Control of Dioxins (and other Organochlorines) from the Pulp and Paper Industry under the Clean Water Act and Lead in Soil at Superfund Mining Sites: Two Case Studies in EPA's Use of Science

Mark R. Powell

Discussion Paper 97-08

March 1997 (Revised)

1616 P Street, NW
Washington, DC 20036
Telephone 202-328-5000
Fax 202-939-3460

Abstract

This paper discusses EPA's acquisition and use of science in addressing dioxins (and other organochlorines) from the pulp and paper industry under the Clean Water Act and lead in soil at large Superfund mining sites. The common thread between both cases is the challenge posed by administering national pollution control programs while considering site-by-site variability in factors that influence environmental risks. In the first case study, high levels of dioxin in fish downstream of pulp and paper mills were inadvertently detected in 1983 as part of an EPA effort to determine background levels of dioxin in areas presumed to be relatively uncontaminated. These findings quickly got the release of dioxins from pulp and paper mills on EPA's *research* agenda. News reports beginning in 1987 elevated the issue onto the *regulatory* agenda, but more than a decade has passed without EPA taking final regulatory action. Meanwhile, the pulp and paper industry has dramatically reduced, but not eliminated, dioxin discharges from mills. The key scientific issue now confronting EPA decisionmakers is how much weight to give to a water quality indicator called AOX. AOX is not statistically related to dioxin at the levels under consideration. Environmentalists justify using AOX because it serves as a surrogate measure for the entire toxicologically uncharacterized "soup" of organocholorines discharged from bleaching mills. Additionally, EPA estimates that discharges of dioxin from plants at levels below the analytical detection limits will continue to result in exceedances of stringent federal ambient water quality criteria under some local conditions. Industry counters that reductions in AOX do not achieve any measurable or monetizable environmental benefits. This case illustrates EPA's use of science to evaluate the cost-effectiveness of nominally technology-based water pollution controls. In the second case study, the Superfund program does not have the option of following its standard operating procedures for evaluating risks and determining Preliminary Remediation Goals for lead-contaminated sites because EPA has no numerical health-based standard for ingested lead (the agency's goal for lead is based on the level of lead in children's bloodstream). The study, therefore, illuminates the challenges and opportunities posed by developing and using rigorous site-specific scientific information. Potentially Responsible Parties (PRPs) generated rodent bioassay data which suggested that the bioavailability of lead in soil at mining sites would be much lower than EPA's default assumption. However, the agency disputed the validity of using mature rodents as animals models for the population of concern, children. In response, EPA conducted experiments with juvenile swine. The results indicated considerable variability in the bioavailability of lead in soil among the sites tested, with some higher, some lower, and some about the same as the agency's default assumption. Consequently, EPA cannot generalize across sites where similar mining activities occurred or draw any general distinctions between different types of mining sites, as had been presumed. This case illustrates that selection of the most appropriate animal model for toxicological studies involves tradeoffs between cost, experimental power and control, fidelity to human physiology, and the value of information for decisionmaking. Determination of the "optimal" animal model depends on the evaluative criterion being used. Although the new scientific data generated by EPA suggests higher bioavailability of lead in soil at some sites than the agency's default assumption, in terms of the final remedy selection, it appears that all of the results will be either beneficial or essentially neutral to Large Area Lead Site PRPs because EPA deems the cost of removing the contaminated soil to be excessive.

Abstract

Table of Contents

INTRODUCTION

The case studies included in this discussion paper are part of a project that Resources for the Future (RFF) is conducting under a cooperative agreement with the U.S. Environmental Protection Agency (EPA) and with general support from RFF. The case studies were originally vetted as RFF Discussion Paper 97-08 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

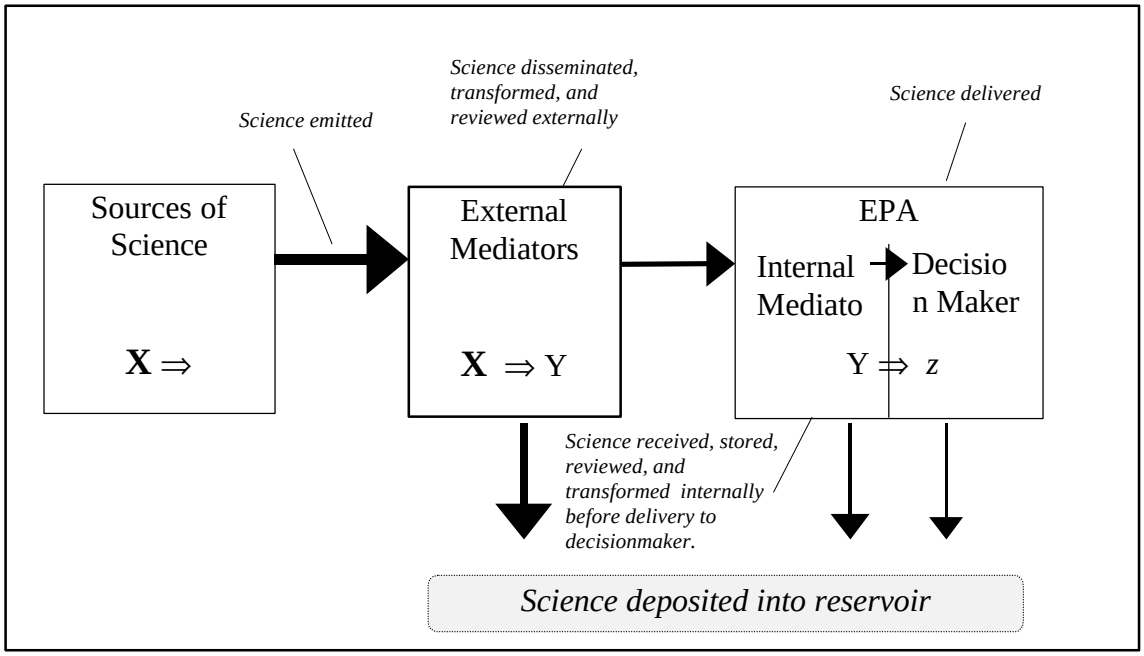
Development of the case studies was based on literature review and interviews with persons inside and outside EPA. The number of interviewees per case study varied roughly from a half dozen to a dozen. There was an effort to ensure balance in the group of respondents for any particular case study, but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

Comments on the case studies should be addressed to:

Mark Powell, Fellow
Center for Risk Management
Resources for the Future
1616 P St., NW
Wash., DC 20036
tel: 202/328-5070
fax: 202/939-3460
email: powell@rff.org

A. Control of Dioxins (and Other Organochlorines) from the Pulp and Paper Industry under the Clean Water Act

1. Background

The term dioxin encompasses a family of organic chemical compounds known as dibenzo-p-dioxins. The dioxins of greatest environmental and public health concern are halogenated dioxins.[1] Because they are the most common, most attention is focused on the group of 75 chlorinated dioxins. Dioxins are not deliberately manufactured, but are a byproduct of combustion, some chemical manufacturing, some bleaching of pulp and paper, and other industrial processes involving chlorine and other halogens. In the U.S., municipal and medical waste incineration are the dominant known sources of dioxin (EPA 1994a), but the total releases of dioxin from all sources (including natural sources such as forest fires) is highly uncertain. Dioxin became notorious in the 1970's when it was identified in the U.S. as "the most potent animal carcinogen ever tested." As one observer phrased it, dioxin earned the reputation as the "Darth Vader" of chemicals (Roberts 1991). In the 1980s, however, Canada and European countries set dioxin limits less stringent than EPA's by two or three orders of magnitude. Officials in these countries concluded that a different cancer model applied to dioxin. More recently, attention has focused on the environmental and non-cancer effects of dioxin and dioxin-like substances that may mimic hormones and act as "endocrine disruptors."

