Criteria and Standards Division for ODW, prior to initiating her lead in drinking water benefits analysis, and a member of Schnare's staff was assigned to be a liaison with OPPE, according to EPA officials. The arrangement took advantage of the expertise on lead issues OPPE had accumulated in the context of the air program. Unlike other program offices which often relied on OPPE for economic analysis, however, ODW had its own economic analysis unit, and David Schnare was the section chief. According to an EPA official formerly in ODW, Schnare's unit was viewed as being "an internal OMB" (Office of Management and Budget). During development of the proposal, a bitter struggle between Schnare and Levin within EPA paralleled that between Ernhart and Needleman outside the agency.

Regarding the agency's risk analysis, typical comments by respondents suggested that agency guidelines and standard operating procedures were not applicable: "This wasn't a cookbook exercise." "EPA didn't have guidance for non-cancer risk assessment." In fact, however, there were standard operating procedures from which the agency departed in this case. As indicated above, the lead in drinking water rule eschewed the convention of a reference dose based on bioassay data. In addition, the exposure analysis was unconventional. For example, the exposure duration described in the agency's *Exposure Factors Handbook* (EPA 1989) for the standard exposure scenario for residential drinking water is in units of days, and given the infrequency of drinking water sampling, a drinking water contaminant concentration would more typically be treated as a constant over a period of months or years. However, the concentration of lead in drinking water was known to vary markedly within days. As well, lead concentrations from pipes newly joined with lead-containing solder (i.e., in new construction or as a result of repairs) remained high for a few years after installation. Thus, highly-aggregated average concentrations would not capture these "pulses" in the lead exposure profile. The departure from standard operating procedures, whether conscious or unconscious, resulted in protracted haggling inside and outside the agency over the appropriate sampling measures (e.g., first flush, second flush, fully flushed) and concentrations to employ for the analysis and monitoring of lead in drinking water.

According to an EPA official, behind this outwardly technical debate over the monitoring scheme were value judgments about the relative seriousness of false positives (type I errors) versus false negatives (type II errors). "There was a policy fight about how much to spend on monitoring." It boiled down to "how much uncertainty you are willing to live with," considering that costs started escalating at a sampling intensity that provided a 50-50 chance of a false negative. Conventionally, this rate of false negatives might seem unacceptable. On the other hand, if one's assessment of the science is that the standard provides a large margin of safety, then one might be willing to live with a relatively high rate of false negatives.

This debate over the monitoring scheme was an outgrowth of the agency's early decision to pursue a lead in drinking water control strategy based on required treatment

actions instead of a numerical Maximum Contaminant Level measured at the tap. According to an EPA research official, Michael Schock and his water chemistry colleagues in ORD laboratories in Cincinnati convinced members of the "Lead Mafia" that they had made overly optimistic assumptions about the efficacy of water treatment technologies, and that consequently, their desired MCL was infeasible. The achievable MCL (at the tap) that the "water engineering faction" favored, however, was considered too high by the "Lead Mafia," according to this source. Thus, to accommodate the limitations and uncertainty of the efficacy of the water treatment processes, the action level/treatment strategy was crafted as a staff-level compromise prior to the 1988 regulatory proposal.[33]

While virtually all of the respondents (90%) characterized EPA's treatment of the available science in the final rulemaking as good-to-very good, and a like number responded that the communication of the risk analysis to agency decisionmakers was very good, a minority felt that internal risk communications were poor as a result of the distorting influence of inter-office and inter-personal struggles. Others felt that the internal controversy, while unpleasant, forced many analytical assumptions to be disclosed and discussed at senior levels in the agency.

Something that went undisclosed to EPA decisionmakers, however, was a reanalysis of an unpublished CDC study (Maes *et al*. 1991) for evaluating the relationship between lead in drinking water and blood lead in children over the age of 6 months. In the final rule, EPA relied on an intermediate iteration of the CDC study which estimated that at water lead levels above 15 ppb (the water lead level triggering the treatment requirements), a one ppb increase in water lead is associated with an increase of 0.06 µg/dL blood lead (*Fed. Reg.*, Vol. 56, p. 26470). However, a later version of the CDC study available in the EPA drinking water docket estimates the same relationship as 0.02 µg/dL blood lead--a figure lower by a factor of 3.

According to a Public Health Service official, the CDC report underwent a number of iterations based on comments from statistical reviewers. An EPA official alleges that the final analysis of the CDC report that contained the 0.02 figure was available prior to the finalization of the drinking water rule, but that it was not published or shared with the EPA Lead Task Force because the people who had access to it and could block its publication as peer reviewers were members of the agency's "Lead Mafia." The Public Health Service official confirmed that the report has never been published.

The version of the CDC report filed in the docket suggests that the reanalysis was concluded *prior* to the final rulemaking. The abstract concludes, "These findings could have implications for the regulatory standards of water lead levels, currently being revised by the Environmental Protection Agency (Maes *et al*. 1991, p. 3). However, the date when the paper was logged-in to the docket was not registered. There is also no

[33] The 1988 proposal contained both an MCL measured at the source and a triggering level measured at the tap, but in the final rulemaking, EPA dropped the MCL measured at the source as an unnecessary complication.

addendum in the docket explaining the discrepancy with the figure used in the final rulemaking.

A member of the "Lead Mafia" recalled reviewing the CDC report in draft and providing comments suggesting that the estimated coefficient should have been higher. (According to this source, the blood lead level of the CDC study population was not equilibrated, and the authors had included too many separate variables in the statistical model in an attempt to control for the drinking water consumption rate.) This source stated that the authors of the CDC report could not accommodate their comments for reasons related to their statistical model. Also, this source claimed to be unaware of any discrepancy between the version of the report used in the final rulemaking and that which is in the drinking water docket.

Other sources have also criticized the CDC study design and analysis. The CDC analysis, for example, included both children and adults exposed to lead in drinking water. But this raises the question of why EPA used the earlier version of the study if it too was so flawed. In his review and reanalysis of the scientific literature as EPA's contractor, Marcus (1990) had concluded that the study of school children (ages 6-9) in Edinburgh, Scotland provided "the most useful data set" for estimating the relationship of water lead to blood lead in older children. Like the CDC Hawaiian study, the Edinburgh study (Laxen *et al*. 1987) attempted to control for other sources of lead exposure (i.e., dust) in addition to lead in drinking water. Marcus' reanalysis of the Edinburgh data estimated that at water lead levels above the action level, a one ppb increase in water lead is associated with an increase of 0.03 μg/dL blood lead (Marcus 1990). This figure is closer to that in the later version of the CDC study (.02) than to the estimate that EPA used in the final rulemaking (.06). A reasonable interpretation of the various estimates is that they simply indicate that there is a range of uncertainty in the relationship between water lead and blood lead in older children.

Determining whether the earlier or later version of the CDC paper or the Edinburgh study is technically superior is beyond the scope of this report. What is germane to this study is that there was a reanalysis of an important study that, for whatever reason, was not available to the regulatory development working group or to decisionmakers. If the information had been available, it might have prompted some assessment of the effect of the uncertainty in the relationship between water lead and blood lead on the estimated health impacts of the regulation prior to finalization. This episode underscores the *potential* for key intermediaries within EPA to withhold scientific information from decisionmakers. This potential exists due to the multiple, overlapping roles of intermediaries as producers of scientific information, peer reviewers, regulatory development working group members, and policy entrepreneurs. In light of the availability of the reviewed and re-analyzed Edinburgh study, this episode also emphasizes the need for EPA to consistently apply independent peer review to studies that substantially impact on major regulatory decisions.

Role of Agency Science Advisors

Most respondents concluded that non-agency scientists played a significant role in the decision-making process and in legitimizing the decision; however, some were clearly more influential than others. A few respondents regarded the SAB Drinking Water Subcommittee as unimportant in the process. The subcommittee, which was chaired by Gary Nelson of Purdue, did not endorse the 1988 proposal. Instead, the group that carried weight was the *ad hoc* Joint Lead Study Group. The Group, chaired by Arthur Upton, was formed at the request of the SAB Executive Committee and included members of the Executive Committee, the Environmental Health Committee, and CASAC.[34] The Group addressed the carcinogen classification of lead; pointed out differences among the research, air, and drinking water offices in target blood lead levels and the definition of populations at risk; and recommended that EPA regulate lead exposures on the basis of neurological effects in children. The broad purview of the Joint Study Group may have limited the extent to which it could conduct a detailed, comprehensive review of the proposed drinking water regulations.[35]

Officially, the drinking water panel, then a subcommittee of the SAB Environmental Health Committee, was requested by EPA to review the procedure for determining what an adequate tap sample is for measuring the drinking water lead concentration. However, the agency "didn't get much feedback from [the committee] on that," according to an EPA official. Instead, members of the drinking water panel commented that they found the health benefits analysis of the proposed MCL for lead "unconvincing" and characterized Levin's 1986 analysis as "sort of an advocacy type of document with a lot of stretching of notions."[36] According to one EPA official, "People [in EPA] blew off the Drinking Water [Subc]ommittee review. They [the Subcommittee] made ridiculous noises. It [the benefits analysis] was presented to them as a *fait accompli*." Another EPA official responded, "Nobody on their [drinking water] [Sub]committee knew anything about the relevant subject matter. There were microbiologists, but no statisticians, no health effects experts, no corrosion control experts." Another succinctly summed the staff's approach to the drinking water panel, "We tried to bulldoze it." Eventually, it was agreed to let the SAB Joint Lead Study Group review the health effects. The Group's "view of the health effects was going to take precedence," according to an EPA official. "We ended up supplementing the drinking water panel with Allan Marcus, our contractor," who provided the panel with statistical expertise.

An EPA staffer noted that the lack of endorsement by the Drinking Water Subcommittee "didn't make any difference. [EPA Administrator] Reilly didn't ask what

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[34] For the Group's report, see: EPA/SAB (1989).

[35] A member of the Joint Lead Study Group claims that the group did not sacrifice depth for scope, saying "we had several intense days of thrashing out the issues." As discussed below, however, an EPA official concluded that the lack of detailed external review of the proposed lead in drinking water rule created problems later in the regulatory development process.

[36] See the Lead Industries Association's summary of the transcripts of the June 2-3 1988 meeting of the Drinking Water Subcommittee in the EPA Drinking Water Docket.

the SAB thought of it." The response of one former senior EPA official was particularly telling, "Was the SAB involved on this? Did they approve or disapprove?"

However, another EPA official concluded that the lack of detailed external review at the proposal stage created problems later in the regulatory development process. "The Drinking Water Office tried to ignore the SAB Drinking Water [Subc]ommittee when it didn't tell us what we wanted to hear. They gamed it...to come up with a pre-determined answer." However, it resulted in "wasting a lot of time later in the drinking water office. Because they didn't get proper review, they had to go back and review" the occurrence, exposure, and benefits analyses in response to public comments prior to promulgating the final rule. In the end, therefore, it appears that the agency paid a price for shortchanging the quality control function during the proposal stage. For its part, the drinking water panel suffered an incursion on its jurisdiction by commenting on an area beyond its charge where it lacked acknowledged expertise.

Science Through the Lens of Policy

In addition to ignoring the SAB drinking water panel, the "Lead Mafia" overcame misgivings from colleagues in other agencies in pursuing their drinking water agenda. An EPA official remarked, "Some of the people in the lead health community who cared about the effects looked at lead in drinking water as a small exposure, a distraction. Vernon Houk from CDC [then-Director of the Center for Environmental Health and Injury Control] railed against doing much in drinking water because he didn't want to disarm lead in paint." Kathryn Mahaffey of NIEHS (and a member of the Joint Lead Study Group) was also mentioned in this regard.

After the 1988 drinking water proposal, Jeff Cohen, who transferred from OAQPS where he had managed the review of the lead NAAQS, took over the reins of the Drinking Water Office's Lead Task Force from Jeanne Briskin. Substantial amounts of the exposure and benefits analyses were performed or revised under the direction of the task force after the proposal. Many of the assumptions used during the proposed rulemaking were revisited and replaced. According to an EPA official, this was especially true regarding the analysis of the effectiveness of corrosion control treatment and lead service line replacement in reducing lead levels at the tap. Also, the benefits of reduced cardiovascular effects from lead in adult males were characterized as less certain. Ronnie Levin continued to be involved from her new post in the Office of Research and Development's regulatory evaluation group, but the aggressive tactics she successfully employed in setting the regulatory agenda were less welcome in the negotiations leading up to the final promulgation. From the Office of Drinking Water, David Schnare continued to attack the assumptions underlying the estimated health benefits of the rule and highlight the expense of compliance with the complex, prescriptive regulations.

A former ODW official commented that the internal advocates of the lead regulation acted in an "extremely partisan" manner on the basis of nothing definitive but rather a "gut feeling" about the science. "History," the official believes, "has shown them

to be right." However, a minority remains steadfast in opposition to the Lead Mafia's foray into drinking water. The lingering disagreement might be construed as questioning whether the policy entrepreneurs within the agency "got the science right" or attempted to inflate the quantifiable health benefits of the rule in order to compensate for an inability to quantify other benefits to the satisfaction of reviewers.[37] Like the disagreement between Ernhart and Needleman, the dispute boils down to policy differences. Regarding the lead in drinking water benefits analysis, one EPA official notes, "of the 23 million kids with benefits, 18 million of them got benefits of 0.1 IQ points. They were contending that by shifting the entire distribution [of children's PbB] slightly, that these small changes in the mean [of the distribution] are important. This is not an at-risk strategy. The way to manage risks is to attack the upper tail of the distribution!"

The lead in drinking water rule, however, was one component of the agency's multi-media lead strategy. In pursuing this policy, EPA took the *policy* position that "drinking water should contribute minimal additional lead to existing body burdens of lead" (*Fed. Reg.* Vol. 56, p. 2469). According to one EPA official, "we decided that every source should contribute as little as possible" due to what the agency considered a narrow margin of safety between typical levels of lead exposure in the general population and those associated with adverse effects. Recognizing that drinking water was not the primary source of exposure for the children at greatest risk, the official acknowledges, "We knew that we weren't going to bring lead-poisoned kids relief. The drinking water rule was a general population strategy rather than an at-risk strategy." In this view, pursuing small increments in risk reduction aggregated over a large population is a legitimate risk management strategy and can be just as worthy as targeting large risk reductions for a small sub-population under some circumstances.[38]

The success of EPA's "Lead Mafia" in setting the regulatory agenda in this case can be attributed in large part to an alliance of health effects scientists from ORD coupled with economists, statisticians, and policy analysts from OPPE. These offices and the technical disciplines from which they drew were able to trump drinking water experts inside and outside the agency. According to one EPA official, the drinking water staff has some "talented analytical chemists; the health effects area is where they are weaker." This

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[37] It is interesting to note that some spillover health benefits of corrosion control were not be captured by EPA regulatory analyses. According to an agency research official, EPA realized that there could be some ancillary health benefits, because implementing the corrosion controls would permit drinking water suppliers to use less chlorine to achieve the same level of disinfection, and thereby reduce the formation of hazardous disinfection byproducts. What EPA failed to recognize at the time, however, was that corrosion control would also lead to reduced microbial formation in the drinking water delivery system, for example, by reducing pitting of the pipes that provides microbes with tiny refuges where they are safe from contact with disinfectants.

[38] An EPA research official points out that the decision of which control strategy is adopted--attacking the upper tail of the risk distribution or shifting the entire distribution--has consequences regarding research design and risk assessment tools. EPA's IEUBK Model for Lead in Children, for example, predicts changes in mean PbB that would be difficult to convert into changes in the numbers in the tail of the distribution of children at risk from lead exposure. This official observes that the agency is sometimes not explicit regarding which strategy it is pursuing, making it more difficult to develop data and tools that will be useful for decisionmaking.

primacy of health-related disciplines was also reflected in the pecking order and makeup of the agency's official science advisory panels. The complexity and statistical sophistication of the Lead Mafia's regulatory analysis also appears to have been rewarded by considerable deference from decisionmakers and others without the time or ability to penetrate it.[39]

As a group of entrepreneurial staff which mobilized scientific arguments to advance a preferred policy, EPA's "Lead Mafia" is not unique. The principal reasons that such networks form are shared views and values and an agreement on the strategy of using science as a means to achieve the desired ends. If the direction of policy change that policy entrepreneurs seek to effect is guided by substantive concerns, they can represent a positive force in a political milieu that is responsive to constituent demands and ideological arguments. Such groups gain influence not only when their arguments are compelling on the merits but also when they resonate with policymakers. In this respect, the "Lead Mafia" had the wind at its back: the population of primary concern was children, agency policymakers were familiar with lead issues, and the initial regulatory analysis had suggested substantial net economic benefits.

4. Science in the Final Decision

Of those responding (7), all interviewees agreed that the level of consideration by agency decisionmakers to the scientific issues was thorough-to-very thorough. The means of risk communication to agency decision makers were diverse. According to an EPA official, "every possible path--memos, options meetings, review meetings, policy meetings, briefings, etc.--I've ever seen was used in that case." LaJuana Wilcher, who became Assistant Administrator for the Office of Water in 1989 was given staff briefings and traveled to ORD labs in Cincinnati to be briefed on corrosion research being conducted there. Deputy Administrator Henry Habicht was directly and deeply involved through a series of briefings and meetings. According to an EPA official, Administrator William Reilly got involved just before signing the rule because he was "hauled up before [Rep. Henry] Waxman's Subcommittee."