Dioxin discharges into surface waters from pulp and paper mills arose unexpectedly as a regulatory issue more than a decade ago. In 1982, EPA promulgated Clean Water Act (CWA) effluent limitations and technology-based standards ("effluent guidelines") for most of the pulp, paper, and paperboard industry.[2] A year later, as part of the EPA's overall "Dioxin Strategy," the agency initiated a national survey of environmental dioxin levels. In the process of testing what were believed to be "reference streams" to determine background dioxin concentrations in fish in relatively uncontaminated waters, the agency detected surprisingly high levels of dioxin.[3] According to an EPA official, the reference streams where fish had elevated dioxin concentrations had one feature in common, "when you looked upstream, they all had chlorine bleaching

Over time, the list of toxic water pollutants of concern related to chlorine pulp bleaching was broadened to include a variety of more abundant chlorinated organic compounds (organochlorines). These include polychlorinated phenolic compounds, which are considered representative of a various polychlorinated organic materials that may accumulate in food chains, and chloroform, a volatile organic compound. Indicative of the

---

[1] Halogens include chlorine, bromine, iodine, etc.

[2] Effluents are wastewater discharges into surface waters.

[3] For example, dioxin concentrations in fish in a Wisconsin reservoir were more than 50 ppt (parts per trillion), leading the state to close a commercial fishery. Samples in Maine and Minnesota found dioxin concentration in fish of up to 85 ppt (Harrison and Hoberg 1991). By comparison, measured background levels of dioxin in fish are 0-2 ppt (EPA 1994a).

relative magnitude of their production by the U.S. pulp and paper industry, discharges of dioxins and a group of dioxin-like chemicals called furans are measured in terms of grams per year, while the discharges of other organochlorines are expressed in units of metric tons per year. Despite this disparity in the quantity of environmental releases, dioxins and furans have dominated the debate over regulatory controls of the effluents from pulp and paper plants that use chlorine bleaching because chlorinated phenols and volatile organochlorines are estimated to be very much less toxic. Some individuals and groups remained concerned, however, about the heterogeneous soup of organochlorines discharged in bulk from pulp and paper mills because most of these compounds have not been toxicologically analyzed and because the chemical transformations organochlorines undergo in the environment are not fully understood. Staking out a precautionary position in the face of scientific uncertainty, some interested and affected parties argue that all organochlorines should be considered "guilty until proven innocent."

The regulatory control of dioxins, furans, and other organochlorines discharged from pulp and paper mills into surface waters traces its origins back to October 1984, when the Environmental Defense Fund (EDF) and the National Wildlife Federation (NWF) filed a citizen's petition under the Toxic Substances Control Act (TSCA, Sec. 21). The petition requested that EPA regulate dioxins and furans from all known sources.[4] (At the time, despite the questions raised by the detection of dioxin in streams below pulp and paper mills, the bleaching plants were not yet recognized as a source of dioxins and furans.) EPA denied the petition, prompting a 1985 lawsuit by EDF and NWF (*EDF v. Thomas*, DC Dist. Court, Civ. No. 85-0973). Following a series of news reports about EPA's cooperation with industry to investigate the formation and release of dioxins at pulp and paper plants and a 1987 front page story in the *New York Times* regarding the detection of dioxin in household paper products, EPA signed a consent decree with the plaintiffs in 1988. The agreement required EPA to perform a comprehensive risk assessment of dioxins and furans considering sludges, water effluent, and products made from pulp produced at 104 bleaching pulp mills. The agreement also required the agency to propose regulations under TSCA (Sec. 6) to control pulp sludge disposal and under the Clean Water Act to address discharges of dioxins and furans into surface waters from the mills by October 31, 1993 (as amended in 1992). The agency's 1993 proposal to control dioxin and furan releases into surface waters is the primary focus of this case study. The proposal was submitted as a combined set of water effluent limitations and standards and national emission standards for hazardous air pollutants for the pulp, paper, and paperboard industrial sector (also called the proposed "pulp and paper cluster rule," *Fed. Reg.*, Vol. 58, pp. 66078-66216). The pulp and paper cluster rule had not been finalized as of press time. But it appears that the crucial subplot for the effluent limits involves an arcane debate over a Swedish water quality test measure called AOX.

[4] The term furans refers to chlorinated dibenzofurans.

Regulation of Toxic Water Pollutants

The goal of the CWA (also known as the 1972 Federal Water Pollution Control Act (FWPCA) Amendments) is to eliminate entirely discharges of pollutants from point sources (i.e., individual discharging facilities) into surface waters. Although eliminating pollutant discharges may be achievable under some circumstances through process changes that prevent pollutant formation or recycle wastes, the goal is largely rhetorical. The statutory goal of eliminating discharges has potential distorting effects on the use of science because achieving the goal does not require point sources to eliminate all discharges into surface waters. Consequently, attainment depends to some extent on what substances are classified as pollutants subject to regulation under the statute. Furthermore, pursuing discharge elimination from one point source may result in offsetting releases of pollutants. For example, on-site waste recovery to prevent surface water discharges may require extra energy inputs, resulting in additional releases of contaminants to the atmosphere.

The CWA contains both "water-quality based" regulatory controls, which vary according to the designated use (e.g., drinking water source, fishable, swimmable) and attributes (e.g., volume and rate of flow) of the receiving water body, and "technology-based" effluent standards that are achievable using available pollution control technology. Legally, the environmental quality standards dominate the technology-based standards in the sense that additional regulatory action may be required if the technology-based limits do not achieve the ambient quality standard in a specific location. In practice, the technology-based standards are emphasized. This is due in part to the practical difficulties experienced prior to 1972 with state attempts to control surface water pollution. The FWPCA relied on water quality standards which required state regulatory authorities to demonstrate that a given level of pollution was "unreasonable" or "unacceptable" under local environmental and socioeconomic conditions. Under the CWA, Congress has emphasized the approach of the technology-based effluent standards that "do not quibble with judgments of reasonableness" (Fogarty 1991). The emphasis on technology-based standards also avoids the potentially greater time and cost associated with developing, administering, and complying with myriad geographically-specific pollutant discharge limits that must be tailored to meet ambient water quality standards.

Under the 1972 provisions, EPA was to develop a list of national standards for toxic water pollutants that would be applied without regard to industrial source. Implementation of this chemical-by-chemical approach was more difficult than Congress expected, and dissatisfaction with the progress lead to litigation and, eventually, a 1976 consent decree between the Natural Resources Defense Council and EPA.[5] The approach laid out in this settlement was ratified in the 1977 CWA Amendments. Sec. 307 of the CWA now requires Best Available Technology (BAT) economically achievable by industrial sector to limit toxic pollutant effluents from point sources into surface waters. The settlement originally identified a list of 65 "toxic" chemicals and classes of chemicals,

[5] NRDC *et al.* v. Train, 8 ERC 2120 (D.D.C. 1976). Later modified as 12 ERC 1833 (D.D.C. 1979).

which were later subdivided into 129 individual substances or "priority" pollutants (CRS 1993).[6] Dioxin (TCDD - 2,3,7,8-tetrachlorodibenzo-p-dioxin) was originally placed on both lists of toxic pollutants.[7]

The CWA directs EPA to develop BAT for toxic water pollutants "that will result in reasonable further progress toward the national goal of eliminating discharges" (Sec. 301(b)(2)). Factors to be considered in developing BAT for toxic water pollutants include the affordability of achieving effluent reductions ("economic achievability"), engineering criteria, non-water quality environmental impacts, and "such other factors as the Administrator deems appropriate" (Sec. 304(b)(2)). The BAT basis for regulating toxic pollutants is in contrast to the control of "conventional" pollutants (e.g., suspended solids and fecal coliform). Under Sec. 304 of the CWA, conventional pollutant limits are achieved by Best Conventional Pollutant Control Technology (BCT). Determination of BCT depends on the relationship between costs and benefits (essentially a BAT standard moderated by a test of economic reasonableness) (Fogarty 1991). Thus BAT control of toxic pollutants is intended to be less sensitive to cost considerations than BCT, but it acknowledges that alternative technologies can be compared in terms of environmental benefits. (That is, for one technology to be the "best" it must achieve environmental benefits superior to another technology.) Section 307(a) also allows EPA to impose more stringent toxic effluent standards if the BAT standard is inadequate to protect human health with an "ample margin of safety." For some toxic pollutants, however, the only means of providing any margin of safety (ample or otherwise) may be to prohibit discharges altogether because there may be no discernible threshold level of incremental exposure below which no adverse effects will occur.[8]

Through its 1993 proposed pulp and paper effluent regulations, EPA sought to limit the precursors to the formation of dioxins, furans, and other organochlorines in the pulp and paper manufacturing process. The technology-based approach proposed by the agency involves: 1) substituting elemental chlorine with chlorine dioxin or other bleaching agents (e.g., peroxide or ozone) and 2) reducing the extent of chlorine bleaching required to achieve a given quality of product through alternative means of pulp delignification (i.e., extended cooking or oxygen delignification prior to chlorine bleaching). The agency estimates that its proposed effluent limits for the pulp and paper industry would reduce,

---

[6]See 40 *CFR* 401.15 and *Fed. Reg.* Vol. 57, pp. 60911-15 respectively for complete lists. EPA has since reduced the number of priority pollutants to 126. Priority pollutants are carcinogens, suspected carcinogens, or pollutants known to be seriously toxic at low levels. The priority pollutant list originated from a 1975 EPA water toxics regulatory strategy developed in response to the NRDC lawsuit (CRS 1993).

[7] Although furan (TCDF - 2,3,7,8-tetrachlorodibenzofuran) is not explicitly listed as a priority pollutant, EPA treats it as a dioxin-like compound. The International Joint Commission (IJC) has identified TCDD and TCDF as two of eleven "Critical Pollutants" for the Great Lakes (AET 1995).

[8] Toxic effluent standards are pollutant-specific, nationally uniform, and applicable across all categories of industry and all dischargers. By 1976, EPA had promulgated such standards for aldrin/dieldrin, DDT, endrin, toxaphene, benzidine, and polychlorinated biphenyls (PCBs), but stringent procedural and scientific requirements have prevented more extensive development of toxic effluent standards under Sec. 307(a)(2) (Fogarty 1991).

but not eliminate exceedances of health-based water standards for dioxins and furans (see Table A-4 below). However, EPA's ambient water quality criteria are not necessarily the last word. Under the Clean Water Act, EPA and the States share responsibility and authority for: setting risk-based ambient water quality standards; identifying specific segments of water bodies where technology-based pollutant controls may be inadequate to achieve uses designated by the States; and developing strategies for achieving ambient water quality standards in these impaired waters.

Section 303 of the 1987 Clean Water Act Amendments required States to adopt binding numeric criteria for all priority pollutants in cases where discharges could reasonably be expected to interfere with the designated use of water bodies. Congress also authorized EPA to set the criteria if States failed to do so by February 1990 or to develop replacement standards if the agency believes a State's standards do not meet minimum requirements (Copeland 1993; Fogarty 1991). In practice, EPA has permitted the States some discretion in developing their criteria. Under EPA's 1983 revisions to water quality regulations, States retain the right to modify EPA criteria to reflect site-specific conditions or adopt numerical values based on "other scientifically defensible methods" (Executive Enterprises 1984, citing 40 *CFR* 13.11(b)(1)).

In 1990, for example, the State of Maryland proposed a water quality standard for dioxin 10-fold higher than EPA's numeric criteria based on an allowable one in one hundred thousand (10^{-5}) cancer risk. By EPA's reckoning, Maryland's proposed standard suggested a cancer risk (10^{-4}) of potential concern. Acknowledging that there are a variety of equally defensible scientific assumptions that can be made, however, the agency approved Maryland's standard. In the State's proposal, many of the scientific assumptions were the same as those of EPA; where they differed (e.g., the estimated carcinogenic potency of dioxin), Maryland used alternative assumptions employed by the Food and Drug Administration (Moore *et al*. 1993; Thompson and Graham 1997). Thus, the Clean Water Act is unusual among federal environmental statutes in the extent to which EPA and the States share authority to set risk-based public health standards.

Section 304 of the 1987 Clean Water Act Amendments directed States to develop lists of their impaired waters by 1989. Impaired waters are those bodies that do not meet or are not expected to meet ambient water quality standards, even after implementation of technology-based controls implemented by point sources. The States were also required to identify point sources causing the water quality impairments and develop individual control strategies to control those sources further (Copeland 1993). Under the CWA, developing these controls is to be done by setting the total maximum daily load (TMDL), the maximum quantity of a pollutant a water body can receive daily without violating ambient water quality standards under local conditions. The TMDL is then to be allocated among the various sources contributing to the problem. Finally, the National Pollution Discharge Elimination System (NPDES) permits for regulated point sources are to be revised, as warranted.[9] If the States failed to identify a list of impaired waters and develop

[9] Although non-point sources such as runoff from most farms and roadways and atmospheric deposition may contribute to exceedances of ambient water quality criteria, enforceable limits can only be placed on

TMDLs, the 1987 CWA Amendments required EPA to develop a priority list for the State and make its own TMDL determination. In response, EPA mandated that the list of impaired waters include those receiving discharges from pulp and paper mills and called for specific limits on dioxin discharges by 1992 (Thompson and Graham 1997).

In 1990, at the request of Oregon, Washington, and Idaho, EPA established its first TMDL for eight pulp and paper mills discharging into the Columbia River Basin (which includes the Snake and Willamette Rivers). Each of the States had adopted the same ambient water quality standard for dioxin (0.013 parts per quadrillion (ppq)).[10] Based on considerations of regional hydrology, other sources of dioxin, etc., EPA set a TMDL for dioxin (of 5.97 milligrams per day) and allocated 35% of the load to U.S. pulp and paper mills operating in the river basin. Environmental groups sued EPA for not setting a more stringent TMDL, and the pulp and paper mills sued the agency for setting the TMDL before finalizing new effluent guidelines for the entire industry (Thompson and Graham 1997). In 1995, the U.S. Ninth Circuit Court of Appeals upheld EPA's TMDL for dioxin in the Columbia River basin (*Environment Reporter*, 6/30/95, p. 493).

In general, the impaired waters listing process/TMDL program has labored under the Clean Water Act's system of shared EPA-State responsibility. With the CWA requiring EPA to serve as a backstop, state environmental agencies may have little incentive to allocate limited resources to the program and take the heat for controversial decisions. The TMDL program has come under increased fire from environmental groups, tribes, industry, and local communities. A series of recent court decisions citing EPA's failure to complete the tasks after States failed to do so within the statutory time limits could force the agency to make an incredible number of geographically-specific determinations under demanding time, data, and resource constraints. (In the State of Idaho alone, for example, a federal district court has required EPA to set TMDLs for over 900 water segments in a five-year time period (*Inside EPA*, 10/4/96, p. 4).) For EPA and state environmental agencies, the analytically and politically daunting task of setting and allocating innumerable TMDLs makes it all the more appealing to formulate national, technology-based effluent guidelines so as to limit the number of water bodies expected to exceed ambient water quality standards. Environmentalists seek to avoid the cost and delay involved in case-by-case regulation and are wary that States may be reluctant to impose additional controls on firms within their borders. Individual firms or plants also have an interest in assuring that geographically-specific pollution controls do not put them at a competitive disadvantage. Thus, the CWA provisions requiring EPA and the States to consider geographically-specific conditions may influence the use of scientific information in national rulemaking.[11]

---

point sources. Non-point and mobile sources may contribute to background levels of dioxins and other organochlorines that end up in surface waters, sediments, and aquatic organisms.

[10] 1 ppq is 1×10^{-15} .