The factor most frequently cited by respondents as facilitating the consideration of science in this case was the agency's accumulated experience with lead issues in the air program. One EPA official recalled, "agency decisionmakers had had a lot of time to

[39] An EPA research official comments that those with the time and ability to penetrate the Lead Mafia's statistical analysis also found it impressive. "It's not always clear when complexity is ornamentation and when it is necessary," says this official, "but there were good reasons for complexity in this case." A member of the Lead Mafia observes, however, that the increased sophistication of EPA's use of scientific information has "created a black box" that makes the risk assessment process less transparent to regulatory program managers and policymakers. Thus, the increased complexity of risk assessment makes it easier for undisclosed assumptions to be buried, consciously or unconsciously, in the analysis and, in some cases, may shift decisionmaking power within EPA from program managers and policymakers to risk analysts.

think about the issues. We were able to benefit from the agency history on the issue with all of the experience on the air side. We didn't have to start at ground zero." Another agency official felt that the high stakes in terms of compliance costs associated with the rule "caused the decisionmakers to focus on the substance...and devote a lot of time to the substance of lead." But others pointed to the receptivity of decisionmakers, particularly Deputy Administrator Habicht, to being involved in substantive matters. Other factors mentioned as facilitating the consideration of science were the estimated net economic benefits of control, the internal advocacy and analysis roles of the "Lead Mafia", and the broad consensus among health scientists inside and outside the agency about the effects of lead.

A former senior EPA official found, "there was more science on lead than on most things that we regulated, direct epidemiological data on the relationship between blood lead and childrens' IQs." The fact that "the science was based on epi [epidemiological] data rather than animal studies...made it more reliable" in this official's judgment. Another former senior official, commented that the decision was "driven by real world epi evidence on lead" and stated that science played a particularly large role in the lead/copper rule. "There was adequate science to inform the decision to revise the standard," according to this official. "The big question was how far to go. Should all lead pipes be replaced, including the service lines? Was the science good enough to justify large expenditures?" The answer, of course, depended on the estimated benefits of the rule, and to the extent that the agency's use of science increased the regulatory benefits estimate, it could rationalize more stringent and costly monitoring and treatment specifications. During the final stages of the rulemaking, the lead/copper drinking water rule was detained by OMB reviewers, but ultimately in a departure from normal procedure, Deputy Administrator Habicht signed-off on the final rule as Acting Administrator without OMB clearance.

"There was adequate science to inform the decision to revise the standard," according to a former senior EPA official. "The big question was how far to go." The answer, of course, depended on the estimated benefits of the rule.

Although the high stakes in the lead rule may have been responsible for causing some agency decisionmakers to focus on scientific matters, the factor most frequently cited as impeding a thorough consideration of the science was the high cost of compliance ($500-$790 million per year). A former senior agency official concluded that "the high level of emotion impeded the use of detached scientific information." Others pointed to the personalized controversies, i.e. Levin v. Schnare inside the agency and Needleman v. Ernhart outside, and turf battles between ORD and OPPE, on the one hand, and ODW, on the other. Another factor was that the much of the exposure and benefits analysis remained to be done after the proposal prior to finalization, and given the level of congressional scrutiny, the 1991 deadline was not one that could be missed without consequences.

5. Concluding Observations

Deputy Administrator Habicht's willingness to engage in substantive debates effectively created high-level demand for analysis of the existing science. However, given the rate of turnover of agency political appointees (whose tenure is typically limited to two or three years), there are limits to the role of agency leadership in creating the demand necessary to generate original research. Basic research has the potential of filling data gaps in regulatory science analysis but often requires several years to complete. The short time-horizon of politicos helps to explain the chronic failure of strategic planning for environmental regulatory science. In terms of a fate and transport analogy, there is frequently no feedback loop between the ultimate sources of science (i.e., basic researchers) and the endpoints (i.e., regulatory decisionmakers).

Continuing with the fate and transport analogy, an EPA official formerly in ODW observed that the epidemiological studies regarding neurological impairment in children exposed to lead which had been relied on so heavily in the air program formed "an existing body burden" of science in a separate "body compartment" within the agency that was unusual. This body burden was formed by a series of longitudinal epidemiological studies of children involving some measure of cognitive function which included the work of Needleman and colleagues as well as studies by Bellinger *et al*. (1987) and Dietrich *et al*. (1987).

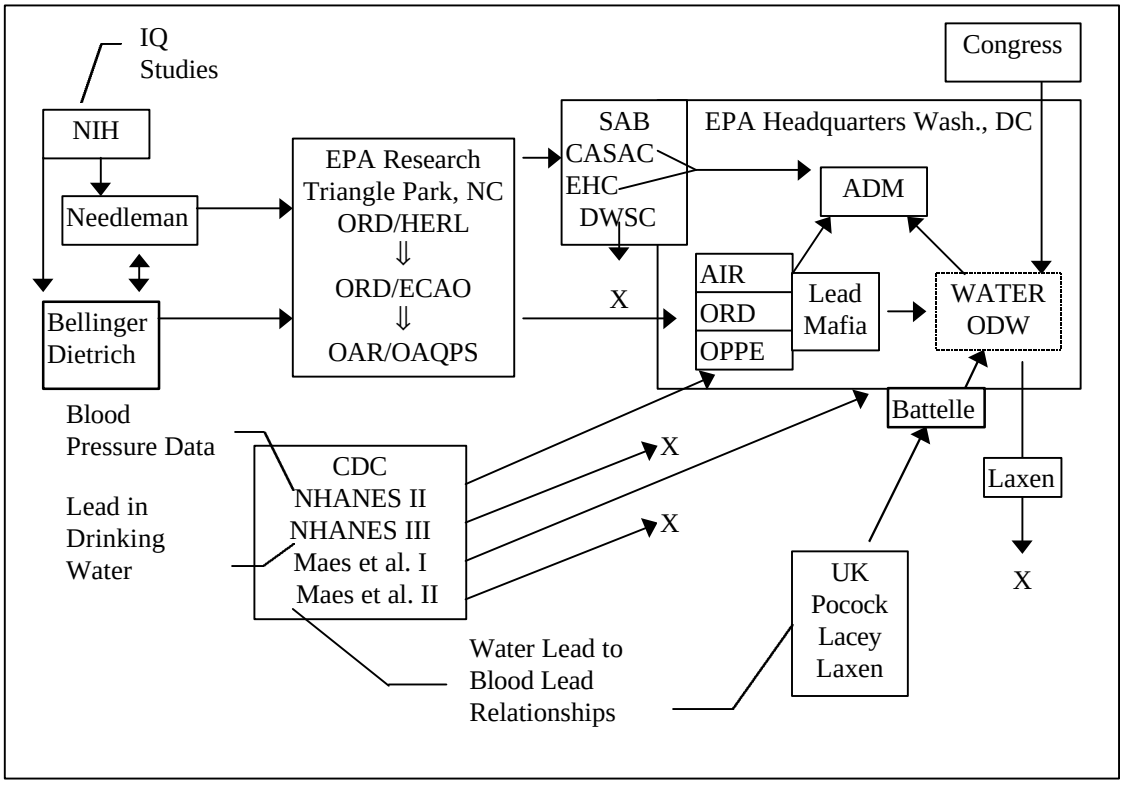
The major source of support for the domestic studies was NIEHS. According to an independent scientist, the Institute's involvement in lead dates back to the early 1970s when NIEHS was under the directorship of David Rall. This researcher suggested that EPA had little or no role in creating the demand for this science: "EPA wasn't in a position to be making demands on its sister agencies until the early '80s." EPA's initial "exposure" to the accumulating epidemiological database was through ORD's Health Effects Research Laboratory (HERL) and Environmental Criteria and Assessment Office (ECAO) in Research Triangle Park, NC. The information accumulated first in the air program "compartment" of the agency, and the further analysis, review, and legitimacy it received there made it both "bioavailable" and "potent." It was also in this context that agency decisionmakers first became exposed and "sensitized" to the information.

The basic science regarding the health effects of lead was supported by other agencies. However, EPA's official science advisors had to confer legitimacy on the science before it was available to the agency for use in regulatory decisions. EPA also had to expend considerable resources "metabolizing" (reanalyzing) existing or newly generated exposure information to render it useful for regulatory purposes. It also required the data gathering, analysis, and advocacy of a group of internal policy entrepreneurs to "transport" the information into the drinking water program and overcome "barriers" (in terms of standard operating procedures and culture) to "assimilation" in this compartment. At the same time, these barriers were becoming more permeable, promoting interchange of information among the agency's body compartments, as a result of high-level personnel changes stemming ultimately from external political pressures to increase the drinking water office's rate of developing and revising drinking water standards.

The controversies inside and outside the agency over the science appear to have had both distorting *and* illuminating effects. In addition, certain data either was never available or was "excreted" in the process. For example, ODW derailed plans for a national lead in drinking water survey under NHANES III. The "quick" (and presumably dirty) ODW drinking water consumption pattern survey was rejected. In some cases, data that was used was "selectively available." For example, both Needleman and Schwartz refused to allow their opponents access to raw data. Also, a CDC manuscript that could have lessened the estimated benefits of the final rule was unavailable to the regulatory development working group and to decisionmakers.

Figure A-1 illustrates the fate and transport dynamics for some of the key sources of scientific information in the lead in drinking water rulemaking.

Figure A-1. Fate and Transport Dynamics for Science in the Lead in Drinking Water Decision.



Outside the agency, the 1986 SDWA Amendments ban on lead in plumbing reduced the incentives for the lead industry to mount a serious counter-argument to applying the epidemiological data in the drinking water program area. Although the drinking water utilities generally protested the lead regulations, their ability to critique, minimize, or distort the epidemiological findings were extremely limited by the sector's lack of expertise on the health effects of lead and by the pre-existing legitimacy conferred on the science by the environmental health scientists operating in the air program.

Epilogue: Conflict over the lead in drinking water rule continued after its promulgation. For example, shortly after finalization, the California Department of Health Services informed EPA that the state would not initiate the process for implementing the lead/copper rule due to a lack of resources (*Inside EPA*, 11/22/91, p. 1). In response to a 1994 D.C. Circuit Court remand resulting from Natural Resources Defense Council and AWWA challenges to the 1991 lead and copper rule, in April 1996, EPA proposed revisions to the rule to eliminate a number of requirements and to clarify conditions under which lead service line replacement would be required (*Fed. Reg*., Vol. 60, pp. 16347-16371).

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LIST OF ABBREVIATIONS

AO	Administrator's Office
ATSDR	Agency for Toxic Substances and Disease Registry
CAG	Cancer Assessment Group
CASAC	Clean Air Scientific Advisory Committee
CDC	Centers for Disease Control and Prevention
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
ECAO	Environmental Criteria and Assessment Office
EPA	Environmental Protection Agency
FDA	Food and Drug Administration
HERL	Health Affects Research Laboratory
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
µg/dL	micrograms per deciliter
NAAQS	National Ambient Air Quality Standard(s)
NHANES II	Second National Health and Nutrition Examination Survey
NHANES III	Third National Health and Nutrition Examination Survey
NIEHS	National Institute of Environmental Health Sciences
NPDWR	National Primary Drinking Water Regulation
NRC	National Research Council
OAQPS	Office of Air Quality Planning and Standards
OAR	Office of Air and Radiation
ODW	Office of Drinking Water
OMB	Office of Management and Budget, White House
OPPE	Office of Policy, Planning and Evaluation
ORD	Office of Research and Development
OSWER	Office of Solid Waste and Emergency Response
PbB	blood lead
ppb	parts per billion
ppm	parts per million
RCRA	Resource Conservation and Recovery Act of 1976
RfD	reference dose
RTP	Research Triangle Park, North Carolina
SAB	Science Advisory Board
SARA	Superfund Amendments Reauthorization Act of 1986
SDWA	Safe Drinking Water Act of 1974

B. The 1995 Decision Not to Revise the Arsenic Drinking Water Rule

1. Background

Arsenic is a naturally occurring toxic substance found in drinking water supplies of the United States and other countries. Arsenic is also found in food, but most of the arsenic in food occurs in organic forms that are much less toxic than inorganic forms of arsenic (Abernathy and Ohanian 1992).[40] Prior to the development of synthetic pesticides in the 1940s (and for a considerable time thereafter), inorganic arsenical compounds (e.g., lead arsenate and copper acetate arsenate, "Paris Green") were widely used in agriculture. Arsenic is still used commercially in wood preservatives and is also released into the environment as a result of smelting nonferrous metal ores, particularly copper. Because arsenic does not degrade in the environment, contamination from historical releases is cumulative.

In 1942, the Public Health Service (PHS) set a 50 ppb standard for arsenic in drinking water based on information about its acute poisonous effects.[41] In 1962, the PHS recommended a limit of 10 ppb. In response to the 1974 Safe Drinking Water Act (SDWA), EPA adopted an "interim" standard of 50 ppb in 1975. The 1986 SDWA Amendments required EPA to finalize the Maximum Contaminant Level Goal (MCLG) and the enforceable Maximum Contaminant Level (MCL) for arsenic by 1989. After the deadline passed, a group of citizens in Oregon, commonly referred to as the Bull Run Coalition, sued EPA for failure to comply. The agency negotiated, and missed, a subsequent series of Court-decreed milestones for revisiting the standard. At each step, the agency cited the need for further research. In January 1995, Robert Perciasepe, EPA Assistant Administrator for the Office of Water, filed a declaration with the US District Court of Oregon stating that the agency would be unable to propose a new standard for arsenic by the court-ordered deadline of November 30, 1995. Perciasepe cited remaining uncertainties in the health risk assessment for ingested arsenic and the arsenic control technologies and the "extremely high costs" that some regulatory options would impose on public water systems, "especially on a large number of smaller systems" (Declaration of Robert Perciasepe, Amended Consent Decree, *Donison, et al. v. EPA*, No. 92-6280-HO (and consolidated cases) U.S.D.C. Oregon, January 9, 1995).

Over the past 20 years, EPA has addressed other sources of arsenic in the environment. In 1978, EPA began a review of remaining inorganic arsenical pesticide uses.[42] By 1988, EPA had banned all pesticidal uses of inorganic arsenicals except wood-

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[40] Seafood in particular contains high levels of organic arsenic. For the majority of the US population living in areas with low arsenic levels in their drinking water, food is most likely to be the major source of inorganic arsenic ingestion.

[41] Parts-per-billion (ppb) is considered equivalent to micrograms per liter (μg/l). In this context, acute poisoning refers to high doses administered over a short duration, typically to calculate a lethal dosage for a given percentage of test animals.

[42] Many uses of inorganic arsenicals as agricultural pesticides and defoliants were banned in 1967 prior to EPA's establishment (CAST, 1976, p. 26).

preservative products. Since September 1993, all agricultural uses of the inorganic arsenicals have been prohibited (www.epa.gov/docs/fifra17b/Arsenic_Acid.txt.html). Arsenic was also one of the original seven hazardous air pollutants (HAPs) listed by EPA between 1970-84 under Sec. 112 of the Clean Air Act. In 1983, EPA proposed a national standard for arsenic emissions from copper smelters.[43]

Arsenic is also an important issue in EPA contaminated site and waste management programs. Arsenic is a key contaminant at many abandoned mining, milling, and smelting sites. Under current regulatory practices, current *or proposed* drinking water MCLs or MCLGs are frequently the operative Superfund remedial objectives for groundwater contamination.[44] The Reference Dose (RfD) for ingested arsenic provided in EPA's Integrated Risk Information System (IRIS), may be used in establishing remedial objectives for soil contamination. Arsenic also represents approximately half of the estimated carcinogenic potential in coal fly ash (SEGH 1994), a waste generated by coal-fired power plants. Since 1976, electric utility wastes have been exempted from the hazardous waste provisions (Subtitle C) of the Resource Conservation and Recovery Act (RCRA) (TNCC 1995). Under current hazardous waste program practices, however, there is a linkage between the MCL for a substance in drinking water and the RCRA regulations covering its treatment, storage, and disposal.[45] As a result of the considerable spillover effects that could result from changes in the MCL for arsenic (or the RfD for arsenic), the attentive regulated community is not limited to public drinking water suppliers.

Recent debate over the arsenic drinking water standard also occurs in the broader context of the debate over "unfunded mandates."[46] The regulatory compliance burden on local governments from the Safe Drinking Water Act is a prime focus of this debate. Most

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[43] The only plant in the nation that would have been affected by the standard was the ASARCO copper smelting plant outside Tacoma, Washington. After considerable public debate and input regarding the balancing of economic and health considerations, however, declining world copper prices forced the ASARCO plant to close. As a result, EPA never issued final regulations (Landy *et al*. 1994, pp. 253-254).