[11] In 1996, EPA began drafting a strategy to administratively reform the TMDL program and convened a Federal Advisory Committee Act group to develop recommendations. (See *Inside EPA*, 11/22/96, pp. 4-6 for a summary of the agency's draft strategy).

Mirroring the shared regulatory authority between EPA and the States, the CWA is implemented in an environment in which Congress and the Executive Branch continuously wrestle for control over regulatory policy. Spurred by a series of Executive Orders dating back to the Nixon administration requiring some form of economic analysis for proposed regulations (but in particular, the 1981 Reagan administration Executive Order 12291 requiring Office of Management and Budget (OMB) review of new regulations), EPA has deemed it "appropriate" to *consider* cost-effectiveness comparisons when proposing BAT for toxic water pollutants (see discussion of Sec. 304(b)(2) above). According to sources in the EPA Office of Water, however, the program generally regards cost-effectiveness analysis as an imprecise tool that only permits a rough screening of regulatory options, and the agency has not explicitly made any BAT decisions on the basis of cost-effectiveness.

Thus, while the CWA prods EPA to do what is "doable" to reduce toxic water pollution, OMB pulls the agency toward what it thinks is "reasonable." As discussed in greater detail below, the projected benefits of the nominally "technology-based" regulations to limit dioxin and other organochlorines from pulp and paper mills are estimated using the tools of environmental science and risk assessment. Disagreements about the agency's regulatory proposals are often conducted in the language of science and technology and are, in part, over how to properly assess its environmental benefits. The subtext, however, is whether those benefits are reasonably associated with compliance costs.

In addition to its prominent role in the proposed pulp and paper cluster rule, dioxin has a long and highly publicized history. As Finkel (1988) noted, our national preoccupation with dioxin stems largely from the notoriety of TCDD as the most potent animal carcinogen ever tested, and its ubiquity as a contaminant of pesticides, incinerator smoke and ash, and bleached paper consumer products such as diapers and coffee filters. More recently, the dioxin story has segued into the broader debate over "endocrine disruptors," a class of hormone-like chemicals suspected of having a variety of reproductive and other non-cancer effects. Endocrine disruptors are the subject of the much-discussed popular science book entitled, *Our Stolen Future*, which argues that background levels of chlorinated organics and other industrial chemicals may play a role in development of breast cancer, falling sperm counts and other male reproductive disorders, as well as developmental effects in wildlife and humans (Colborn *et al*. 1996).[12]

Forty years ago, a European researcher identified the impurity TCDD as causing the skin disease chloracne in chemical workers involved in the production of the herbicide 2,4,5,-T (Moore *et al*. 1993). But dioxin first came to public light in the early 1970s as a result of concerns about the exposure of Vietnam Veterans and South Vietnamese children

[12] See Hirshfield *et al*. (1996) for a thoughtful review of *Our Stolen Future* and comparison to Rachel Carson's 1962 *Silent Spring*, which publicized the environmental effects of pesticides and is associated with the birth of environmentalism as a mass movement.

to the defoliant Agent Orange (which included 2,4,5,-T).[13] EPA promulgated a partial ban on the herbicide in 1971. The animal studies that resulted in dioxin (TCDD) being labeled as the most potent carcinogen were conducted in 1978. One year later, EPA issued a controversial reanalysis of an epidemiological study conducted in Alsea, OR (Alsea II) which associated miscarriages with herbicide spraying, leading to accusations that EPA had "cooked" the data to inflate the risks (Whelan 1985), and the agency suspended essentially all remaining uses of 2,4,5-T.[14] The problem of dioxin emissions from municipal waste incinerators was identified in 1979 and gained public notoriety as the main plank of Barry Commoner's 1980 presidential campaign platform. (Later, debates over the location and siting of incinerators gave impetus to the environmental justice movement.)

In 1974, the federal Centers for Disease Control (CDC) identified dioxin as a toxic substance in Missouri waste oil. In 1982, EPA detected high dioxin levels from TCDD-contaminated oil sprayed on streets in Times Beach, MO, and dioxin was implicated in illnesses in horses and possibly children. Flooding in December raised concerns about contamination spreading to other sites (though it did not) (OTA 1991). In 1983, the CDC and the Missouri Division of Health recommended that the town be evacuated, and EPA and the Missouri Department of Natural Resources paid $36 million to buy all 801 homes in Times Beach and relocate its residents because of the unavailability of demonstrated treatment technologies and the uncertainty about when the cleanup would be completed. A $200 million cleanup of the town's 400 deserted acres was later initiated. In the summer of 1990, Vernon Houk, head of the Center for Disease Control's Center for Environmental Health and Injury Control, told a congressional committee that new evidence suggested the risk of dioxin historically was vastly overstated.[15]

EPA's first health assessment of dioxin was conducted in 1981 and was revised in 1985. Animal studies by Dow Chemical Co. researchers (Kociba *et al*. 1978) and the National Toxicology Program (NTP 1982) were important sources of scientific information for the agency's assessment. The 1985 assessment is the current *official* basis of dioxin cancer risk estimates used by EPA for all regulatory decisionmaking, including the 1993 proposed pulp and paper cluster rule. However, EPA has been in the process of reassessing the risks of dioxin for several years. During the 1980s, some researchers

---

[13] A 1969 National Cancer Institute study found a link between TCDD and birth defects. According to Smith (1992), other studies by U.S. scientists critical of the Vietnam War also reported teratogenic effects of TCDD. Restrictions on domestic uses of 2,4,5,-T were first announced in 1970 by the Secretary of Agriculture. In promulgating a partial ban on the herbicide in 1971, EPA Administrator William Ruckelshaus rejected the advice of an *ad hoc* scientific panel chaired by Emil Mrak, Chancellor of University of California, Davis, and accepted the counsel of a group Food and Drug Administration (FDA) scientists who had conducted earlier animal tests on 2,4,5,-T. Critics of the Mrak panel had received leaked copies of the report prior to its release. Both advisory groups were informally convened prior to the advent of the 1972 Federal Advisory Committee Act (FACA), and the episode crystallized support for FACA (Smith 1992, p. 24-25).

[14] See Jasanoff 1990, pp. 24-26 for a more balanced discussion of the 2,4,5-T controversy.

[15] Two sources interviewed for the overall study of science in environmental regulation volunteered the Times Beach buy-out as an EPA decision in which science played little or no role.

postulated that dioxin might "promote" rather than "initiate" cancer and that, as a result, EPA may have overestimated the cancer risks from dioxin. With the backing of Assistant Administrator for Pesticides and Toxics John Moore, an *ad hoc* EPA committee in 1986 recommended moderating the dioxin cancer risk estimate. At about the same time, OMB highlighted the large scientific uncertainty of dioxin cancer risk estimates in its annual report on federal regulatory programs (Roberts 1991; Moore *et al*. 1993). The following year, EPA issued a draft reassessment suggesting that the risk of cancer from dioxin was 17 times less than the agency had assumed. According to Finkel (1988), however, the agency developed its revised estimate not on the basis of any new data, but by essentially splitting the difference between two "fundamentally irreconcilable theories about the carcinogenicity of dioxin." Regardless of whether the decision was "right for the wrong reasons," as some felt, the agency's approach could not withstand scrutiny. In reviewing the agency's draft, the EPA Science Advisory Board (SAB) criticized EPA's current cancer risk assessment methodology but found no new data to support changing the dioxin cancer risk estimate (Moore *et al*. 1993).

In 1990, Robert Scheuplein of the Food and Drug Administration (FDA), toxicologist Michael Gallo of the Robert Wood Johnson Medical School in New Jersey, and Dutch scientists organized the "Banbury Conference," (held at New York's Cold Spring Harbor Lab) which formally marked a new scientific consensus about a series of biological steps occurring at the molecular level that precede most if not all of the observed effects of dioxin and other similar chemicals (Roberts 1991).[16] Some scientists interpreted this to mean that the very low levels of dioxin in the environment would result in negligible cancer risks. In 1991, an epidemiological study conducted by National Institute of Occupational Safety and Health (NIOSH) researchers (Fingerhut 1991) reported a statistically significant increased cancer risk in U.S. chemical workers exposed to high levels of dioxin but detected no increase in workers exposed to low levels. As a result of the Banbury Conference and the NIOSH study, external pressures mounted for EPA to move beyond research and initiate a formal reassessment of dioxin. According to press reports, the paper industry was a leading voice in persuading the agency to revisit dioxin (*Rachel's Environment & Health Weekly*, 8/31/95, p. 1). In April 1991, EPA Administrator William Reilly announced that the agency would comprehensively reassess the cancer and non-cancer risks of exposure to TCDD and related compounds.

While EPA slightly moderated the cancer risk estimate for dioxin and similar compounds in its *draft* reassessment released in 1994, it also concluded that there was potential for a variety of adverse non-cancer effects in the range of current background exposures to dioxin and similar compounds (EPA 1994b). In reviewing the draft reassessment, a majority of SAB members concluded that agency tends to overstate the possibility for danger at near-ambient levels, but several SAB members regard the agency's characterization of the risks as appropriately conservative within the context of

[16] Consensus broke down, however, on just what such a biologically-based model would predict in terms of dioxin's cancer risks (Roberts 1991). See Powell (1996) for a discussion of Gallo's role in promoting EPA's use of biologically-based risk assessment models. According to a former senior EPA official, industry, notably the Chlorine Institute, played a role in initiating the Banbury Conference.

public health protection (EPA/SAB 1995). An environmentalist now says, "Reilly's decision to conduct the dioxin reassessment did not turn out the way he dreamed it would. Industry and he thought they would have a slam dunk on dioxin's carcinogenicity."

Although it appeared likely to many in 1988 when EPA began to formulate the new pulp and paper effluent limits that our "national preoccupation" with dioxin would wane, the agency's subsequent dioxin reassessment has highlighted the non-cancer effects of dioxins and helped launch the issue of endocrine disruptors onto the environmental regulatory agenda. Some environmentalist groups (notably Greenpeace) have responded by calling for a ban on chlorine. This proposal was afforded a measure of mainstream legitimacy in February 1994 when the International Joint Commission (IJC), the Canadian-American bilateral organization established to monitor the Great Lakes Water Quality Agreement, recommended phasing out the use of chlorine and chlorine-containing compounds as industrial feedstocks. It is in this context that EPA will try to finalize the effluent regulations for the pulp and paper industry. Table A-1 provides a summary background of dioxin science and policy. Table A-2 summarizes the development of the pulp and paper cluster rule.

Table A-1. Summary Background of Dioxin Science and Policy

1949	USDA registers 2,4,5-T as a pesticide.
1957	TCDD identified as causing chloracne.
1966	USDA and FDA establish residue tolerances for 2,4,5-T in food.
1969	Initial laboratory studies link 2,4,5-T and TCDD with birth defects.
1970	U.S. halts use of Agent Orange in Vietnam.
1971	EPA restricts domestic use of 2,4,5-T.
1972	Controversy over EPA's 2,4,5-T decisionmaking process crystallizes congressional support for Federal Advisory Commission Act.
1974	CDC identifies dioxin as toxic substance in Missouri waste oil.
1976	Industrial accident releases large quantities of dioxin in Sveso, Italy.
1977	Clean Air Act Amendments list dioxins and furans as hazardous air pollutants.
1978	First EPA study regarding linkage between miscarriages and herbicides in Alsea, OR. Dow Chemical Co. researchers report that TCDD is a carcinogen in laboratory studies.
1979	Alsea II reevaluates miscarriage-herbicide data. EPA accused of inflating risks. EPA suspends use of 2,4,5,-T. Vietnam veterans start class action suit. Dioxin and furans identified in emissions from municipal waste combustion plants.
1980	Barry Commoner's presidential campaign elevates concerns about dioxin releases from waste incinerators.
1981	Sveso 5-year report finds no dioxin effects other than chloracne. EPA's Cancer Assessment Group estimates that dioxin is one of the most potent carcinogens known.
1982	National Toxicology Program reports results of dioxin animal cancer study.
1983	Times Beach, MO buyout. EPA issues congressionally-mandated national strategy to investigate, identify, and remediate dioxin contaminated areas.
1984	EPA cancels 2,4,5-T registration. Hazardous Solid Waste Act requires EPA to evaluate risks posed by dioxin emissions from municipal waste combustion facilities.

Table A-1. Summary Background of Dioxin Science and Policy (cont'd)

1985	EPA revises its dioxin health assessment, lowering the cancer risk estimate by more than a factor of 2, but retains the agency's default linear cancer model.
1986	*Ad hoc* expert committee advises EPA that linear cancer model is inappropriate for dioxin.
1987	EPA scientific group recommends moderating cancer risk estimate. EPA develops Toxic Equivalency Factors (TEFs) for dioxin and dioxin-like chemicals.
1989	EPA SAB finds no new data to support change in cancer risk estimate; critical of current cancer model; accepts TEFs as an interim approach.
1990	Banbury Conference supports receptor-mediated event for dioxin activity. EPA promulgates New Source Performance Standards for municipal waste combustion facilities requiring best management practices to limit total dioxins and furans to 30 ng/m^3.
1991	NIOSH epidemiological study suggests that dioxin is a human carcinogen, but perhaps only at high levels of exposure. EPA initiates dioxin reassessment.
1994	EPA draft dioxin reassessment reports potential for adverse non-cancer health effects within the range of current background levels. Chlorine ban proposed by Henry Waxman (D-CA), Barry Commoner, and others (*Environment Reporter*, 9/30/94, p. 1133).

Table A-2. Development of the Pulp and Paper Cluster Rule.

1983	EPA initiates national dioxin survey, detects elevated dioxins downstream from pulp and paper mills.
1984	EDF and NWF file TSCA petition requesting EPA to regulate dioxins and furans from all known sources. EPA denies petition. EPA issues Ambient Water Quality Criteria report for dioxin.
1985	EDF and NWF file lawsuit.
1986	June. EPA, NCASI, and American Paper Institute (API) agree to undertake the "5 Mills Study," detect TCDD and TCDF in effluents, pulp and sludges of pulp and paper mills. December. Information on the agreement between EPA and the pulp and paper industry reported. Greenpeace initiates Freedom of Information Act (FOIA) request seeking all available information on the pulp mill dioxin problem.
1987	Clean Water Act Amendments establish deadlines for EPA and States to address toxic pollutants. January. Letter from EPA to API leaked to environmentalists indicates EPA officials had agreed to notify the industry "immediately" of receipt of any requests under FOIA and that, barring such requests or results indicating a potential threat to human health, the agency did not intend to release any results until publication of the final report on the study. August. Greenpeace USA releases report alleging an EPA cover-up. September. *New York Times* front-page story reports traces of dioxin detected in household paper products. Report based on the "5 Mills Study" and analyses of dioxin in paper products.
1988	EDF, NWF and EPA sign consent decree requiring agency to perform a comprehensive risk assessment of dioxins and furans considering sludges, water effluent, and products made from pulp produced at 104 bleaching pulp mills and (as amended in 1992) to propose regulations addressing discharges of dioxins and furans into surface waters from the mills by October 31, 1993. EPA issues "interim strategy" to address dioxin emissions from pulp mills, which included requiring pulp mills to monitor for dioxins and adopt short-term control measures (Hanmer 1988; EPA-V 1988). EPA and industry begin the "104 Mill study."

Table A-2. Development of the Pulp and Paper Cluster Rule (cont'd).