[44] This practice arises from the ARARs (applicable or relevant and appropriate requirements) provision of the 1986 Superfund Amendments and Reauthorization Act (SARA) (Walker *et al*. 1995). See GAO (1996) for a discussion of the variety of means used by states in setting groundwater standards for contaminated site cleanups.

[45] Approximately 70% of coal combustion waste products are currently managed in surface impoundments, landfills, mines, and waste piles. States are authorized to regulate coal ash under the RCRA non-hazardous waste provisions (Subtitle D), resulting in considerable interstate variation in waste management standards (TNCC 1995). The 1984 overhaul of RCRA strongly discouraged land disposal of hazardous wastes in response to concerns about groundwater contamination (Dower 1990). EPA was under a court-ordered deadline of February 1997 for issuing a final hazardous waste identification rule (HWIR, to identify wastes exempt from the management standards of RCRA Subtitle C). However the EPA Science Advisory Board judged the multi-pathway risk assessment model proposed by the agency for the HWIR to be inadequate (*Environment Reporter*, 3/15/96, pp. 2131-2132). In early 1997, EPA secured a one-month extension in order to renegotiate a new deadline for finalizing the HWIR (EPA's RCRA Hotline, 3/97).

[46] Unfunded mandates refers to federally required actions by state and local governments that are unaccompanied by transfer of resources required for implementation.

of the public drinking water supplies that violate the current arsenic drinking water standard of 50 ppb are well water systems serving fewer than 500 people.[47] Many of the public drinking water systems with high arsenic levels occur in western states with a tradition of resisting federal authority.[48] Also, the 1991 Lead/Copper drinking water rule was one of the most expensive drinking water rules ever adopted by EPA and one which did not consider the affordability for small systems (see accompanying case study). While the agency was considering whether to tighten the standard for arsenic, a former EPA drinking water official points out, "We were taking a lot of crap over the lead in drinking water standard."[49]

Legislative negotiations on amending the SDWA also began in earnest during the 103rd Congress, and ultimately, the 1996 SDWA Amendments established a State Revolving Fund for drinking water investments (analogous to the existing revolving fund for wastewater treatment and surface water) and overhauled the process for selecting drinking water contaminants for regulation as well as the criteria for standard setting.[50] However, during the legislative negotiations, controversies regarding four standards EPA had been working on--arsenic, radon, sulfate, and disinfection byproducts--were helping to frustrate efforts to form the necessary legislative coalition. As the former EPA official recalls, "Industry and the local groups wanted arsenic to go away, but the environmental groups and Waxman's people [Rep. Henry Waxman (D-CA), former chair of the House Health and the Environment Subcommittee] were pushing arsenic." For EPA, the case of arsenic in drinking water had particularly ominous parallels to the proposed drinking water standard for radon, which Congress had severely criticized, because drinking water is not considered to be the major source of exposure for most people in either case.[51] If EPA were to propose to dramatically lower the level of (inorganic) arsenic permitted in drinking water, it would appear to the non-specialist as being inconsistent with the higher

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[47] Between 1989 and 1991, 27 public drinking water supplies reported arsenic levels above the current standard of 50 ppb. All 27 supplies had groundwater sources and most of the supplies served fewer than 500 people (EPA/OW 1993).

[48] Arsenic in drinking water is not strictly a western issue. For example, parts of New England also experience higher than normal levels. According to a former EPA drinking water program official, the pressure exerted on EPA from the "Sagebrush Rebellion" movement was much higher in the case of radon than for arsenic.

[49] Shortly after its passage, the California Department of Health Services informed EPA that the state would not initiate the process for implementing the lead/copper rule due to a lack of resources (*Inside EPA*, 11/22/91, p. 1). During the 103rd Congress, Senators John Glenn (D-Ohio) and Dirk Kempthorne (R-Idaho) introduced S. 993, the "Community Regulatory Relief Act." According to a National Governors Association report (*Backgrounder*, July 17, 1994, p. 2), then-Office of Management and Budget (OMB) Director Leon Panetta and President Clinton endorsed the bill.

[50] The 1996 SDWA Amendments dropped the requirement that EPA issue drinking water standards for 25 new contaminants every three years. Instead, EPA is now required to develop a list of unregulated contaminants and make determinations of whether or not to regulate at least 5 of these contaminants every 5 years.

[51] It should be noted that the SDWA requires EPA to regulate radionuclides and arsenic under its standard setting provisions, but the agency has no authority to regulate radon in indoor air, and while EPA establishes permissible pesticide tolerances under the Federal Food, Drug and Cosmetic Act, the Food and Drug Administration regulates the safety of seafood, which can contain high levels of organic arsenic.

levels of (mostly organic) arsenic that the Food and Drug Administration permits in seafood that it is responsible for regulating. A direct comparison, however, is inaccurate due to the differential toxicities of organic and inorganic arsenic forms. Recalling the radon debacle and mindful that such nuances tend to get overlooked in political debates, EPA drinking water officials might wonder, "Do we want to go through the same thing on arsenic?"

Finally, recent debates over arsenic in drinking water occur in the broader context of efforts to instill greater biological sophistication and realism into EPA risk assessment methods. Because there is some evidence which suggests that human metabolism may have some capacity to detoxify the naturally occurring metalloid, arsenic represents to some a potentially high-impact test case for departure from the agency's linear, no-threshold cancer risk model that has been the target of much external criticism. To others, arsenic is an equally important test case in establishing the hurdle that human epidemiological studies must clear in the absence of supporting evidence from experimental toxicology to form the scientific basis for environmental regulation.

Table B-1 provides a summarized background of the 1995 decision to pursue additional research rather than revise the drinking water standard for arsenic. Since Assistant Administrator Perciasepe petitioned the court in 1995, the 1996 SDWA Amendments required EPA to develop a plan for additional research on cancer risks from arsenic, propose a standard for arsenic by January 2000, and promulgate a final standard by January 2001.

Table B-1. Background on the 1995 Arsenic in Drinking Water Decision.

1942	Public Health Service (PHS) sets a 50 ppb standard for arsenic in drinking water based on its acute poisonous effects.
1962	PHS recommends a 10 ppb standard; 50 ppb is grounds for rejection of the supply.
1968	Tseng *et al*. report association between arsenic in drinking water and skin cancer in Taiwan.
1975	EPA sets "interim" standard for arsenic in drinking water of 50 ppb.
1977	National Research Council (NRC) *Drinking Water and Health*, Vol. 1, suggests the 50 ppb standard may not provide an adequate margin of safety.
1978	EPA initiates review of arsenical pesticides.
1980	NRC *Drinking Water and Health*, Vol. 3, recommends further investigation of possible beneficial nutritional effects of arsenic at low doses. Int'l Agency for Rsrch on Cancer (IARC) concludes that there is sufficient evidence that inorganic arsenic in drinking water is a skin carcinogen in humans based on the Taiwan study. EPA Office of Water prepares draft arsenic water quality criteria document.
1981	Southwick *et al*. report Utah epidemiological study finding no cancer in a group of 145 people consuming drinking water containing arsenic levels of approximately 200 ppb.
1983	NRC *Drinking Water and Health*, Vol. 5 concludes that US epidemiological studies fail to confirm the Taiwanese results and states, "It is therefore the opinion of this committee that 0.05 mg/liter [50 ppb] provides a sufficient margin of safety..." In the absence of new data, arsenic should be presumed an "essential" nutrient for humans based on mammalian animal studies.

Table B-1. Background on the 1995 Arsenic in Drinking Water Decision (cont'd).

1984	EPA/ORD reports assessment of inorganic arsenic. 50 ppb drinking water standard results in an upper-bound skin cancer risk estimate of 2 percent, based on Taiwanese database.
1985	EPA/ODW relies on evidence of essentiality of arsenic to human nutrition in a proposed rulemaking (50 *Fed. Reg.* 46959). Chen *et al*. report association between arsenic and internal cancers in Taiwan.
1986	SDWA Amendments require EPA to review arsenic in drinking water standard by 1989. SARA includes ARARs provisions, broadening implications of arsenic drinking water standard. NRC *Drinking Water and Health*, Vol. 6. states EPA should consider metabolism and pharmacokinetics in assessing the risks of drinking water carcinogens. Peer review workshop of Draft EPA/RAF *Special Report on Ingested Inorganic Arsenic*.
1987	NRC *Drinking Water and Health*, Vol. 8: Pharmacokinetics in Risk Assessment.
1988	June 21. EPA Adm. Lee Thomas memo permits managers to down-weight ingested inorganic arsenic risks by an "uncertainty" factor of 10 because skin cancer is generally non-lethal. July. EPA/RAF *Special Report*. 50 ppb drinking water standard results in a skin cancer risk estimate of 0.25 percent, based on Taiwanese database. Down-weighting yields skin cancer "risk" on the order of 10^{-4}.
1989	September. Bull Run Coalition of Oregon sues EPA for failing to meet 1986 SDWA deadlines. September 28. EPA/SAB/DWC recommends the agency revise its arsenic risk assessment to consider "the possible detoxification mechanism that may substantially reduce cancer risk from the levels EPA has calculated using a linear quadratic model fit to the Tseng [Taiwan study] data." Recommends arsenic's nutritional essentiality should not be an influential factor.
1990	EPA agrees to propose arsenic rule by November 1995 and finalize the rule by November 1997. Various *ad hoc* groups inside and outside EPA begin to formulate arsenic research agendas.
1992	Smith *et al*. estimates 50 ppb standard represents US internal cancer risks on the order of 10^{-2}. EPA sets a reference dose (RfD) for arsenic non-cancer risks using a range of values.
1993	EPA begins SDWA negotiations with Democratic 103rd Congress. Brown raises problems with Taiwanese epidemiological study dose-reconstruction. Hopenhayan *et al*. finds no consistent evidence for arsenic threshold hypothesis in humans. EPA/SAB/DWC finds an association between internal cancer and exposure to high levels of arsenic in drinking water but suggests evidence of non-linear arsenic pharmacokinetics.
1994	November. Republicans gain majority in 104th Congress. December. Decisional briefing for AA/OW Perciasepe at EPA Headquarters.
1995	January. Perciasepe petitions USDC Oregon for more time to conduct research on

2. Scientific Issues

Responses among interviewees were evenly split (6 yeas-6 nays) as to whether there was adequate scientific information available in 1995 to inform a decision to revise the MCL for arsenic in drinking water.[52] According to an independent risk analyst, "virtually everyone agrees that ingested [inorganic] arsenic causes human skin cancer, and there is persuasive evidence that it causes cancer in the bladder and other internal organs as well. The debate is on the shape of the dose-response curve" in the low dose region. The split-opinion arises from a disagreement about what EPA can or should do in the face of this uncertainty.

Interviewees who believe that the available evidence is at least adequate maintain that the information used to quantitatively estimate skin cancer risks from arsenic in drinking water is "as strong or stronger" than the scientific basis for most EPA regulatory decisions. An EPA official summarizes this argument, "We have human data and exposures in the range of general population exposure. We're not extrapolating from high doses based on limited animal studies."[53] Some of these respondents also point to the evidence of non-cancer health effects of chronic arsenic exposure (such as vascular or neurological damage in the extremities, non-cancerous skin lesions, and potential reproductive effects) that were not quantitatively estimated.

Interviewees who believe that the available evidence provides an inadequate basis for modifying the arsenic drinking water standard claim that the strength of the human data from epidemiological studies has been mischaracterized and point to the evidence of the human body's capacity to methylate, and subsequently eliminate through excretion, some ingested inorganic arsenic. Methylation has been suggested as a detoxification pathway for arsenic that becomes increasingly inefficient or perhaps saturated with increasing exposure. While these respondents would agree that EPA often makes regulatory decisions on the basis of weaker scientific information, they believe that the available information is inadequate in the context of the arsenic drinking water standard.

The principal scientific issues include: 1) interpretation of epidemiological evidence of skin cancer; 2) the epidemiological evidence and assessment of internal cancers; 3) the possibility of an arsenic detoxification pathway in humans; 4) arsenic in drinking water exposure assumptions; 5) the lack of understanding of arsenic's mechanism of toxicity; and 6) interpretation of the evidence of arsenic's nutritional essentiality. There was general consensus among respondents that the greatest source of scientific uncertainty was the correct form of the dose-response curve (i.e., linear or nonlinear, no-threshold or threshold) for estimating the risk of cancer from low levels of arsenic in drinking water. However, only one respondent, an EPA water program scientist, believes that there is

[52] The 12 respondents in no way represent a statistically valid sample.

[53] Although the arsenic exposures observed in some epidemiological studies include levels considerably higher than the current U.S. drinking water standard of 50 ppb, standard procedures for chronic animal studies include the maximum tolerated dose, which would be much higher, relatively speaking, than the doses observed in human populations.

currently sufficient evidence to justify departing from the agency's default procedure of employing the no-threshold linear model. Another EPA scientist stated that arsenic is "the best case I know of where you might challenge the default. Whether it's good enough, I don't know."[54]

Epidemiological Studies of Skin Cancer

EPA relies on the results of animal studies to assess the toxicity of most regulated carcinogens. For assessing skin cancer risks of ingested arsenic, however, scientists have "lousy animal models," according to an independent toxicologist. "You wouldn't expect skin cancer in a rat because it's histologically different from humans" (i.e., the anatomy of its tissues differ). Therefore, scientists have focused on human epidemiological studies to assess arsenic skin cancer risks.

One such study (Tseng *et al*. 1968) conducted in rural Southwest Taiwan where Blackfoot Disease[55] is endemic formed the cornerstone of the 1984 Office of Research and Development Health Assessment Document (HAD) for arsenic (EPA/ORD 1984) and the 1988 Risk Assessment Forum (EPA/RAF 1988) arsenic report. The Taiwan database has considerable strengths. First, it is extraordinarily large.[56] This is important because epidemiological studies must be very large to detect even substantial cancer rates.[57] Second, given equal sample sizes, an epidemiological skin cancer study of an Asian population is more powerful (i.e., it can detect smaller effects) than one of a Caucasian population because sun-induced skin cancer is relatively uncommon in Asian populations.[58] Third, the types of skin cancers (mainly on the extremities, rather than on sun-exposed surfaces) observed in the Taiwan study population are believed to be diagnostic of arsenic exposure. Fourth, the well water tested in the study area displayed a broad range of arsenic concentrations (<10 - 1820 ppb), permitting coverage of the relevant concentrations of concern in the US (<50 ppb) as well as higher levels that would be expected to result in higher-than-background levels of skin cancer. Fifth, the entire study group was physically examined and pathological studies confirmed over 70% of the observed skin cancer cases (EPA/RAF 1988). Therefore, there is high confidence that the health effects have been accurately measured. (Most epidemiological studies rely on available or easily collected information such as medical records, surveys, or public health registries to estimate the incidence of health effects. Such data are generally inaccurate and often suffer from the bias of underreporting.)

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[54]Recently, an EPA risk assessment of certain pesticide-caused thyroid tumors in rodents incorporated a departure from linearity. According to an ORD scientist, however, arsenic is generally viewed with greater interest and has greater likelihood of impacting agency procedures because the stakes are higher.

[55] Blackfoot Disease is gangrene of the extremities caused by damage to the peripheral vasculature.

[56] The study group contained 40,421 individuals and the control group was 7,500.

[57] There were 428 cases of skin cancer in the study group and no cases in the control group. Based on the skin cancer rate for Singapore Chinese from 1968-77, the expected skin cancer rate in the control population of 7,500 was 3.

[58] According to an EPA official, in Caucasian populations, any skin cancer effect of arsenic is going to be dwarfed by sun exposure, whereas in an Asian population, arsenic-induced skin cancer would "stick out like a sore thumb."

Like all environmental studies, however, the Taiwan study has weaknesses. Dietary and food preparation sources and activity patterns that may have contributed to total arsenic exposure were not assessed, and the reported health effects of arsenic may also be confounded with those of a number of additional substances (e.g., humic acids) found in the well water that were not taken into account.[59] Some have argued that other aspects of the Taiwanese study, while not deficiencies of the study *per se*, limit the relevance of the study to US populations or its utility for estimating risks from low-level exposures. For example, the diet of the Taiwan study population was lower in protein and some amino acids than the typical US diet, and these nutrients are required for biomethylation. The implications of this observation for the US arsenic drinking water standard, however, remain unclear.[60]

The main limitation of the Taiwanese dataset for regulatory purposes, however, is the "dose-reconstruction," according to an independent toxicologist. In the course of research conducted *after* the 1988 EPA arsenic reassessment was reported, independent statistical consultant Ken Brown discovered previously undetected problems with the Taiwan arsenic in drinking water exposure database.[61] While investigating the association between arsenic in drinking water and *internal* cancers in the Taiwan study population with Taiwanese investigator C.J. Chen using a different exposure database than had been used by Tseng and colleagues (see Brown and Chen 1993), Brown discovered that within Taiwan villages the wells often varied in arsenic concentration by a wide range.[62] Consequently, it was recognized that it was impossible to precisely estimate the levels of arsenic in drinking water to which individuals in Tseng's study population were chronically exposed prior to medical examination.[63] Measurement errors in the "dose

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[59] Mushak and Crocetti (1995), however, dismiss the role of humic acids and suggest that arsenic present in crops may have been primarily in less toxic organic forms. Paul Mushak of PB Associates, which specializes in toxicology and health risk assessment of metals, is a former faculty member of the University of North Carolina, was a principal external author for the 1984 ORD Health Assessment Document for arsenic, contributed an issues document on carcinogenicity and essentiality to the 1988 RAF assessment, and has served as a member of and consultant to the EPA Science Advisory Board and Clean Air Act Science Advisory Committee. Slayton *et al*. (1996) respond to Mushak and Crocetti (1995) by citing some evidence that inorganic arsenic accounts for a considerable portion of total arsenic in some foods. See Mushak and Crocetti (1996) for their rebuttal.