1988	Swedish studies generate adsorbable organic halides (AOX) indicator used in EPA's 1993 proposed effluent limits.
1989	EPA initiates inter-agency, inter-office assessment of pulp and paper sludges, effluents, and consumer products. OTA report discusses Swedish pulp mills' compliance with more stringent regulatory standards for organochlorine emissions (OTA 1989). March and June. First results of "104 Mill study," released.
1990	EPA issues Assessment of Risks from Exposure of Humans, Terrestrial and Avian Wildlife, and Aquatic Life to Dioxins and Furans from Disposal and Use of Sludge from Bleached Kraft and Sulfite Pulp and Paper Mills. Based on 104 Mill study, assessment estimates that preventing adverse wildlife effects would require TCDD soil concentrations 4-400 times lower than levels needed to prevent unacceptable human health risks.
1991	May. Under court consent decree, EPA proposes pulp and paper mill sludge rule under TSCA Sec. 6. Proposal would set a 10 ppt maximum allowable dioxins/furans concentration for land application (resulting in an estimated human health risk less than 10^{-4}) and includes provisions for mills to submit annual reports and maintain records on land, application, and laboratory analysis. July. OMB objects to proposal's information collection request (*Environment Reporter*, 8/16/91, p. 1058).
1992	EPA announces it would seek a voluntary agreement with industry on the pulp and paper mill sludge rule (*Environment Reporter*, 12/24/93, pp. 1545-1546).
1993	September. NRDC and 55 other environmental groups petition under CWA Sec. 307 (a) for EPA to ban dioxin discharges by the pulp and paper industry by prohibiting the use of chlorine rather than manage dioxin through BAT standards under pulp and paper cluster rule (*Environment Reporter*, 9/17/93, pp. 889-890). December. EPA proposes pulp and paper cluster rule based on BAT standards.
1994	February. At hearing on proposed cluster rule, industry representatives claim that EPA's environmental benefits analysis does not employ sound science and overstates benefits. Future EPA Assistant Administrator for Research and Development Robert Huggett reports that substitution of chlorine dioxide for elemental chlorine reduces chemicals that accumulate in fatty tissues to the limits of detectability (*Environment Reporter*, 2/18/94, pp. 1783-1784). April. EPA and pulp and paper industry announce voluntary agreement regarding land disposal of dioxin-tainted sludge formalizing best management practices. No restrictions on use of sludges if concentration of dioxin and furan is less than 10 ppt. For pasture lands, the concentration limit is 1 ppt (i.e., background levels). At 50 ppt, sludge cannot be land applied.
1996	On the basis of new data regarding the environmental performance of pulp and paper mills that have completely substituted chlorine dioxide for elemental chlorine, EPA announces that it is considering two BAT options for the major pulp and paper subcategory (bleached paper papergrade kraft and soda).

2. Scientific Issues

Dioxin and Related Compounds

The major scientific controversy over dioxin and its chemical cousins is not whether high levels of exposure can cause cancer in humans but rather the risks posed by

background levels and incremental releases of all dioxin-like compounds. Although dioxins and other organochlorines have been associated with a variety of non-cancer effects, the conventional focus of scientific investigation has been on cancer. According to Lucier *et al*. (1993), several long-term bioassays have been conducted on TCDD in several species. All studies have produced positive results. It is clear the TCDD is a multisite carcinogen in both sexes of rats and mice. It also is a carcinogen in the hamster, which is considered the most resistant species to the acute toxic effects of TCDD. TCDD is also found to increase cancer incidence in animals at doses well below the Maximum Tolerated Dose. While TCDD appears to be a chemical that strongly promotes cancer development once initiated, it seems to have weak or no potential to initiate cancer itself. The general consensus is that TCDD is an example of a carcinogen whose action is mediated by a specific receptor within cells, suggesting that there may be a threshold dose below which dioxin is not carcinogenic. It may be possible, however, that non-cancer health effects result at levels below the threshold dose for cancer.

A considerable body of studies of people exposed to dioxin provides suggestive evidence of its human carcinogenicity, but according to an EPA official, the epidemiological evidence is inconclusive due to a number of factors. First, scientists cannot be certain about how much dioxin and other chemicals the subjects were exposed to. Second, in most studies, the numbers of people exposed through accidents or in the workplace have been too small to allow scientists to detect substantial changes in cancer rates. Third, those individual who were exposed to dioxin (mostly healthy adult males) may not have been the most sensitive group. Finally, not enough time may have elapsed between exposures and study completion for most cancers to develop (many cancers only develop 15-30 years after exposure). The first dioxin epidemiological study sufficiently large enough to detect a substantial increase in cancer doses, according to this EPA official, was Fingerhut *et al*. (1991). This NIOSH study, which took nearly 13 years to complete and examined 5172 male U.S. chemical workers exposed to dioxin on the job from 1942-84 presented what many consider the strongest evidence that dioxin is a human carcinogen--but perhaps only at very high doses (Roberts 1991). The EPA Science Advisory Board has agreed that although human data are limited, dioxin is a probable human carcinogen under some exposure conditions (EPA/SAB 1995). In February 1997, an International Agency for Research on Cancer (IARC) Working Group also concluded that TCDD should be considered carcinogenic to humans (http://www.iarc.fr/preleases/115e.htm).

Extrapolating from rodent studies using a linear model of cancer risk, EPA's Cancer Assessment Group derived an extraordinarily high cancer potency factor (4.25×10^5 $(mg/kg/day)^{-1}$) for dioxin in 1981. An important basis of this estimate was a reanalysis of the pathological evidence from the Dow Chemical researchers' rat study (Kociba *et al*. 1978) performed by Robert Squire of Johns Hopkins University Medical School.[17]

[17] Pathology includes laboratory analysis of animal tissue slides to characterize and enumerate abnormalities such as tumors. It is traditionally descriptive and can be fairly imprecise, but standardized protocols and quantitative and chemical techniques have been developed to promote consistency and precision.

Squires' reinterpretation of the tissue samples resulted in a cancer potency factor approximately two times higher than the one derived using the original diagnoses. In 1985, EPA revised its dioxin cancer potency estimate downward by more than a factor of two (to 1.56×10^{-5} $(mg/kg/day)^{-1}$) by adjusting for the early mortality of study animals observed in Kociba *et al*. (1978) and by essentially splitting the difference (taking the geometric mean) between the original pathology assessment and Squires' reanalysis (Thompson and Graham 1997). The agency had moderated its dioxin hazard assessment somewhat, but it still estimated that a one in a million (10^{-6}) cancer risk was associated with exposure to the infinitesimally small quantity of 0.006 pg/kg/day (picograms (10^{-12} g) per kilogram body weight per day).

Although EPA indicated in 1985 that there was inconclusive evidence that dioxin was a mutagen (able to initiate carcinogenisis), the agency determined that the available data on dioxin's biological activity (carcinogenic mechanism and pharmacokinetics) were insufficient to support deviation from the default linear dose-response model for cancer. Canada and European countries, however, rejected the linear cancer model as inappropriate for dioxin because it is not considered genotoxic (i.e., dioxin does not directly initiate cancer by causing mutation or DNA damage), and set their limits at 1-10 pg/kg/day. There were also differences in dioxin cancer potency estimates within the U.S. government between EPA, CDC, and FDA. FDA's cancer potency estimate is almost an order of magnitude smaller than EPA's 1985 estimate, and CDC's is intermediate between the two. The inconsistent estimates resulted from the agencies applying the same linear cancer model but making a variety of different scientific assumptions and data treatments.[18]

In the 1970s, Alan Poland of the University of Wisconsin initiated the first studies on dioxin's biological mechanisms (Thompson and Graham 1997). At the 1990 Banbury Conference, scientists agreed that the biological activity of dioxin and dioxin-like compounds was mediated by first binding to a specific molecular receptor in cells, the aryl hydrocarbon (Ah) receptor (an intracellular protein).[19] Theoretically, dioxin molecules may have to occupy many Ah receptors sites before any biological response is seen, and even once activity begins, the cell's internal regulation system has some capacity to adapt to changing hormonal levels and maintain the mix within the range of tolerance. In the view of some scientists, this theoretical argument suggests a threshold below which dioxin cannot cause cancer and implies that EPA's linear cancer model is invalid for dioxin. "If

[18] The agencies' estimation procedures differed in how to extrapolate from rat to man (body weight or surface area); which pathology results were used (Kociba and colleagues', Squire's, or both); whether early mortality was taken into account; the assumed average human body weight (80 kg or 70 kg); and how the dose was measured (concentration in the tissue or administered dose) (Thompson and Graham 1997). Using surface area to scale the administered dose between animals and humans leads to a higher potency estimate than does using body weight as a scaling factor. Currently, EPA uses a scaling factor of body weight raised to the 2/3 power. According to an academic, there is a proposal for all federal agencies to adopt a scaling factor of body weight raised to the 3/4 power, but FDA continues to scale on the basis of body weight.

[19] In 1995, the EPA Science Advisory Board reported that it was also possible that dioxin may produce toxic responses that are not mediated through the Ah receptor (Thompson and Graham 1997).

we can't do it [depart from the linear default model] for dioxin, for which we have so much information, then we probably can't do it for anything," said Banbury Conference organizer Robert Gallo (quoted in Roberts 1991).[20]

However, there may be considerable variability among individuals in the threshold level at which carcinogenesis begins. In addition, a continuum of biological activity occurs beginning at relatively low levels of Ah receptor occupancy. There is, however, considerable controversy regarding the health *significance* of the activities initiated at lower levels of occupancy. (In practical terms, this means that setting dioxin limits low enough to prevent cancer may be insufficient to prevent other biological effects, but the "so what?" question has yet to be resolved by scientific consensus.) In response to the 1991 decision to conduct a dioxin reassessment, scientists at EPA's Office of Research and Development and the National Institute of Environmental Health and Safety (NIEHS) began research to characterize a threshold for dioxin in humans. The results, reported in 1992-93, suggested that enzyme induction occurs at existing background levels of dioxin-like compounds (Thompson and Graham 1997). Instead of cancer being initiated at the lowest dose levels, it is now hypothesized that reproductive, developmental, and immune-system impairments may be the most sensitive health effects of dioxin. For these non-cancer effects, says an environmentalist, the old toxicological adage that "the dose makes [21] Instead, the timing--not the quantity--of exposure may be the critical factor. This source is concerned, for example, that exposure to a trace quantity of dioxin that might be irrelevant in terms of cancer risk could result in a substantial developmental risk if maternal exposure occurs at a critical period of fetal development.

Further, the biological system responds to the cumulative exposure of dioxin and similar chemicals that bind to the Ah receptor rather than to the exposure to any single dioxin-like compound. As a result, much disagreement now centers on just how close existing background levels of all dioxin-like compounds occurring in the environment and stored in human tissues are to the levels required to cause adverse health effects.[22] As Thompson and Graham (1997) suggest, the significance of this dispute is that the concept of a threshold level of Ah receptor occupancy may be irrelevant to decisions about additional releases of dioxin-like compounds if typical body burdens already exceed the threshold.

Of the group of 75 chlorinated dioxins, only TCDD has been subjected to long-term animal carcinogen experiments. To account for the cumulative exposure to compounds that, like dioxin, would bind to the Ah receptor, in 1987, the EPA Risk Assessment Forum developed Toxic Equivalency Factors (TEFs). These TEFs derive from a relative ranking scheme based on assigning a TEF of 1.0 to TCDD, since it shows the greatest affinity for binding to the Ah receptor. Other dioxin-like compounds are

---

[20] Similar statements have been made regarding departure from the linear model for ingested arsenic. See Powell (1996).

[21] This means that too much of anything--even something essential to life in normal doses--can be harmful.

[22] Background levels would include accumulations of both natural and anthropogenic sources.

assigned a fractional weight proportional to their binding affinity relative to that of TCDD. The TEFs are intended to be additive weighting factors. The TEF for TCDF, for example, is 0.1--its affinity for binding to the Ah receptor is 1/10th that of TCDD (EPA 1989). (Thus 5 g of TCDD plus 5 g of TCDF yields the estimated equivalent of 5.5 g of TCDD.) There is not a perfect correlation, however, between Ah receptor binding affinity and the potency for various toxic effects. Consequently, there is considerable uncertainty about how accurately TEF equivalent weights reflect cumulative effective exposures.[23]

As indicated earlier, dioxins are produced in very small quantities. EPA (1994a) estimates annual emissions from known sources for the entire U.S. at 3,300 - 26,000 *grams*, with the total possibly being as high as 50,000 g/yr.[24] However, dioxins are extremely insoluble in water, environmentally and biologically stable, persist in the environment for long periods, and tend to accumulate in animal tissues. Thus, the predominant route of human exposure is probably through the food chain rather than inhalation or drinking water. Relative to other foods, measurements of background levels of dioxin are particularly high in fish. Currently, bleaching pulp and paper mills are the only significant known source of dioxins released into surface waters (EPA 1994a). According to an EPA official, the agency estimated a very wide range of risks resulting from dioxin and furan released from pulp and paper mills, much of which was explained by the size of the receiving water body into which plant effluent was being discharged.

Formation of Dioxin and other Organochlorines from Bleaching Pulp

Lignin is a natural polymer that binds and supports cellulose fibers of woody plants, but it discolors and weakens paper products. Chemical pulping dissolves a large fraction of lignin using nonoxidizing chemicals (e.g., alkalis or sulfites) while preserving a large fraction of the desired cellulose fibers. Various forms of chlorine and other bleaching agents are used to further remove lignin from pulp to produce durable white paper products (like this page). For many decades, elemental chlorine (Cl_2) has been the bleaching agent of choice for much of the U.S. pulp and paper industry due to its relatively low cost. Chlorine dioxide (ClO_2) is more selective for lignin and thus can achieve the same level of pulp bleaching with a substantially lower input or "charge" of chlorine, but it costs more than elemental chlorine. Using a process called oxygen delignification (OD), oxygen may also be used as an initial bleaching agent to reduce the chlorine charge

[23] According to a former Science Advisory Board member, the Environmental Defense Fund encouraged EPA to develop the TEF scheme. When the Board reviewed the scheme in the late 1980s, says this source, "The SAB said, 'We'll accept that as an interim procedure, but more research is needed to substitute for TEFs.' Now the TEFs are getting locked in, and the research wasn't done. People get used to using the old numbers, and they take on a life of their own. There's a 'check the box' mentality, a resistance to revisiting old decisions. Risk assessment needs to be an iterative process."

[24] These figures are for all dioxin-like compounds weighted by toxic equivalency factors, but they are dominated by TCDD (about 90% of the total).

required to achieve a given level of pulp brightness. However, OD is a capital-intensive technology.[25]

When dioxin was first detected in streams below pulp and paper mills, the first culprits identified were oily defoamers and woodchips treated with polychlorophenols. Addressing these sources, however, did not eliminate dioxin formation from bleaching pulp and paper mills. This suggests that some dioxin and furan precursors might occur in trees naturally (Berry *et al*. 1991). It now appears that the only way to entirely prevent formation of dioxins, furans, and other organochlorines by the pulp and paper industry is to eliminate the use of chlorine as a bleaching agent. By substituting the more lignin-selective ClO_2 for elemental chlorine, however, the formation of organochlorines--and particularly the persistent, bioaccumulable polychlorinated organics or greatest concern--can be dramatically reduced.

According to Berry *et al*. (1991), of the chlorine used in pulp bleaching, about 90% ends up as common salt (e.g., calcium chloride) and about 10% binds to organic material removed from the pulp. About 80% of this organically bound chlorine occurs in high-molecular weight material that does not permeate cell walls and is relatively water soluble.[26] Most of the organically bound chlorine which occurs in low-molecular weight compounds that can permeate cell walls is relatively water soluble and is readily hydrolyzed or metabolized. A small fraction (about 1%) of the total organically bound chlorine is relatively fat soluble and potentially bioaccumulable and toxic. A component of particular concern in this fraction is the polychlorinated organic material, which includes dioxin, furan, and polychlorinated phenolic compounds. The polychlorinated phenolic compounds, however, are considered much less toxic than dioxin. For example, EPA estimates the cancer potency of 2,4,6-Trichlorophenol to be seven orders of magnitude lower than that of TCDD (EPA 1993a, Table 3-1).