[60] Mushak and Crocetti (1995) estimate that the study group's nutritional status relevant to biomethylation is more than adequate. See Slayton *et al*. (1996) and Mushak and Crocetti (1996) for more on this debate.

[61] Brown's work was supported by American Water Works Association (*Risk Policy Report*, 9/16/94, p. 11). AWWA is the trade association of public drinking water suppliers. Brown also co-authored a paper with EPA water program scientist Charles Abernathy suggesting that food may be a greater source of arsenic ingestion in Taiwan than in the US, indicating that previous assessments may have overestimated risks in the US (see discussion of exposure assumptions below).

[62] C.J. Chen is a Taiwanese researcher trained at Johns Hopkins University.

[63] Tseng *et al*. roughly estimated the level of arsenic in drinking water at the village level (i.e., all individuals in a village were assumed to drink water with the same arsenic concentration), and villages were assigned to one of three broad dose levels. The problem lies in then-unrecognized variable arsenic concentrations between the wells within a village, largely due to a mix of shallow wells (with low arsenic levels) and deep artesian wells (with high arsenic levels), and in having only one well test for 24 (40%) of

reconstruction" would undeniably produce inaccurate skin cancer risk estimates; the rub is whether any resultant bias is severe enough to warrant discounting the best available human data.[64] The lack of precision in dose or exposure estimation is generally more serious for epidemiological studies than for toxicological studies.[65] There is a tradeoff, however, between experimental control and relevance to real-world human health.

Other epidemiological studies in Germany, Mexico, Argentina, and Chile (reviewed in NRC 1983 and EPA/RAF 1988) also suggest an association between ingested arsenic and a variety of skin diseases, including skin cancer, but these studies were weaker than the Taiwanese study. EPA/RAF (1988) compared the predictions from the quantitative analysis of the Taiwan study to the observations in Mexico and Germany and concluded that the results were consistent. Three epidemiological studies conducted in the US (in Alaska, Oregon, and Utah, reviewed in NRC 1983) failed to detect any positive relationship between arsenic in drinking water and disease. Southwick, *et al*. (1981), arguably the best domestic study, did not find any statistical differences in cancer incidence or death rates Millard County, Utah. However, the study's small sample size and the frequent occurrence of skin cancer in the US limit the study's power to detect differences that could be considered substantial.[66] The principal strength of the epidemiological studies in developed countries is that medical records are better than those in developing countries.

Internal Cancers

EPA/RAF (1988) quantitatively analyzed the risks from arsenic in drinking water in terms of skin cancer, which rarely metastasizes (i.e., spreads to other organs) and is rarely fatal in the US. However, Chen *et al*. (1985, 1986, 1988) provided evidence from Taiwan of a relationship between arsenic in drinking water and internal cancers (bladder, kidney, colon, liver, and lung). EPA/RAF (1988) concluded that the summary data in Chen's published reports were insufficient to quantitatively assess dose-response for internal cancers. Applying a linear model to the raw Taiwanese data, Smith *et al*. (1992)

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the 60 villages. An EPA scientist suggests that "the epidemiological exposure measures are so poor at lower levels that you can't distinguish between 5-100 µg/l [ppb]."

[64] It is not self-evident that the dose-reconstruction problems discovered by Brown revealed an overestimation of the cancer risk. Measurement error in dose reconstruction does serve to bias the risk estimates, and Brown *et al*. (1997a) suggest a plausible scenario under which prior risk estimates for the low exposure group would be overstated. However, neither the magnitude nor even the direction of the *true* bias may be estimable because reliability measures of the proxy measure for true exposure are absent. (If good reliability measures were present, reanalysis of the Taiwan data could have laid the issue to rest. Instead, new epidemiological studies are being planned.) Therefore, the dose-reconstruction problems cast doubt on the reliability of the Taiwan study for risk estimation in the low dose region but do not necessarily indicate that risks derived from the study are overestimated. If there is a statistical bias in either direction, its magnitude could be either negligible or non-negligible.

[65] It should be noted that even animal experiments present problems controlling administered dose levels. For example, because rodents eat their feces, they may be "redosed" with excreted substances.

[66] The Utah study compared 145 people in a community consuming drinking water with 200 ppb arsenic and 105 in another community whose drinking water levels of arsenic averaged 20 ppb. The study was done under a research grant from EPA/ORD's Health Effects Research Lab (HERL).

estimated the risk of internal cancers in the US due to consuming drinking water containing 50 ppb arsenic to be on the order of 10^{-2} (one-in-one hundred), an order of magnitude *higher* than EPA/RAF's 1988 estimated skin cancer risk.[67]

In reviewing EPA's 1993 Draft Drinking Water Criteria Document on Inorganic Arsenic, the EPA Science Advisory Board's Drinking Water Committee[68] agreed that the Taiwanese data demonstrate an association between internal cancer and exposure to high levels of arsenic in drinking water. However, the panel pointed to evidence of non-linearities in the pharmacokinetics of arsenic and to dietary sources of arsenic exposure in Taiwan (discussed below) that represent uncertainties in directly extrapolating the results to low-level drinking water exposures in the US (EPA/SAB/DWC 1993).[69]

Detoxification

There is both human and animal evidence suggesting that methylation constitutes a detoxification pathway for ingested inorganic arsenic.[70] There is also some evidence suggesting that methylation becomes increasingly inefficient or perhaps even saturated with increasing doses of ingested arsenic. If there is a discrete point at which methylation becomes saturated, then a threshold cancer model may be appropriate. If, as seems more likely, methylation becomes increasingly inefficient with increased exposure, then a nonlinear (sub-linear) cancer dose-response model may be appropriate. Both the nonlinear

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[67] Prof. Allan Smith is an epidemiologist with the School of Public Health, University of California, Berkeley whose work was supported by the National Institute of Environmental Health Sciences Superfund Basic Research Program (www.niehs.nih.gov/sbrp/newweb/resprog). This underscores the linkage of the drinking water standard to contaminated site programs. Carlson-Lynch *et al*. (1994) criticized Smith and colleagues for not using a curvilinear statistical model to reflect the hypothesized detoxification of arsenic in well-nourished humans below ingestion exposures of 200-250 μg/day and for assuming Taiwanese water consumption rates below those used by EPA in deriving the agency's Reference Dose (RfD) for ingested arsenic. Both the nonlinear model and the higher drinking water consumption figures would yield lower risk estimates. Smith *et al*. (1995) responded that they employed the Taiwanese drinking water intakes used in EPA/RAF (1988). Brown *et al*. (1997b) find that the Taiwan internal cancer data are statistically consistent with either a linear or a non-linear dose-response model (a "hockey stick" shaped curve formed by two linear segments with different slopes). (See discussion below regarding detoxification, exposure assumptions, and setting the RfD.) Lynch and colleagues are with the environmental consulting firms ChemRisk and Gradient Corporation. Lynch co-author Barbara Beck represented EPA Region I on the 1986-87 EPA Risk Assessment Forum and is currently with Gradient, which provides environmental consulting services to the Atlantic Richfield Company. ARCO owns the Anaconda Minerals Superfund Site in Montana (a former mining site where arsenic is a key contaminant) and has played a key role in supporting research and analysis on the risk of ingested arsenic.

[68] The current SAB Drinking Water Committee was formerly a Subcommittee of the SAB Environmental Health Committee.

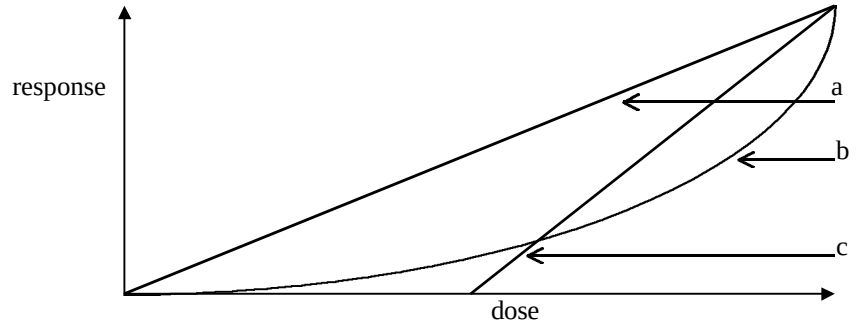
[69] As discussed below, Smith suggests that ongoing epidemiological studies in Argentina and Chile serve to strengthen the evidence of an association between arsenic ingestion and internal cancers (Allan Smith, Professor of Public Health, University of California, Berkeley, personal communication).

[70] Methyl is the organic molecule CH_3. Methylated arsenic is excreted in urine more rapidly than inorganic forms of arsenic, and as more methyl groups are added to arsenic during metabolism, arsenical compounds become less toxic (EPA/RAF 1988). A number of short-term animal experiments (reviewed in EPA/OW 1992) indicate 70-95% urinary excretion of soluble forms of arsenic.

and the threshold models (or a hybrid of the two resulting in a curve with an inflection point) would indicate lower risks in the low dose region than would be estimated by a no-threshold, linear model (see Figure B-1).

"In the absence of adequate information to the contrary," EPA's 1986 Guidelines for Carcinogen Risk Assessment called for using a linear statistical model to estimate risks in the low-dose region by extrapolation from the available data on higher-doses (EPA 1986). NRC (1986, 1987) recommended that EPA generally consider the absorption, metabolism, distribution, and elimination of toxic compounds in the body (i.e., pharmacokinetics or toxicokinetics) when assessing drinking water carcinogens.[71]

Figure B-1. Comparison of no-threshold, linear (a); sub-linear (b); and threshold (c) dose-response models.



In 1989, the EPA Science Advisory Board concluded "that at dose levels below 200 to 250 μg As^{3+}[trivalent arsenic]/person/day, there is a possible detoxification mechanism [methylation] that may substantially reduce cancer risk from the levels EPA has calculated using [a] linear quadratic model fit to the Tseng [Taiwan skin cancer study] data" (EPA/SAB 1989).[72] Some participants in the arsenic debate (e.g., Carlson-Lynch

[71] The amount of a substance consumed in drinking water represents an "administered" dose which may differ from the "internal" dose that is absorbed and available for biological interaction because ingested substances may be excreted from the body or metabolized into different forms that are more or less toxic than the original substance.

[72] Trivalent arsenic is more acutely toxic than pentavalent arsenic (EPA/RAF 1988). There has been some confusion in the literature regarding the statistical analysis used by EPA/RAF (1988). Various sources have inaccurately stated that EPA employed the "Linearized Multistage" (LMS) model. Under the 1986 guidelines, this is the default model EPA applies to the results of animal carcinogen studies. When EPA reports point estimates for cancer risks from animal studies using the LMS, it uses an upper-limit estimate (i.e., the 95th percent upper confidence limit) of the dose-response slope. However, because the Taiwan data were from an epidemiological study, the model used by EPA/RAF (1988) differs in some important respects from the standard animal study assessment practices. The "linear quadratic model" differs from the simple linear regression model in that it includes both linear and quadratic (squared) terms (i.e., $y = b_0 + b_1x + b_2x^2$). This model provided a better statistical fit to the Taiwanese data than if only a linear term were used. Consistent with the 1986 Guidelines, however, the model is linear in the low-dose region. The model also differs from the simplest model (which considers only dose levels) by incorporating duration of exposure information. EPA/RAF (1988) also based its skin cancer risk estimate on the predicted slope of the curve in the low-dose region (i.e., the maximum likelihood estimate or

1994; Beck *et al*. 1995) have argued that the hypothesized saturable detoxification pathway suggests that EPA should employ a non-linear shaped dose-response model. However, as indicated above, a majority of respondents believe that the current scientific findings are insufficient to override the default. A brief discussion of the most frequently cited evidence follows.[73]

Valentine *et al*. (1979) observed that blood arsenic was only elevated when drinking water arsenic was above 100 μg/L. Mushak and Crocetti (1995) argue, however, that the "threshold" observed by Valentine *et al*. may simply represent the detection limit of the analytical tests available at the time.[74] Buchet *et al*. (1981) monitored the urinary excretion of arsenic in four volunteers exposed to arsenite at dose levels of 125, 250, 500, and 1,000 μg/day for 5 days and found that the arsenic methylating capacity was hampered in the two high-dose subjects. If the results are generalizable, this would suggest that total urinary excretion of arsenic may be compromised at high doses leading to increased deposition in body tissues. However, even at the daily dose of 1,000 μg/day, the arsenic methylation capacity was not completely saturated. Consequently, Smith *et al*. (1995) conclude that "the evidence of *any* metabolic saturation from this study is not conclusive." Furthermore, there may be important differences among individuals in methylation efficiency that prevent generalization from four individuals to the general population. Failure to consider parameter uncertainties and inter-individual variability in pharmacokinetic modeling can lead to misleading results, or at least, overconfidence in the results obtained.[75]

Work by How-Ran Guo and colleagues (e.g., Guo 1993) has also been cited as evidence of a possible nonlinear dose-response relationship between arsenic exposure and urinary cancer.[76] The study is based on data collected from 243 townships in Taiwan

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MLE). The MLE is less than the upper limit slope estimate (i.e., the MLE is a less conservative risk estimate).

[73] See EPA/RAF (1988, Appendix E) and EPA/OW (1992) for a more complete discussion of the metabolism of inorganic arsenic.

[74] Mushak and Crocetti (1995) also state that blood arsenic is a poor indicator of chronic or prior exposures, with urine being a more stable indicator of chronic exposure and hair being the best measure of cumulative exposure. An EPA official explains that standard blood analyses make no distinction between forms of arsenic (organic or inorganic). "You can spike your arsenic blood with seafood" [containing organic arsenic]. Beck *et al*. (1995), however, argues that the ratio of methylated urinary metabolites of arsenic could be a more sensitive indicator of methylation saturation than percent inorganic arsenic in urine. See Slayton *et al*. (1996) and Mushak and Crocetti (1996) for further debate over the evidence for nonlinearities in the arsenic dose-response curve.

[75] For example, in 1991, EPA reduced its risk estimate for methylene chloride by an order of magnitude on the basis of research on the pathways through which the substance is metabolized. After the agency's reevaluation, however, research began to focus attention on parameter uncertainties in the pharmacokinetic modeling which had not been considered during the reassessment. At least one analysis suggested that according to the new information, EPA should have raised, rather than lowered, its original risk estimate (NRC 1994).

[76] Guo has conducted work on arsenic as a post-doctoral researcher at the University of Cincinnati and at the Washington, DC-based firm RegNet Environmental Services. Guo's study of arsenic exposure and

(including a population of 11 million). As described by Beck *et al*. (1995), a strength of the Guo study is that it considers the distribution of arsenic concentrations in wells within a township, rather than simply applying the median arsenic concentrations for all wells in a village to represent exposure to each village inhabitant, as was done by Tseng *et al*. (1968). Guo's work reportedly has two principal shortcomings. First, the study used records of individual family ownership of wells to determine which families drank from which wells (*Risk Policy Report*, 9/16/95, p. 11). According to an ORD scientist, well ownership is a crude surrogate for arsenic exposure.[77] Second, the study assessed health outcomes using death certificates and a Taiwan cancer registry begun in 1979 (*Risk Policy Report*, 9/16/95, p. 11). The ORD scientist points out that death certificates do not permit an assessment of non-fatal cancers and suggests that the cancer registry is incomplete.[78]

According to an industry scientist, "There's not sufficient evidence available to depart from the low-dose linearity default. The disagreement is over whether it's worthwhile to try to answer that question."

While there is some evidence of methylation efficiency decreasing with increasing doses of ingested inorganic arsenic, Hopenhayn-Rich *et al*. (1993) conclude that current human studies do not support the methylation *threshold* hypothesis. Smith *et al*. (1995) elaborate that "if a methylation threshold for arsenic does exist, the epidemiological and experimental evidence suggest that it must be at exposure levels well above 2000 μg/day, making it completely irrelevant to usual human exposures." A former SAB member finds the current evidence of arsenic detoxification to be "highly suggestive of the value of further information, but not conclusive with respect to the regulatory decision." An industry scientist concurs, "There's not sufficient evidence available to depart from the low-dose linearity default. The disagreement is over whether it's worthwhile to try to answer that question." While there is considerable agreement among respondents that the existing evidence of arsenic detoxification does not meet the criterion of "adequate information to the contrary" required by 1986 Guidelines to depart from standard operating procedures, it remains unclear what constitutes the necessary level of information.

Considering the evidence of a gradual decline in arsenic methylation efficiency with increasing dose levels, the scientific disagreement regarding arsenic detoxification may not be so much about the plausibility of a non-linear dose-response curve for ingested arsenic as it is about what course of action to take in the absence of knowledge of the precise form of a hypothesized non-linearity.[79] Although the current discussion in the larger scientific community tends to focus on the degree, if any, of non-linearity in the dose-

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urinary cancers was done in his capacity as a researcher for RegNet and was supported by ARCO and the International Lead Zinc Research Organization (ILZRO) (*Risk Policy Report*, 9/16/94, p. 11).

[77] Exposure may be poorly related to well ownership because owners of multiple wells may supply water to others who do not own wells.

[78] Because the cancer registry only requires hospitals with 50 beds or more to report, cancer cases may not be reported from smaller hospitals and pathology centers.

[79] See, however, the discussion below about a possible tradeoff between protection against acute arsenic toxicity and chronic cancer.

response relationship for arsenic, some of the combatants on both sides of the issue have caricatured the scientific debate as "threshold v. no-threshold," frequently using the terms "non-linear" and "threshold" interchangeably. As illustrated in Fig B-1, however, a sub-linear dose-response curve need not indicate a threshold dose prior to toxicity.

For scientists who appreciate the distinction, the use of the term "threshold" may be a form of short-hand for "so non-linear as to present a 'virtual' threshold, for all practical purposes." As opposed to the notion of a "virtual" threshold, however, the unqualified threshold hypothesis permits one to defend the current drinking water standard (or even higher levels) as presenting zero risk and needing no revision whatsoever. The uncompromising position of the "default camp" is summed up in the challenge presented by an ORD scientist, "If it is non-linear, where is the threshold, where is the non-linearity?" The poor signal-to-noise ratio in the low dose region, however, may preclude defining a non-linear curve form with absolute precision at *de minimis* risk levels.[80]

Exposure Assumptions

Risk estimates are often sensitive to changes in exposure scenarios. EPA generally assumes a drinking water consumption rate of 2 liters per day (l/day) for US adults (EPA 1992), and ORD did so in its 1984 arsenic Health Assessment Document (EPA/ORD 1984). Reasoning that people performing heavy labor outdoors in a sub-tropical climate would consume more water, the EPA inter-office Risk Assessment Forum assumed Taiwanese water consumption rates of 3.5 l/day for men and 2 l/day for women (EPA/RAF 1988). Later, in deriving the agency's Reference Dose (RfD) for arsenic, an inter-office Integrated Risk Information System (IRIS) work group assumed Taiwanese drinking water consumption rates were 4.5 l/day (for men and women) (Abernathy *et al*. 1989).[81] Adopting a higher drinking water consumption rate for the Taiwanese than that which is applied to the US population lowers the estimated risk from arsenic ingestion in the US. However, there have been no specific measurements of water intake by persons in the Taiwan study area. According to an EPA official, "there was some limited anecdotal evidence that these individuals would consume more than the average for the US. Staff members with experience thought this was reasonable."[82] Indicating the sensitivity of the risk estimate to the assumed drinking water intake, Beck *et al*. (1995)

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[80] For example, NRC (1993) concluded that "even if an agent's mechanisms of [carcinogenic] action are well understood, it will still be very difficult to determine its dose-response relationship accurately enough to predict doses that correspond to [cancer] risk as low as one in a million" (p. 10). NRC (1993, pp. 206-210) also discusses the considerable interindividual variability in factors such as detoxification that affect susceptibility to toxic substances. Such variability contributes to the inherent imprecision of risk estimates.

[81] This figure assumed direct water consumption of 3.5 l/day and indirect water consumption of 1 l/day for cooking rice.

[82] According to an EPA official, the water consumption rates assumed by the agency were based primarily on a discussion between Herman Gibb and some farmers in a Taiwanese medical center. The farmers showed Gibb water bottles that they carried into the field and said that they would generally drink one bottle in the morning and another in the afternoon. Gibb measured the volume of the bottles and agency analysts made some additional assumptions about morning and evening drinking water consumption.

calculate that by using a Taiwanese water consumption rate of 4.5 l/day, as opposed to 3.5 and 2 l/day for males and females, respectively, EPA's current US skin cancer risk estimate would be decreased by approximately 50%.[83]

Similarly, if food contributes much more to inorganic arsenic exposure in Taiwan than it does in the US, the estimated risk from arsenic in US drinking water would be significantly reduced (Brown and Abernathy 1995). Using estimates of Taiwanese inorganic arsenic dietary intake, Yost *et al*. (1994) surmised that EPA's arsenic cancer risk based would be reduced by up to an order of magnitude. An ORD scientist, however, dismisses this evidence as flimsy. "We still don't have any data on food. ARCO paid to have" a very small number of rice and yam samples taken from Taiwan and analyzed.[84] As a result of the small sample size, the uncertainty about the inorganic arsenic contribution from food in Taiwan remains large.[85]

Mechanism of Toxicity

Many observers argue for a better understanding of arsenic's biological mechanism of toxicity in humans as a means of definitively reducing uncertainty in risk assessment. The lack of good animal or *in vitro* models for mechanistic research, however, presents a considerable obstacle.[86] According to an industry scientist, "Mechanistic information is the missing information, but arsenic is a mystery compound. It's not something that rips up DNA or is an ass-kicking clastogen."[87] Mass (1992) suggests that methylation of arsenic may itself present a biological dilemma, protecting against acute, short-term effects, but possibly contributing to cancer over the long-term.[88] As a result, according to an independent toxicologist, "research establishing the biomethylation pathway, while interesting, would not nail down whether low-dose non-linearity occurs." An EPA official estimates that pursuing a mechanistic research agenda would take 15-20 years. If the costs of delay are substantial (i.e., if the high risk estimates for arsenic in drinking water

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[83] As an EPA research official observed, this degree of sensitivity of risk estimates to changes in exposure assumptions is not surprising. As Mushak and Crocetti (1996) note, however, any individual component of uncertainty in risk estimates also should be considered in the context of other potential sources of variability and uncertainty. See Mushak and Crocetti (1995), Slayton *et al*. (1996), and Mushak and Crocetti (1996) for further debate regarding assumed drinking water consumption rates for chronically heat-and-humidity-stressed, active rural populations of Taiwan.

[84] Mushak and Crocetti (1996) estimate that a combined half-dozen food samples were taken. The statistical confidence limits around such a small sample would tend to be large.

[85] Mushak and Crocetti (1995) argue that because the methods used by Yost *et al*. (1994) to analyze the Taiwanese food samples involved strong acid treatment to produce satisfactory recoveries of total arsenic, the measured levels may have been generated from organic forms present in the food as an artifact of the laboratory analytical methods. See Slayton *et al*. (1996) and Mushak and Crocetti (1996) for further details on the debate.

[86] *In vitro* ("in glass") refers to a variety of laboratory-based procedures not involving whole animal testing.

[87] A clastogen is a chemical that is able to cause structural damages in chromosomes, primarily breaks. Chromosomal and DNA damage are two possible cancer mechanisms. Rudel *et al*. (1996) suggest that arsenic's carcinogenic mechanism may result in a non-linear dose-response relationship.

[88] Marc Mass is with EPA's Health Effects Research Laboratory.

are accurate), this would represent a disadvantage to waiting for definitive mechanistic results.

The time required to perform the necessary research depends, however, on the weight of evidence deemed by the *decisionmaker* as sufficient. Although mechanistic research tends to be fundamental and long-term in nature, it is conceivable that a policymaker might accept as sufficient mechanistic evidence that would require less than 15-20 years to produce. An independent risk analyst, for example, believes it may be possible that mechanistic research resulting in a test of the linear dose-response hypothesis for arsenic, something short of a complete mechanistic understanding, could be conducted within a few years. Of course, as indicated above, questions would remain about what precisely to do about the arsenic drinking water standard if the linearity hypothesis were rejected.

Nutritional Essentiality

Numerous studies have indicated that arsenic is an essential nutrient for rats, hamsters, goats, and other animals. Extrapolating from these animal studies, a possible human arsenic nutritional requirement would be 12 μg/day (Uthus 1992). According to an independent toxicologist, however, "as often happens with some new elements that putatively show this [nutritional essentiality] in lab experiments, it's often hard to show this in humans." If arsenic is, in fact, both essential for human nutrition and a human carcinogen, there may not be much separation, if any, between essential and cancer-causing doses. This source points out that this overlap is not unusual for micro-nutrients. What is apparently unusual in the case of arsenic, is that the same chemical form that is carcinogenic may also be essential.[89] EPA/SAB (1989) concluded that attributing a prominent role to the essentiality of arsenic in human nutrition in evaluating health risks is unfounded as a consequence of the lack of convincing evidence in humans.

3. The Process within EPA

Setting the Agenda

The 1989 suit filed by the Bull Run Coalition against EPA for failing to comply with deadlines under the 1986 SDWA amendments was the event most frequently (5) mentioned by respondents as being responsible for forcing arsenic in drinking water on the agency's regulatory agenda. However, activity within the agency dates back to the review of arsenical pesticides begun in 1978. Over the 20 years since EPA set its "interim" drinking water standard, a number of factors elevated arsenic's position on the drinking water regulatory agenda. The 1984 and 1988 skin cancer risk estimates focused attention on arsenic but caused the drinking water office, according to a former staff scientist to question, "is it really that bad?" An independent toxicologist noted that the reported findings of internal cancers (Chen *et al*. 1985) "notched up the issue." The subsequent US

[89] The harmful and beneficial chemical forms generally differ. For example, hexavalent chromium is carcinogenic, but trivalent chromium is essential (Mushak 1994).

risk estimate for internal cancers (Smith *et al*. 1992) caused an even greater sense of urgency that penetrated the drinking water program. According to a former EPA official, the conclusion by the EPA Risk Assessment Forum in 1988 that Chen's 1985 report on internal cancers was insufficient for quantitative risk assessment "became the basis of the agency twiddling its thumbs until the Smith report came out in 1992."

The Health Assessment Process

The process of assessing the health risks of arsenic in drinking water pitted risk assessors in the EPA Office of Water (OW), who appear to support the current standard or something close to it as reasonable, against risk assessors in the Office of Research and Development (ORD) headquarters, who estimate that the arsenic standard does not provide the same level of health protection provided by many other EPA regulations. The ORD risk assessors, among others within EPA, appear concerned by the prospects of case-by-case departures from the agency's default risk assessment procedures, which represent the implementation of EPA science policy statements, and from the standards of scientific proof that the agency customarily requires to take regulatory action.[90]

With limited scientific resources at their command, the EPA water program analysts have enlisted the aid of the agency's science advisors and leveraged the resources of the regulated community to challenge ORD's arsenic health assessment. The OW staff also found an ally within ORD in the Health Effects Research Lab (HERL) in Research Triangle Park, NC. According to a former EPA official, there were some staff within the EPA Office of Ground Water and Drinking Water who advocated making the arsenic standard more stringent. However, ORD's principal internal ally in the process appears to be the Office of Pesticides, Pollution Prevention, and Toxic Substances (OPPTS). ORD scientists have staked the legitimacy of their analysis to the inter-office Risk Assessment Forum (RAF) "consensus-building" process which approved the 1988 assessment. Three-quarters of the responses (9) rated EPA's treatment of the available scientific information as good-to-very good (independent of the quality of the underlying data). However, some respondents distinguished between the water program's analysis (EPA 1992) and the ORD analysis endorsed by the RAF. (One respondent rated the water analysis as good and the ORD analysis as poor.) Most respondents viewed the ORD analysis as representing the agency's "official" scientific assessment, but some respondents were unaware of the existence of the competing OW analysis.

The 1974 SDWA (Sec. 1412 (e)) required EPA to enlist the advice of the National Academy of Sciences to identify health effects associated with specified drinking water contaminants and research needs. The apparent intent was to have the Academy conduct an independent scientific assessment for EPA to use in developing drinking water regulations. In 1977, the first report issued by the National Research Council's Drinking Water and Health Committee suggested that the 50 ppb drinking water standard for

[90] See the ethylene dibromide case study in Powell (1996) for a discussion of the consequences of an episode in which an EPA policymaker during the early years of the Reagan administration attempted to depart from standard risk assessment procedures in pursuit of a regulatory relief policy.

arsenic may not provide an adequate margin of safety (NRC 1977).[91] However, in 1980, the NRC committee changed its posture somewhat, recommending further investigation of possible beneficial nutritional effects of arsenic at low doses (NRC 1980).

In the same year, the EPA Office of Water developed ambient water quality criteria documents covering 65 contaminants, including arsenic (EPA/OW 1980).[92] This early arsenic criteria document, according to an independent toxicologist, never got out of draft.[93] This source suggests that it was during this exercise that senior careerists in Office of Drinking Water (ODW) were exposed to and may have become skeptical of the Taiwan skin cancer study (Tseng *et al*. 1968). In the same year, however, the International Agency for Research on Cancer concluded on the basis of the Taiwan study that there was sufficient evidence that ingested inorganic arsenic causes human skin cancer (IARC 1980). This international scientific consensus presented a challenge to those who viewed the interim drinking water standard as sufficiently protective. When Southwick *et al*. (1981) reported no association between arsenic in drinking water and cancer in a Utah population, their EPA-supported study provided an opportunity for another review of the epidemiological evidence of the adverse health effects of arsenic in drinking water.

As part of its fifth report (NRC 1983), the NRC Drinking Water and Health Committee conducted this review and concluded that US epidemiological studies failed to confirm the Taiwan study. Shifting from its earlier questioning of the protection provided by the interim arsenic standard, the committee stated, "It is therefore the opinion of this committee that 0.05 mg/liter [50 ppb] provides a sufficient margin of safety..." Building on its 1980 recommendation, the committee also recommended that, in the absence of new data, arsenic should be presumed an "essential" nutrient for humans based on the results of multiple mammalian animals studies. The charge to the NRC committees is negotiated between the Academy staff and the sponsoring institution. Joe Cotruvo, then-Director of the Office of Drinking Water Criteria and Standards Division,[94] and William Marcus, a drinking water program staff scientist, served as EPA project officer and liaison, respectively, to the NRC Drinking Water and Health Committee. The committee's membership changed over time, but members who advocated replacing risk assessment default models and assumptions with more biologically sophisticated methods were prominently represented.[95]

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[91] The NRC is the primary operational arm of the National Academy of Sciences.

[92] The ambient water quality criteria documents developed for the surface water program would include both human health and environmental effects information. Only a subset of the toxic pollutants for the surface water program would be germane to the drinking water program.

[93] According to this source, the draft criteria document for arsenic suggested an ambient water quality standard below the practical detection limits of analytical technology.

[94] Prior to a 1991 reorganization, the EPA Office of Drinking Water (now the Office of Ground Water and Drinking Water (OGWDW)) conducted its own risk assessments. As part of the reorganization, the separate health and ecological risk analytic staffs from the surface water and drinking water programs were consolidated in the Office of Water in the Office of Science and Technology (OST). See further discussion of the reorganization and its effects below.

[95] For example, committee member Michael Gallo, a toxicologist and currently director of the NIEHS Center of Excellence at New Jersey's Robert Wood Johnson Medical Center, has been described as a

Taking advantage of scientific material accumulated by the pesticides and air toxics program offices, ORD issued its Health Assessment Document (HAD) for inorganic arsenic in 1984 (EPA/ORD 1984).[96] Based on the Taiwan study, the HAD estimated that the 50 ppb arsenic drinking water standard resulted in an upper-bound skin cancer risk estimate of approximately 2×10^{-2} (0.02 or two percent), much higher than the 10^{-4} - 10^{-6} (one-in-ten thousand to one-in-a million) range to which EPA customarily regulates health risks.[97] Rejecting ORD's assessment, ODW relied on the evidence of arsenic's nutritional essentiality in a 1985 proposed rulemaking (50 *Fed. Reg.* 46959). Congress interceded the next year with the SDWA Amendments, which set a 1989 deadline for EPA to review and finalize drinking water standards for 83 specific contaminants, including arsenic.

However, the 1984 RCRA amendments and SARA of 1986 expanded the arsenic "scope of conflict" to include the hazardous waste community. Until this point, the capability of the EPA drinking water program to counter ORD's arsenic drinking water assessment was limited by its own modest scientific resources and those of the drinking water suppliers. A byproduct of the hazardous waste legislation was that it enabled ODW and the drinking water suppliers to subject ORD's arsenic assessment to increased scientific scrutiny by capitalizing on the resources of new stakeholders--companies with hazardous waste liabilities (e.g., ARCO) and the research arms of affected industrial sectors (e.g., ILZRO, the International Lead Zinc Research Organization, and EPRI, the Electric Power Research Institute).

The ODW also extended the scope of the scientific debate by focusing on the role of pharmacokinetics in risk assessment. In 1986, the NRC *Drinking Water and Health* report recommended that EPA consider pharmacokinetics in assessing the risks of drinking water carcinogens. A year later, the committee published its final report, the proceedings of a conference on pharmacokinetics in risk assessment (NRC 1986, 1987).[98]

"leader of the campaign" to replace risk assessment assumptions such as the linear dose-response model with methods based on a biological understanding at the molecular level (Stone 1993).

[96] The Washington, DC-based Office of Health and Environmental Assessment (OHEA) produced the HAD. This office was successor to the Cancer Assessment Group in ORD and predecessor to the current ORD National Center for Environmental Assessment.

[97] Shortly thereafter, ODW and the regulated drinking water suppliers criticized ORD's assessment for using an epidemiological study to quantitatively estimate the risks of low-level arsenic ingestion, treating the Taiwanese study population as comparable to the US population when the study population had a diet lower in protein, discounting the evidence from animal studies of arsenic's nutritional essentiality, making no distinction between Taiwanese and US drinking water consumption rates (using 2 l/day for both), and using a simple linear dose-response model (least-squares linear regression). See the discussion of these scientific issues above.

[98] The 1986 SDWA Amendments required EPA to request comments from the SAB "prior to proposal of a maximum contaminant level goal and national primary drinking water regulation," virtually eliminating the role of the NRC in evaluating specific drinking water contaminants. However, the law allows the SAB to respond "as it deems appropriate," and makes clear that SAB review must be conducted within the statutory timetable for promulgation of drinking water standards. In practice, the SAB Drinking Water Committee (formerly a subcommittee of the Environmental Health Committee) has selectively commented on the more controversial or high-stakes proposed drinking water standards, including arsenic.

Thus, ODW scientists had an external source of legitimacy for challenging the agency's no-threshold, linear cancer risk model. According to a former EPA official, the water program's health risk assessors "needed a smoking gun before they saw anything as a contaminant" and are "pushing for arsenic as a test case" to establish a precedent for a "threshold carcinogen." The threshold issue was particularly salient to the drinking water program, because consistent with its no-threshold default cancer model, the agency has implemented the SDWA's goal of preventing any known or anticipated health effects with an adequate margin of safety by setting the MCLG at zero for all drinking water carcinogens.[99] (The threshold issue may be less central to standard setting now because the 1996 SDWA Amendments authorize EPA not to promulgate the feasible MCL if the benefits do not warrant the costs. However, the estimated benefits of regulatory controls at low dose levels will continue to be sensitive to the presumed form of the dose-response curve.)

Meanwhile, in response to inter-office disagreements, EPA had referred revision of the 1984 ORD arsenic HAD to the Risk Assessment Forum for reassessment. The RAF is designated as the agency's inter-office forum for resolving scientific disputes. Its membership in 1986-87 consisted of four scientists from ORD, four from the Office of Pesticides and Toxic Substances (OPTS), and one each from the OW and Region 1. The 1986-87 RAF was chaired by Peter Preuss, former Director of the ORD Office of Health and Environmental Assessment (OHEA). The principal authors of the 1988 RAF report were Herman Gibb and Chao Chen (ORD/OHEA), Tina Levine, Amy Rispin, and Cheryl Siegel-Scott (OPTS), and William Marcus (ODW). Gibb and Chen were primarily responsible for the quantitative risk estimation and contracted with Ken Brown, then a statistician at Research Triangle Institute.[100] Gibb is the EPA scientist most commonly associated by respondents with the RAF-endorsed reassessment.

In December 1986, a draft report was peer reviewed at a public workshop at which the statistical reviewers endorsed the quantitative analysis of the Taiwan study. A revised draft was presented to the RAF in March 1987, but the Forum was unable to bring the reassessment to closure by consensus. In July, the final report was elevated to the EPA Risk Assessment Council (RAC), a management level inter-office forum for resolving science policy disputes within the agency. The ODW requested that the RAC address the issue of whether the risk of skin cancer from ingested arsenic should be down-weighted (i.e., downgraded or depreciated) relative to internal cancer risks because skin cancer is generally treatable and non-fatal. There appears to have been little scientific basis, however, for deriving the proposed down-weighting factor (i.e., dividing the skin cancer risk by 10). An ORD scientist charges, "The drinking water office would never admit it, but in the original [1988] risk assessment, they said, this is much too high, we've got to do something about this. Let's say that skin cancer is fatal 10% of the time." A former EPA drinking water official allows, "the Office of Drinking Water's assumption that 10% of skin cancers were health-threatening was arbitrary."

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[99] The House Report on the 1974 SDWA bill stated that if there is no safe threshold for a contaminant, the health-based goal should be set at zero (*Fed. Reg.* Vol., 56, p. 26460).

[100] RTI is a contract research organization in Research Triangle Park, NC.

The ORD scientist points to an inconsistency in down-weighting skin cancer relative to internal cancers. "Other cancers, including some internal cancers, are not 100 percent fatal. Should we be applying [down-weighting] factors to all non-fatal cancers?" Noting the inconsistency and precedent-setting potential, the RAC responded to ODW that the agency would not explicitly identify the factor of 10 as down-weighting due to non-lethality. Instead, it was negotiated that the factor of 10 would be called an "uncertainty factor" that would not be part of the risk assessment but would be presented in a memo from Administrator Lee Thomas to EPA offices.

In its charge to the SAB Environmental Health Committee to review the RAF arsenic reassessment, EPA asked the committee to review the propriety of the "uncertainty factor." The SAB's leaders perceived this as a policy judgment thinly veiled as science, and the committee declined to review the report. According to a respondent who was an SAB member at the time, Board chairman Norton Nelson was angered that EPA would even ask the SAB to review the matter.[101] In June 1988, Administrator Lee Thomas endorsed the RAC's recommendation in a memorandum permitting managers to down-weight ingested inorganic arsenic risks by an "uncertainty" factor of 10 (Thomas 1988). EPA released the RAF *Special Report* a month later, but without the benefit of SAB's imprimatur.

Based on the Taiwanese database, the RAF reassessment concluded that the 50 ppb arsenic drinking water standard resulted in a skin cancer risk estimate of 2.5×10^{-3} (or 0.25 percent). To attain a 10^{-4} cancer risk using the 1988 estimate, the MCL for arsenic would have to be lowered to 2 ppb (EPA/OW 1994). However, applying the "uncertainty" factor of 10 yielded a skin cancer "weighted-risk" on the order of 10^{-4}, or just within the bounds of what EPA ordinarily considers an "acceptable" risk (10^{-4}-10^{-6}). After the RAF report was issued, press reports suggested that ODW was considering proposing an MCL of between 30 and 35 ppb, but other offices were seeking a lower standard (*Inside EPA*, 10/7/88, p. 6).

On a separate track, the Drinking Water Subcommittee of the SAB Environmental Health Committee met days prior to Thomas's June memo to review arsenic in drinking water. Richard Cothern, then Executive Secretary for the committee, was a former colleague of Bill Marcus's in ODW.[102] During a June 1988 meeting in Cincinnati, Cothern introduced to the SAB evidence synthesized by Marcus and Amy Rispin (OPTS) during the course of the RAF arsenic reassessment. Marcus and Rispin had brought to light information concerning methylation as a possible detoxification pathway for arsenic.[103] The RAF report concluded that the issue merited further investigation but found that the

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[101] New York University's Norton Nelson was appointed SAB chairman during the post-Gorsuch years and refused to allow the Board to be drawn too closely into the policy sphere on a number of occasions (see discussion in Jasanoff 1990).

[102] The SAB Secretariat is housed in the Administrator's Office and staffed by EPA employees.

[103] See, for example, the discussion of Valentine *et al*. (1979) and Buchet (1981) above. Marcus and Rispin's report was included as an appendix to EPA/RAF (1988).

available studies were inconclusive regarding a methylation saturation point or the relevance of the pathway to carcinogenesis. A former drinking water program scientist expected the SAB Drinking Water Subcommittee to be similarly equivocal.

Foreshadowing things to come, however, in 1988, SAB Director Terry Yosie observed that the Board's "continuing efforts to persuade EPA to utilize pharmacokinetic data in risk assessment has begun to see results" (Yosie 1988). In September 1989, the SAB recommended that the EPA revise its 1988 arsenic assessment considering the potential reduction in cancer risk due to detoxification (EPA/SAB 1989). "Unlike its own scientists, EPA couldn't ignore the 1989 SAB report," said the former drinking water program scientist. In the same month, EPA was sued by the Bull Run Coalition.[104]

Setting the RfDs

After the SAB advised EPA to revise its skin cancer risk estimate for ingested arsenic, the agency set up an interoffice workgroup to develop an oral Reference Dose (RfD) for arsenic for the EPA's Integrated Risk Information System (IRIS). The RfD is designed as an allowable exposure (mg/kg-body weight/day) for non-cancer risks.[105] The ensuing internal fight over the RfD value was a proxy battle over the drinking water standard, according to a former ORD official. (See discussion below regarding the drinking water equivalent level.) Charles Abernathy, who had assumed the arsenic portfolio from William Marcus in 1989, was the lead Office of Water representative on the workgroup.

During the course of the RfD negotiations, the assumed Taiwanese drinking water consumption rate was increased from 3.5 l/day for men and 2 l/day for women to 4.5 l/day for both sexes, effectively lowering the risk estimate for arsenic (see exposure assumptions discussion above). Other negotiations concerned the appropriate uncertainty factor to employ. EPA's standard procedure in deriving an RfD is to ascertain the "no observed adverse effect level" (NOAEL) and then divide that by an uncertainty factor to provide a margin of safety. With the proposed values of 1 (corresponding to no safety factor), 3, and 10 on the table, the staff could not come to agreement. (Recall that the agency had adopted an "uncertainty factor" of 10 to down-weight the risk of skin cancer.) The issue was elevated to the RAC and, according to an ORD scientist, ultimately set at 3 by then-Deputy Administrator Henry Habicht.

The reason there was such a fight over the RfD value for arsenic is that following standard agency procedures would have suggested an allowable arsenic intake

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[104] According to sources from EPA and industry, the "Bull Run Coalition" is simply an Oregon lawyer who takes advantage of statutory provisions that his fees are paid in cases where the agency fails to meet its legal obligations. Bill Carpenter is the attorney for the group (*Environment Reporter*, 8/25/95, p. 805). Such provisions were enacted to permit citizen groups to play a watchdog function over regulatory agencies.

[105] Chronic exposure to arsenic can lead to neurological, dermatological, vascular, hepatic, and other signs of non-cancer toxicity (EPA/OW 1993).

considerably lower than the existing MCL. The Taiwan study (Tseng *et al*. 1968) identifies a NOAEL of 0.8 µg/kg/day and thus suggests an arsenic RfD value of 0.3 µg/kg/day (using the uncertainty factor of three). Assuming water consumption of 2 l/day by a 70 kg adult yields a "drinking water equivalent level" (DWEL) of 10.5 ppb (EPA/OW 1992). Because this value for the RfD would suggest an "allowable" oral arsenic ingestion level nearly 5-fold *lower* than the current drinking water MCL,[106] a member of the IRIS workgroup says, "the arsenic RfD is not a number, like all others, but it's a range. It is a compromise....The point estimate became non-palatable due to the economic and political implications." According to a former EPA official, the endpoints of the "range" for the arsenic RfD listed in IRIS are, in fact, two separate point estimates derived by irreconcilable factions on the workgroup.[107] Because the RfD for a substance also may be used in establishing remedial objectives for soil contamination, the lack of consensus on the value for arsenic presents a decision-making challenge to Superfund site managers.

Developing Research Agendas, Buying Time

In response to the 1989 SAB recommendation, the EPA Office of Water developed a draft Drinking Water Criteria Document on Arsenic in September 1992 (EPA/OW 1992) and submitted it to the SAB Drinking Water Subcommittee in March 1993.[108] The committee found that Taiwan data (Chen *et al*. 1985) provided evidence of an association between internal cancer and exposure to high levels of arsenic in drinking water but again suggested evidence of nonlinear arsenic pharmacokinetics should be considered. Members also raised questions about the precision of analytical procedures to detect arsenic in drinking water in the 2 ppb range (EPA/SAB/DWC 1993). With Democrats controlling both the Congress and the Executive Branches for the first time in more than a decade, environmentalists anticipated a steady stream of regulations to be released from the queue. Some in the EPA water program and the regulated community, however, worked to buy time on arsenic, perhaps in expectation of SDWA legislative reforms or in an effort to build scientific consensus for a precedent-setting departure from standard EPA risk assessment procedures. The vehicle for their holding pattern was research agenda-setting. An ORD scientist alleges that the Office of Water is merely "buying time, waiting for a new administration, so somebody else will have to make the decision. Their ulterior motive for developing a research agenda is buying time, to put something before the court."

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[106] Note that using no uncertainty factor would result in a DWEL point estimate of 28 ppb, still nearly half the current MCL of 50 ppb.

[107] The range provided in IRIS is 0.1 to 0.8 µg/kg/day (EPA/OHEA 1996). By comparison, a DWEL of 50 ppb (the MCL for arsenic) implies an RfD of 1.43 µg/kg/day, exceeding the upper range of the RfD for arsenic provided in IRIS. Strictly speaking, there are other substances for which there are multiple IRIS values (fluoride and zinc), but in those cases, there has been a distinction made between the RfDs for children and adults. No such distinction was made for arsenic. It is worth noting that a 1994 Office of Water document (EPA/OW 1994) summarizing EPA drinking water standards has no RfD listed for arsenic but lists RfDs for other contaminants.

[108] The draft criteria document was prepared under contract by Life Systems, Inc.

In the 1988 reassessment, the RAF identified a broad set of future research directions covering epidemiology, mechanism of skin carcinogenesis, pharmacokinetics, and nutritional essentiality.[109] Since the SAB advised EPA to revise its assessment, at least three *ad hoc* groups inside and outside the agency have formulated arsenic research agendas. However, little original research was funded by EPA or the regulated community. Much of the analysis supported or promoted by the OW and the regulated community did not resolve scientific issues but instead served to expand the recognized scientific uncertainty by casting doubt on the Taiwan database or the analysis used in previous assessments. An ORD scientist observes that "Brown's work [e.g., Brown and Chen (1993)] threw a cloud over the exposure assessment part of the risk assessment, but it's not the type of information that would have led to a decision" (i.e., a decision to revise the standard).

In the absence of new, original research, the OW in its court negotiations has pointed to the development of research agendas as evidence of progress toward the goal of finalizing an MCL for arsenic. However, there has been no consensus among the groups which have formulated arsenic research agendas on what the most critical research needs are or whether the identified research is likely to substantially alter the agency's risk estimate or do so within an appropriate timeframe. A drinking water official says, "People are still debating on how they would address the uncertainties, on what the research needs are. Some say $1 million wouldn't improve the database incrementally because it's so good. Others say it wouldn't make a dent because it's so bad." This lack of scientific agreement suggests that the problem "may be too intractable to take a formal analytic approach to developing a research agenda," says an EPA scientist.[110]

After the 1989 SAB report, EPA formed an *Ad Hoc* Arsenic Research Recommendation Workgroup, headed by ORD/HERL's Jack Fowle. In early 1991, after conferring with the SAB Drinking Water Subcommittee, Fowle's workgroup issued a research agenda addressing the two areas that it felt contributed most to the uncertainty regarding arsenic risk assessment: (1) mechanism of cancer and (2) metabolism and detoxification. Because the agency was being sued for not meeting its regulatory deadline, the workgroup focused on research that could be conducted in 3-5 years but concluded that such research was unlikely to reduce uncertainty in the level below 50 ppb by more than a factor of a few-fold. If time were not a constraint, the workgroup considered the development of a suitable animal model for evaluating arsenic's cancer mechanism to be the top research priority (Fowle 1992).

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[109] The Office of Water's nutritional essentiality argument was seriously weakened after EPA/SAB (1989) concluded that there was a lack of human evidence. According to a former EPA drinking water official, water program risk assessors continued to make the essentiality argument during the 1990s but not as prominently as the methylation argument. According to press reports, the EPA Office of Air and Radiation (which regulates arsenic as a hazardous air pollutant) was especially strong in its opposition to the essentiality argument (*Inside EPA*, 10/7/88, pp. 5-6).

[110] A formal analytic approach would allocate scarce research funds such that they are most likely to achieve the greatest reduction in uncertainty for decisionmaking. However, there is no consensus regarding the source or magnitude of the uncertainties or the likelihood or extent that various research proposals would reduce them.

An ORD scientist claims that ODW "wanted someone to counter" the Fowle workgroup's conclusion that no near-term research was likely to sufficiently reduce the uncertainty in the arsenic risk assessment. This source suggests that "ODW wants to believe that somebody, somewhere can come up with the answer in 3 years." Whatever its intentions, ODW asked a group of ORD scientists who specialized in drinking water health effects research at HERL in Research Triangle Park, NC to develop a new arsenic research agenda. The HERL group's research agenda, coordinated by Fred Hauchman, addressed a variety of issues, including epidemiological study, but the initial focus was laboratory-based research on the role of pharmacokinetics and inter-individual genetic variation affecting arsenic metabolism.

"Hauchman's group wanted to do the research, but it couldn't get clearance to devote ORD money to the issue," remarked a drinking water program scientist. However, one ORD scientist claims that "some of the research won't amount to a hill of beans as far as reducing the uncertainty" in the near term and views HERL's interests in lab-based research as "parochial." The EPA research and water offices have also disagreed about which office bears responsibility for funding the research. Another ORD scientist suggests that "there are some tremendous opportunities for fostering collaborations with [industry]. They have a lot at stake, and we have an interest in getting the best research done."

In May 1991, the agency made plans to reassess arsenic based on current data (Fowle 1992). Shortly before this announcement, as part of an administrative reorganization instituted by then-Assistant Administrator for Water LuJuana Wilcher, Jim Elder was shifted from the Office of Water's surface water regulatory program to replace Mike Cook as Director of the renamed Office of Ground Water and Drinking Water. Elder would be responsible for getting the proposed rule for arsenic out under the court-ordered deadline of November 1995.

In December 1991, the Society of Environmental Geochemistry and Health (SEGH) launched an Arsenic Task Force. The SEGH is a scientific society including researchers from the public and private sectors as well as academics. Fairly or unfairly, some EPA officials perceive that it is dominated by representatives of the regulated community.[111] SEGH created the Arsenic Task Force in response to EPA's court agreement with the Bull Run Coalition to propose an arsenic drinking water rule and in recognition of the impacts that an adjustment of the risk estimate would have not only on drinking water but also on contaminated site remediation and coal fly ash disposal. SEGH appointed Willard Chappell (University of Colorado at Denver) and Charles Abernathy (EPA Office of Water) as Task Force co-chairs. Task Force members include EPA officials, academics, and environmental consultants.[112]

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[111] An independent toxicologist who is a long-time member of SEGH specifically views its issue-specific task forces with some suspicion.

[112] Task Force members Barbara Beck, Ken Brown, and Warner North, Sr. VP of Decision Focus, Inc., work for consulting firms that do work for the water, mining, and electric utility industries, but they have also done work for EPA, as employees and contractors. North has served as a member and consultant to

The SEGH Arsenic Task Force secured sponsorship for a 1993 conference on arsenic exposure and health effects from EPA, the Agency for Toxic Substances and Disease Registry (ATSDR),[113] the American Water Works Association (AWWA), the American Mining Congress (AMC), the International Council on Metal in the Environment (ICME), the Electric Power Research Institute (EPRI), and US Borax. At the conference, two industry-supported researchers cast doubts on EPA's previous risk assessment (EPA/RAF 1988). Ken Brown presented the problems he had uncovered regarding the Taiwan exposure data and discussed their implications for interpretation of the original skin cancer study. How-Ran Guo also claimed to have evidence of a non-linear relationship between arsenic drinking water concentrations and urinary cancers.[114] However, academic researchers Allan Smith and Claudia Hopenhayan-Rich also discussed their skepticism of the methylation threshold hypothesis and presented their internal cancer risk estimates. The running scientific battle was continued at SEGH and Society of Toxicology Meetings in 1994 and 1995.

As EPA considered tightening the arsenic drinking water standard to as low as 2 ppb in light of the internal cancer evidence (Smith *et al.* 1992), the regulated community sought to persuade the agency to pursue a research agenda. During this period, SEGH Arsenic Task Force members Willard Chappell and Warner North met with ORD Assistant Administrator Robert Huggett to urge additional arsenic research. Afterwards, representatives of the American Water Works Association and its Research Foundation met with Huggett to seek ORD's participation in the planning of a workshop to develop a research agenda.[115] AWWARF convened the workshop in May 1995 and identified two research projects that it would pursue immediately, an analytical method for detecting arsenic at low levels in blood and urine and a feasibility study for an epidemiological study. The workshop was held five months after Assistant Administrator for the Office of Water Robert Perciasepe declared that EPA needed to pursue additional research and would be unable to meet its court-ordered deadline of November 1995. According to press reports, the Office of Water planned to use the AWWARF list of research needs in its negotiations with the plaintiffs to demonstrate that significant areas of research must be completed

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committees of the SAB. (See further discussion of North's role below.) According to a Task Force member, when the group was established, the SEGH tried to give it a balanced membership. According to this source, member T. A. Tsongas of Washington St. Univ. has a background in public health and environmental activism. Paul Mushak was a member of the Task Force as it was originally constituted but resigned after the group's 1993 meeting. Rebecca Calderon and David Thomas, EPA/ORD scientists with HERL, are Task Force members. Former SAB staffer Richard Cothern has also been an active member.

[113] The Comprehensive Environmental Response, Compensation, and Liability Act of 1980 established ATSDR. Funded by Superfund, ATSDR develops toxicological profiles for pollutants on contaminated sites.

[114] A former EPA drinking water official says that Guo's work was never presented to the drinking water program management.

[115] Huggett designated Peter Preuss, Director of the ORD Office of Science, Planning and Regulatory Evaluation and chair of the RAF which reviewed the 1988 arsenic reassessment.

before a new standard can be proposed (*Risk Policy Report*, 5/16/95, pp. 16-17). (See further discussion of ongoing research activities in the concluding section.)

Things Left Undone

The major frustration of a former drinking water official concerning arsenic was the lack of new research available when the time for decisionmaking arrived: "The political appointees should have never been put in that type of position." Interviewees offered a variety of reasons why substantial new research had not been done over the last 10 years. Some simply feel that the existing database was more than adequate for the rulemaking and that further research is unwarranted. Others point to the multi-million dollar costs of sufficiently large, sophisticated epidemiological studies or the cost and potentially long delay associated with basic research into arsenic's toxic mechanisms. Another possibility is that the agency simply regards arsenic as a lower priority than other drinking water contaminants such as pathogenic microorganisms and disinfection byproducts to which it is devoting its scarce research resources. In the view of some, arsenic suffers from being "an orphan material," affecting a large number of small communities and having no single, deep-pocketed firm or sector being affected greatly enough to adopt it and fund the research. AWWARF, for example, offered to provide some funding for additional research, but some EPA officials believed that the level of resources available from the drinking water suppliers (on the order of $1 million) would be insufficient to reduce the scientific uncertainties by the necessary order of magnitude.

A former EPA drinking water official, however, rejects the "orphan argument" and suggests, instead, that "a lot of people were comfortable not knowing." This source believes, "It was a conscious act on the part of several people over time. I never could figure out how much of it was from within EPA or from OMB [the President's Office of Management and Budget]. They resisted properly funding the 1986 Safe Drinking Water Act program. It seemed like they wanted to starve the program... There was tension between ORD and ODW on who should fund the research...but no matter who the Administrator was, ODW didn't compete well within the agency... The Office of Water was a program that was heavily earmarked by Congress with the Chesapeake Bay, the Great Lakes, and construction grants. That took away much of our discretion, and there was no chance of a net increase in the budget.... They were relying on how the academic literature was going to turn out for them, and the agency was left holding the bag." Thus, the agency could not rely on others to spontaneously provide it with the scientific information required for regulatory decisionmaking. The question remains, however, whether the research would have been done even if the drinking water program had greater discretionary resources given the comfort of "not knowing."

For several years leading up to EPA's 1995 decision, various groups had developed and debated arsenic research agendas. Two schools of thought disagreed about whether research that could be conducted in a reasonable amount of time would reduce the scientific uncertainties to the extent that it should alter the decisionmaking calculus. In effect, both sides were presuming what sort of evidence was needed to sway regulatory

decisionmakers. The clashing camps were operating in the absence of any formal articulation by EPA policymakers as to what they would consider sufficiently compelling scientific evidence to warrant any particular level of the drinking water standard. It should not be surprising, then, that EPA and the regulated community have been, as an environmental lawyer notes, "strategizing on research rather than doing it."

Effects of the 1991 Reorganization

As indicated above, then-Assistant Administrator LuJuana Wilcher reorganized the Office of Water in 1991. An important change was the separation of the Office's analytical staff from the surface water and ground water/drinking water programs. The health and ecological risk assessors, along with economists and engineers, were consolidated under the new Office of Science and Technology (OW/OST). According to a former EPA drinking water official, the reorganization was intended, at least in part, to institutionally separate the risk assessment and risk management functions. One result of the reorganization, says this source, was that the OST risk assessment staff utilized scientific arguments to pursue an arsenic *policy* independent from the Office of Ground Water and Drinking Water (OGWDW) where some staff, principally on the basis of emerging evidence for internal cancers, advocated tightening the drinking water standard for arsenic. Prior to the 1991 reorganization, the situation within EPA appeared basically as a case of dueling risk assessors, with ODW pitted against ORD. After the reorganization, the internal dynamic became more complex and fragmented, and the agency was speaking to itself and to others with multiple voices. This, in turn, had important effects on the communication of scientific information up the chain of command to policymakers.

Communicating the Science to Agency Leadership

Half of the responses rated the communication of the science to agency decisionmakers as good-to-very good (5); the other half rated it as poor (5). In December 1994, a decisional briefing was held for Assistant Administrator Perciasepe, who had been briefed prior to the meeting by his own staff. According to a former drinking water official, "It was a packed room. Meeting attendance is a good gauge of the importance of a decision. When the GC [then-General Counsel Jean Nelson] and an AA [Lynn Goldman, Assistant Administrator for OPPTS] show up, you know you have a biggy." The Water Office staff critiqued the available scientific studies and recommended pursuing additional research. The former drinking water official commented that "Abernathy was very good at identifying the holes in the Taiwan database" and suggested that in the minds of drinking water program management, the problems with the dose-reconstruction identified by Brown were "incredibly important." Assistant Administrator Goldman and Peter Preuss, representing ORD, both reportedly suggested that there was no research that could be done in the near term that would substantially alter the 1988 risk estimate. Many of those who gave the communications poor marks faulted the Water Office staff for undermining the credibility of the epidemiological evidence. Whether that constitutes a distortion, of course, depends upon one's assessment of the evidence.

Apparently, however, there was a serious miscommunication regarding the issue of internal cancers. The internal cancer evidence--and the risk assessment by Smith *et al.* (1992) in particular--had caused some within the Office of Ground Water and Drinking Water to believe that action was warranted. Although it remains unclear who is responsible for starting it, a rumor got started that Smith later reversed his position regarding the evidence of internal cancers. According to a former EPA official, OGWDW management unwittingly conveyed this misinformation to Assistant Administrator Perciasepe as well as to members of Rep. Waxman's (D-CA) staff to support the recommendation that more research was needed prior to revising the arsenic standard. The rumor was not limited to inside EPA. An environmental lobbyist reported hearing that Smith had changed his mind from sources in the drinking water industry.

The details surrounding the miscommunication within EPA remain somewhat sketchy. According to an EPA official in the Office of Water, at a 1994 scientific workshop on arsenic held in Annapolis, MD, Smith presented preliminary findings from data collected in S. America which had not produced statistical evidence of a correlation between arsenic levels in drinking water and internal (i.e., bladder) cancers. OST staff briefed OGWDW management on Smith's presentation. According to the former EPA official, the OST staff told the OGWDW management that "Smith had changed his mind." The OW official denies this and suggests that Smith has never reversed his position. Smith also became aware of the rumor, and although he "has questioned the rationale for setting the arsenic drinking water MCL below 10 ppb," a level where food appears to become the dominant pathway of ingested inorganic arsenic, he denies backing away from his 1992 analysis. In fact, while the abstract of Smith's 1994 Annapolis presentation suggests what many scientists would consider, by itself, marginal evidence for an association between arsenic in drinking water and internal cancer,[116] it concludes by saying, "these findings provide new support for the evidence from Taiwan that arsenic ingestion increases the risk of bladder cancer" (Smith *et al.* 1994). "If anything," Smith now says, "the evidence [of internal cancers] has gotten stronger" as a result of evidence from the completed epidemiological studies in Argentina and Chile (Allan Smith, Professor of Public Health, University of California, Berkeley, personal communication).

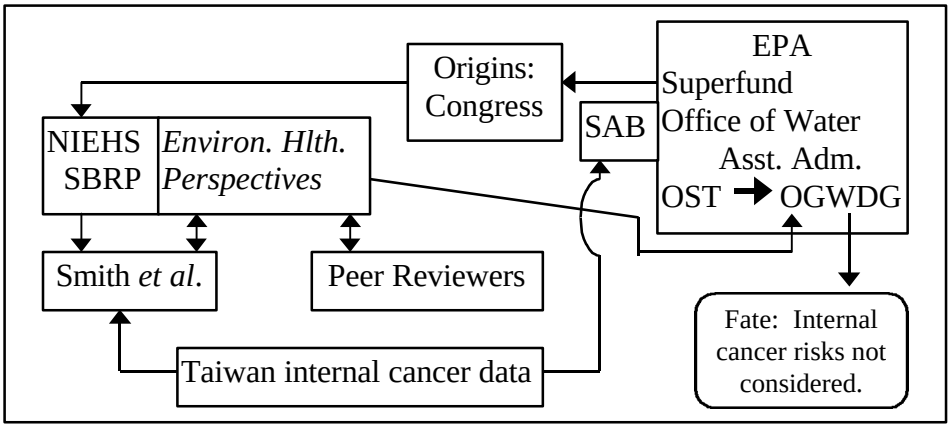
Senior EPA drinking water management read the internal cancer risk analysis by Smith *et al*. (1992) as reported in the NIEHS journal, *Environmental Health Perspectives*. In terms of a fate and transport analogy, management was directly exposed to scientific information that had not first passed through a filter (i.e., the internal scientific gatekeepers in OST). Initially, there was a strong reaction to the information at the management level in close proximity to the political decisionmaker. If OST staff did

[116] The abstract reports a small difference in the prevalence of a biomarker (micronucleated bladder cells) between subjects in N. Chile exposed to high (600 μg/L) and low (20 μg/L) arsenic levels in drinking water. An ecological epidemiological study in the Province of Codoba, Argentina found a relative risk ratio for bladder cancer of approximately 2 for both males and females, using all of Argentina as a referent population (Smith *et al.* 1994). A rule of thumb applied by some epidemiologists is that for a single study to be persuasive by itself, a relative risk ratio of 3 or 4 is the lower limit (see: Taubes 1995).

indeed misrepresent Smith's Annapolis presentation to OGWDW managers, they may have been seeking to "repair the damage" caused by the unfiltered exposure to the scientific information at this intermediate level. It is worth noting that the distinction between: 1) an observed association between internal cancer and exposure to high levels of arsenic in drinking water which the SAB endorsed in 1993 and 2) the ability to confidently estimate the risk of internal cancer at low levels of exposure, also seems to have been blurred somewhere in the communications process. According to the former EPA official, the lack of evidence for internal cancers was the pivotal basis for the OGWDW staff's recommendation to Assistant Administrator Perciasepe that the agency pursue additional research instead of immediately proposing a revised arsenic in drinking water standard.

Figure B-2 illustrates the fate and transport of the internal cancer risk assessment. Note the circuitous route that the flow of information takes. It begins with high-level demand for independent basic research relevant to contaminated site remediation, the Congressionally-designated interagency transfer of resources from EPA to the NIEHS Superfund Basic Research Program (SBRP). It ends with the decision not to consider the potential risks of internal cancers.

Figure B-2. Fate and transport of the assessment of internal cancer risks.



The Role of External Scientists in the Process

Two-thirds (8) of responses rated the role of external scientists in the decision-making process as significant-to-very significant. According to an EPA drinking water official, the lack of unity among the agency scientists provided an opportunity for external scientists to play prominently. The most significant aspect of the participation of external scientists in the process appears to have been legitimizing OW's call for more research prior to revising the arsenic in drinking water standard. As discussed above, the NRC Drinking Water and Health Committee and the SAB drinking water panel both promoted consideration of arsenic pharmacokinetics and departure from the linear cancer model. As was the case in the 1991 lead drinking water rulemaking (see accompanying case study), some EPA scientists asserted that the SAB drinking water panel had exceeded its legitimate scope of review by commenting on the health assessment. (The SAB Health and Executive Committee chairs, however, signed-off on the 1989 review since the drinking water panel was, at the time, a subcommittee of the SAB Health Committee.)

A number of external scientists who have played particularly large roles in the process were, to a greater or lesser extent "insiders." For example, many observers associate Warner North closely with the 1989 SAB report. At the time, North was Vice Chair of the SAB Environmental Health Committee through which the Drinking Water Subcommittee reported. When the Subcommittee's EPA staffer Richard Cothern approached North with the panel's report from its 1988 meeting in Cincinnati, the controversial issue of a possible detoxification pathway for arsenic was framed as a false dichotomy between the linear, no-threshold dose-response model and the threshold or "hockey stick" model. By pointing out that there are any number of scientifically plausible non-linear dose-response curves lying between these two extremes, North helped negotiate the report through the internal SAB review process. (Note, however, that despite the carefully crafted language of the 1989 SAB report, the false dichotomy between the linear, no threshold and threshold models has proved to be a hardy perennial.) North later presented a paper describing the 1989 SAB report and its history at a meeting of the SEGH in 1991 organized by Jack Fowle and Barbara Beck.

Beck, a former EPA scientist and the Region I representative to the 1986-87 Risk Assessment Forum, has been a prominent critic of the 1988 RAF arsenic reassessment from her new position with Gradient Corporation. Ken Brown, an EPA contractor on the RAF reassessment, later, serving as a consultant to the AWWA, unearthed evidence that damaged confidence in the 1988 skin cancer risk estimate. Paul Mushak, a principal external author of the 1984 ORD arsenic Health Assessment Document, has prominently defended the agency's risk assessment against its critics. In addition to EPA's official science advisors, "there has been an opportunity by those outside the agency to present information to the leadership of the Office of Water," according to an industry scientist. An EPA drinking water official says Perciasepe met to discuss scientific issues with "outside bodies that lobbied him, drinking water utilities (primarily from California), the AWWA, and also some people from ARCO concerned about Superfund."[117]

4. Science in the Final Decision

Mirroring the polarized opinion about the adequacy of the available science for decisionmaking, the factors most frequently mentioned by respondents as impeding the use of science in the decision of whether to revise the arsenic drinking water standard or pursue additional research were: economic and political considerations (5) and reluctance to consider new scientific findings (i.e., the evidence for a non-linear dose-response model and/or the problems with the Taiwan study dose-reconstruction) (5). A narrow majority of responses (7 of 12) rated the impact of the scientific information on the decision to call for further research as low.

By most accounts, the decisionmaking authority was concentrated in the hands of Assistant Administrator Perciasepe. Assistant Administrator Lynn Goldman, a credentialed scientist,[118] however, "was satisfied that the information justified lowering the standard," according to a former EPA official. But Perciasepe's staff did not agree. "Perciasepe had a conversation with Goldman, and they decided that they wouldn't elevate it. It was brokered at the AA [Assistant Administrator] level rather than bringing in the DA [Deputy Administrator Robert Sussman]." This source observes that decisionmaking authority vested in the Assistant Administrator was typical of EPA regulatory decisionmaking in the Browner administration.

Based on Assistant Administrator Perciasepe's January 1995 court declaration, not only the scientific uncertainties but also the costs and distribution of those costs were important in the decision to pursue additional research (Declaration of Robert Perciasepe, Amended Consent Decree, *Donison, et al. v. EPA*, No. 92-6280-HO

Not only the scientific uncertainties but also the costs and distribution of those costs were important in the decision to pursue additional research.

[117] The intent here is to simply map out who presented scientific information to the decisionmaker. As a former EPA official noted, such access to EPA policymakers by advocates on both sides of the issue is not unusual.

[118] Goldman is a physician with a Masters in Public Health.

(and consolidated cases) U.S.D.C. Oregon, January 9, 1995). A 1994 draft Water Office staff analysis provides estimates of the regulatory impacts under five MCL options that were available at the time of the 1995 decision (Table B-2):[119]

Table B-2. Summary of Regulatory Impacts for Arsenic under Five MCL Options

MCL options	2 ppb	5 ppb	10 ppb	20 ppb	50 ppb
Total Annual Cost ($ Millions)	2,086	617	266	74	24
Annual Household Cost for Small[a] Systems ($)	261-1,454	252-1,423	266-1,412	275-1,301	269-1,266
Number of Systems Needing Treatment	12,386	4,924	1,949	595	160
Number of People with Reduced Exposures (millions)	31.7	11.0	4.0	1.0	0.2
Annual Skin Cancer Cases Avoided	127.3	73.9	34.3	17.7	7.6

a - Small systems serve between 25 and 3,300 people.
Source: EPA/OW Summary of Regulatory Impacts. Draft: 11/30/94 (mimeo)

A variety of political forces also were aligned against making the arsenic standard more stringent. The administration wanted to avoid disrupting ongoing SDWA negotiations with Congress. "We were afraid that it [revising the arsenic standard] would hurt legislative negotiations" on amending the SDWA, according to a former drinking water official. An EPA official observes, "It was yet another imposition on the small [drinking water] providers with whom EPA already has its biggest problems." Although it may be secondary to the broader issue of unfunded mandates, the EPA official suggests that the arsenic standard, "is also tangled up in western water supply issues. The costs are pretty big, not astronomical, but the distribution of the costs is disproportionately on vocal, organized, western small systems.... They are a more homogeneous constituency than the eastern municipal providers, and they're already stressed--those are deserts that they're watering." It should also be recalled that the court declaration came on the heels of November 1994 elections in which Republicans gained a congressional majority. Although the House of Representatives in September 1994 passed a bill (H.R. 3392) to amend the SDWA that called for a National Academy of Science study on arsenic and postponed the deadline for proposing a new standard until the end of 1996, after the fall elections, EPA was firmly in the cross-hairs of a hostile Congress, and the tide for dramatic regulatory reform seemed unstoppable. If it had not been a foregone conclusion beforehand, the tenor of the early days of the 104th Congress closed the door on EPA proposing a revised arsenic drinking water standard by November 1995.

According to a former EPA drinking water official, "We didn't think the evidence was strong enough on the risk assessment to feel comfortable that we could withstand outside review. In the environment we had, we felt we couldn't get away with anything but an iron-clad proposal. There was some chance that it [a proposed rulemaking] would be remanded, but there was a larger context.... There had been a crush of criticism of the

[119] EPA's cost estimates may have changed since this analysis was conducted. According to an environmental lawyer, lower cost water treatment technologies have been identified.

scientific validity of our decisions. We didn't feel that we could say it's affordable to go below 50 ppb. The decision had a bigger context than arsenic in drinking water." An industry scientist says, "Many non-agency scientists feel that additional research is needed, but that's not the primary cause for calling for more research. It is driven by the large economic stakes" that extend beyond the drinking water sector to include hazardous waste and contaminated site remediation issues.

An ORD official asserts "The situation is really very simple. The science indicates a high risk from current arsenic levels. But the problem is expensive to deal with and is mostly a Western problem, so the agency doesn't want to act. However, it doesn't want to say that it doesn't want to act. So, the managers turn back to science for a way out. On arsenic, [Assistant Administrator] Perciasepe wants to do more research to punt—[this source] and others have told him that more research won't change the situation. The science was not used because the risk managers didn't want to make the decision called for by the risk information. This is often an issue."

While not discounting the political expediency of "punting" on arsenic, the situation may not be quite so clear-cut.

While not discounting the political expediency of "punting" on arsenic, the situation may not be quite so clear-cut. A factor that appears to have been decisive in the thinking of some EPA water program managers was that the perceived health costs of delay to pursue additional arsenic research were not unduly large because of the limited health threat posed by skin cancer. "Skin cancer is curable. So, delay would not be that harmful for that many people," says a drinking water official. In fact, a number of EPA sources noted that Margaret Prothro, who was Acting Assistant Administrator for Water prior to Perciasepe's confirmation, was successfully treated for skin cancer and minimized its gravity.

Initially, the evidence of a link between internal cancers and arsenic in drinking water had caused considerable concern. A former EPA drinking water official recalled his initial response to "the Smith paper"(Smith *et al.* 1992), "My god, we've got to do something!" According to this source, the senior program management were prepared to recommend "a zero MCLG" if they had been given "convincing science that there were internal cancers." Agency scientists, however, disagreed about the interpretation of the evidence. In the face of conflicting opinion, the message that "Smith had changed his mind" was apparently sufficient for decisionmakers to discount the consideration of internal cancers (at least at levels below the current standard).

The precedent that EPA established in interpreting the SDWA appears to have been another factor impeding a more candid discussion of the role of science in the decision to pursue additional research. According to an ORD official, arsenic is "an example of where the statute's wrong because it doesn't allow the decision maker to be honest." An EPA drinking water official says, "If the statute had been more flexible, we

could have gone to a mid-range. But the statute forced you to a low level."[120] Feeling "hamstrung by the act," water program decisionmakers had an incentive to focus attention on the scientific uncertainties to rationalize their decision. However, prior to the 1996 SDWA Amendments, implementation of the statute had been conditioned by the precedent EPA set in interpreting what is economically "feasible." In implementing the SDWA consistently with its default, no-threshold cancer model, EPA has set the unenforceable health goal (MCLG) for carcinogens at zero. The enforceable standard (MCL) is to be set as close to the MCLG as feasible, considering available technology and cost. Treatment technology for arsenic exists (i.e., reverse osmosis) but is capital-intensive, making it less affordable for the small suppliers who would be most affected by a more stringent standard. However, EPA has interpreted economic "feasibility" to mean reasonably affordable for large public water systems.[121] (Citing this interpretation, EPA downplayed questions of small system affordability in the case of the 1991 Lead/Copper Drinking Water Rule. See accompanying case study.)

An ORD official reports that the some agency scientists recommended that EPA grant waivers for small communities for whom treatment might be unaffordable instead of characterizing the science as inadequate for regulatory decisionmaking. However, "attorneys from the OGC," says this source, "were troubled that this [waivers] was not a viable option."[122] The agency's lawyers may have been troubled by this option because it was inconsistent with precedented statutory interpretation. This case, therefore, illustrates both the rationale for, and perhaps the aftermath of, an OGC policy begun in the 1980s to shift more of the burden of support for EPA rulemaking from singular statutory interpretations onto science.[123]

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[120] The 1996 SDWA Amendments now make the issue moot; however, a former EPA official contends that EPA had sufficient flexibility prior to the amendments to adopt a mid-range standard for arsenic. As evidence on this flexibility, the former agency official cites EPA's prior use of Practical Quantitation Limits (PQLs, i.e., setting standards based on technology-based analytical detection limits) to "come up with a mid-range" for other drinking water contaminants. Another EPA official disagrees, however, stating that although the SAB Drinking Water Committee questioned the reliability of detection at 2 ppb, the PQL for arsenic was low enough that it would not provide a means of justifying a mid-range standard. Therefore, in this case, disagreement regarding whether EPA had sufficient flexibility under the SDWA may turn on differing judgments about the PQL for arsenic (which would involve a judgment about acceptable rates of false positives and false negatives) and the affordability of a standard based on the PQL for small systems.

[121] The agency based this interpretation on 1974 House report language and a 1986 senatorial statement in the Congressional Record (54 *Fed. Reg.* 22093-22094). This interpretation of feasibility was reinforced by the Senate Report (S. Rept. 104-69, Discussion of Section 6) for the 1996 SDWA Amendments: "Feasible means the level that can be reached using the best available treatment technology that is affordable for large, regional drinking water systems."

[122] A former EPA drinking water official differs with this interpretation, suggesting that the decision never reached the point of seriously considering options such as waivers because the scientific uncertainties were a threshold issue.

[123] According to a former EPA official, in the 1970s through the early 80s, "the rationale to justify a certain standard was generally that it was a matter of statutory interpretation, when in fact, if you look behind it, it's a policy call. It was a lot easier for the agency to say that the law forced it to act and not to admit that there are other valid interpretations." This source suggest that Frank Blake, the General Counsel during the Thomas administration, "was more of a purist in terms of making a distinction

5. Concluding Observations

Representatives from other EPA offices are concerned with the scientific precedent that OW is setting, fearing that the hurdle of scientific proof will become so high that virtually all rulemaking could be challenged on the basis of insufficient science. Many also view arsenic in drinking water as a test case for what EPA considers sufficient epidemiological evidence for regulatory decisionmaking. EPA's 1986 cancer risk assessment guidelines state that "If available, estimates based on adequate human epidemiological data are preferred over estimates based on animal data" (EPA 1986). To determine what constitutes adequate data, some would like to see a consistent minimum standard applied. However, the "value of information" depends on the decisionmaking context. The adequacy of data will depend upon the compliance costs and the consequences of inaction. EPA drinking water officials considered the consequences of inaction--as they understood them, i.e., skin cancer--not to be too severe. A drinking water official acknowledges the conditionality on cost, saying, "The costs were too high given the uncertainties in the science."

Many observers also see arsenic as an important test case for determining the weight of evidence that is necessary to justify departing from EPA's default linear cancer model. Unresolved, however, "is the question of what's adequate data for departing from the default," according to a drinking water official. "We have resolved to pursue additional research but are holding off the decision as to what constitutes enough." Essentially, this indecision places researchers in the position of not knowing what hypothesis to test, makes it less likely that further research will facilitate future decisionmaking, and also provides little incentive for research supporters to allocate resources to the problem. There has been some diffuse feedback from the EPA decisionmakers to scientists that more research is desired, but the investigators have no idea what evidence the policymakers will consider compelling.[124] Therefore, while decisionmakers may find it difficult to specify what evidence they (or their successors) would consider compelling, and while it may be irrational to establish across-the-board guidance on what constitutes "adequate" science for all regulatory purposes, not answering the question in a specific case also presents problems.

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between policy and law." Blake was also concerned that to revise decisions based on statutory interpretations required changing the law, whereas policy decisions could be changed on the basis of adding new scientific information to the record of decision. Thus EPA lawyers shifted more of the burden of supporting policy decisions from statutory interpretations onto science.

[124] According to an independent risk analyst, EPA's decision not to regulate gasoline vapors on the basis of liver tumors in male rats provides a counter-example in which then-Assistant Administrator for Pesticides and Toxic Substances John Moore explicitly stated what experimental evidence he would consider sufficiently compelling to depart from the agency's default assumption that humans are at least as sensitive as the most sensitive animal species. In the gasoline vapor case, the question of "how much science is enough" was formulated as a testable hypothesis, the studies were done, and the cancer mechanism in rats was found to be inapplicable to humans.

Some respondents claimed that EPA careerists had overstepped their boundaries into policymaking. It is clear from their comments that the opposing scientists and analysts are heavily invested professionally and intellectually in their points of view. Whereas the lead in drinking water case study provides an example of a group of EPA staff outside the water program using science as policy entrepreneurs to push through a regulatory rulemaking, the arsenic in drinking water case gives the impression of a group of agency water program staff strategically employing science to promote departure from the standard risk assessment procedures (in this case, the linear, no-threshold cancer model) that originate outside the program. The pharmacokinetic information the regulatory program staff mustered, however, was not "actionable." Using a fate and transport analogy, an EPA scientist suggests that "somewhere [in the agency] there are barriers to exposure" that prevent consideration of the information. "The information is not being excreted or actively attacked, but it's being stored in the fat." It remains to be seen what is necessary to strengthen and mobilize the information for use in regulatory decisionmaking.

Epilogue: Under the provisions of the 1996 SDWA Amendments, EPA was scheduled to issue a research plan for arsenic in drinking water by February 1997. The legislation also authorized $2.5 million for the research. EPA allocated $2 million and AWWARF and the Association of California Water Agencies contributed a combined $1 million to a joint request for applications (RFA) for research grants. In a December 6, 1996 *Federal Register* notice, EPA solicited public comments on the proposed research topics.[125] The proposed joint RFA and ORD's in-house arsenic research plans were both submitted for review to the Board of Scientific Counselors, a newly-formed EPA official science advisory panel that reports directly to the Assistant Administrator for ORD. In addition, AWWARF is supporting University of California's Allan Smith and colleagues to evaluate possible epidemiological studies that might be done on arsenic. The National Research Council has also formed a committee to consult with EPA on the agency's study of arsenic in drinking water.

[125] The 1996 SDWA Amendments required EPA to consult with the National Academy of Sciences, other Federal agencies, and interested public and private entities in conducting the arsenic study. EPA's December 1996 solicitation was reportedly made in response to a complaint filed by the National Resources Defense Counsel that there had been no opportunity for public input into the research plan.

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LIST OF ABBREVIATIONS

AA	Assistant Administrator
ARCO	Atlantic Richfield Company
ARAR	applicable or relevant and appropriate requirements
ASARCO	ASARCO Incorporated
ATSDR	Agency for Toxic Substances and Disease Registry
AWWA	American Water Works Association
AWWARF	American Water Works Association Research Foundation
CAG	Cancer Assessment Group
DWC	Drinking Water Committee
DWEL	drinking water equivalent level
EPA	Environmental Protection Agency
HAD	Health Assessment Document
HAP	hazardous air pollutant
HERL	Health Affects Research Laboratory
ILZRO	International Lead Zinc Research Organization
IRIS	Integrated Risk Information System
LMS	linearized multistage (model)
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
MLE	maximum likelihood estimate
NIEHS	National Institute of Environmental Health Sciences
NOAEL	no observed adverse effects level
NRC	National Research Council
ODW	Office of Drinking Water
OGC	Office of General Counsel
OGC	Office of General Counsel
OGWDW	Office of Ground Water and Drinking Water
OHEA	Office of Health and Environmental Assessment
OPPTS	Office of Pesticides, Pollution Prevention and Toxic Substances
OPTS	Office of Pesticides and Toxic Substances
ORD	Office of Research and Development
OST	Office of Science and Technology
OW	Office of Water
PHS	Public Health Service
PQL	practical quantitation limits
RAC	Risk Assessment Council
RAF	Risk Assessment Forum
RCRA	Resource Conservation and Recovery Act of 1976
RfC	reference concentration
RfD	reference dose
SAB	Science Advisory Board
SARA	Superfund Amendments Reauthorization Act of 1986
SDWA	Safe Drinking Water Act of 1974
SEGH	Society of Environmental Geochemistry and Health
USDC	US District Court