Because chlorine atoms are added to organic precursors in a largely sequential process (with the di-chlorinated organics most likely to be formed before tri-chlorinated organic, tri-chlorinated organics most likely to be formed before tetra-chlorinated organics, etc.), Berry *et al*. (1991) concluded that a threshold level of chlorine charge would be required for any TCDD and TCDF formation to occur. They further suggested that 100 percent substitution of ClO_2 for Cl_2 (called "complete substitution") could prevent such formation. However, more recent data from mills employing complete substitution show detectable levels of TCDD and TCDF in bleach plant effluents (ERG 1996). Given the huge number of randomly interacting molecules present in commercial-

[25] Because oxygen is relatively unselective for lignin, OD results in more dissolved organic material. Extended cooking has a similar effect. Consequently, pulp and paper mills using these delignification technologies require more recovery boiler capacity than those mills that do not.

[26] Berry *et al*. (1991) surmise that it is highly improbable that the high-molecular weight chlorinated lignin material would be broken down and transformed in the environment into problematic, polychlorinated compounds because the potentially troublesome aromatic (6-carbon ring) structure of the residual lignin would largely be destroyed by oxidation in the bleach plant. Berry *et al*. (1991) add, however, that further investigation of the environmental fate of this fraction of the organochlorines is needed to confirm that neither it nor its decomposition products are harmful.

scale pulp bleaching, one would expect some trace (perhaps undetectable) amounts of tri-chlorinated organics (such as trichlorophenol) and tetra-chlorinated organics (such as TCDD and TCDF) to be formed at even the lowest chlorine charges, particularly if the pulp and chlorine are not uniformly mixed.[27] Thus, complete substitution of ClO_2 for elemental chlorine would not entirely eliminate dioxin and furan formation. Complete substitution does appear, however, to reduce dioxin and furan formation to the flat portion of the curve, well beyond the point of diminishing returns. (See the data presented in Berry *et al*. 1991.)

Berry *et al*. (1991) also observed that the formation of dioxin and furan is little affected by the lignin content of unbleached pulp. This conclusion has been reinforced by the more recent environmental performance data. TCDD and TCDF were not detected in any industry-supplied sample results from bleached papergrade kraft mills employing complete substitution (of Cl_2 with ClO_2). But TCDD and TCDF were detected in EPA-collected samples at several mills using both complete substitution *and* oxygen delignification (ERG 1996). Therefore, while complete substitution may not entirely preclude dioxin formation, initiating the bleaching process with OD (and thereby further reducing the required chlorine charge) does not appear to prevent it either. In contrast, the lower lignin content of pulp prior to bleaching plays a decisive role in the reduced formation of the less toxic but more abundantly formed chlorinated phenolics (Berry *et al*. 1991).

Releases and Detection of Dioxin and other Organochlorines from Bleaching Pulp

For contaminants like dioxin that are toxic at trace concentrations, damages may occur at environmental levels resulting from the cumulative releases of multiple sources which, when considered individually, may discharge undetectably low concentrations of the pollutant. EPA's 1993 proposed BAT for pulp and paper effluents was expected to yield non-detectable concentrations of TCDD for two subsectors of the industry and of pentachlorophenol for three subsectors of the industry (Table A-3). However, assuming human consumption of both water and organisms, the agency estimated that the proposed effluent limits would reduce, but not eliminate exceedances of the most stringent federal health-based ambient water quality criteria (AWQCs) for dioxin, furan, and other chlorinated organic priority pollutants (Table A-4).[28] Comparing modeled dioxin fish

[27] Berry *et al*. (1991) noted that thorough mixing and good process controls would be essential to ensure that no portions of the pulp are exposed to higher than the minimum chlorine charge.

[28] The 1993 water quality assessment of the proposed pulp and paper effluent guidelines cites the AWQC for TCDD for consumption of water and fish as 1.3×10^{-9} µg/L (equivalent to 0.0013 ppq (EPA 1993a, Table 3-1). In its 1984 water quality criteria report for dioxin, EPA recommended ambient levels of dioxin in the 10^{-5} - 10^{-7} cancer risk range, with 1.3×10^{-9} µg/L corresponding to EPA's estimated risk level of 1×10^{-7} (EPA 1984, p. xi). Thus, the agency's 1993 assessment based its estimates of AWQC exceedances for the proposed pulp and paper effluent guidelines on non-binding federal ambient criteria at the lowest end of the recommended range. As noted above, however, EPA has approved binding, numeric state ambient water quality criteria for dioxin that are even less stringent than the range recommended in 1984. EPA (1993a, Table 3-1) also cites an AWQC for TCDF for consumption of water and fish as 8.10×10^{-8} µg/L (0.081 ppq). According to an EPA water program official, however, the agency has no official

tissue concentrations with the various advisory action levels adopted by States, EPA (1993a) estimates that the BAT proposed in 1993 would substantially reduce (by 70-95%), but not eliminate the number of State dioxin-related fish advisories in place.

Table A-3. Effluent limits (maximum for any 1 day) for existing plants using proposed BAT process.

Subsector	TCDD (ng/kkg)	TCDF (ng/kkg)	Pentachlorophenol	AOX (kg/kkg)
Bleached papergrade kraft and soda	ND	359	ND	0.267
Dissolving sulfite	ND	1,870	ND	3.13
Dissolving kraft	300	415	ND	0.65
Papergrade sulfite	N/A	N/A	N/A	0.1

ng/kkg - nanograms per metric ton (1 ng = 10^{-9}g; 1 metric ton = 10^{6}g)
kg/kkg - kilograms per metric ton (1 kg = 10^{3}g, 1 metric ton = 1000 kg, or about 2200 lbs.)
ND - No detection limits of the analytical methods for TCDD and TCDF are 10 pg/L (pg = 10^{-12}g), or 10 ppq.[29]
AOX - adsorbable organic halides (see discussion immediately below)
Source: *Fed. Reg.*, Vol. 58, pp. 66078-66216.

Because dioxin and other organochlorines may be toxic in trace amounts, EPA proposed to establish effluent limitations for these pollutants measured at the bleach plant within the mill rather than at the end of the pipe. This permits greater detection of these pollutants before they are diluted in down-stream milling processes or wastewater treatment. Given a large number of samples in which toxic pollutants are not detected, their estimated concentration in the pulp mill bleach plant effluent will be sensitive to how the "no-detect" measurements are treated. (The "no-detects" signify that the actual concentration lies somewhere between zero and the analytical detection limits.) Consistent with the agency's standard procedures, EPA analyzed TCDD and TCDF sample data from bleach plant effluents assuming one-half detection limit values for those contaminants not detected in the effluent. The agency noted that a "significant portion of [the estimated] risk is associated with the use of one-half the EPA designated detection limit [5 pg/l] for these" pollutants (EPA 1993a). Any particular value (or point estimate) one could apply to the non-detect samples could be regarded as arbitrary. A probabilistic approach would employ a distribution of values ranging from zero to the detection limit.

---

ambient water quality criterion for TCDF. It appears that the AWQC for TCDF has been inferred from the AWQC for TCDD on the basis of a TEF (i.e., 0.1) and a different estimated bioconcentration factor (BCF). (See EPA 1993a, p. 20. The BCF is used to estimate the concentration of a substance in fish tissue based on its concentration in water). EPA (1993a, Attachment A-12) also estimates that the proposed BAT would result in no remaining exceedances of the AWQCs for pollutants other than TCDD or TCDF if human consumption is assumed to be limited to fish and not to include drinking water.

[29] An 70 kg (154 lb.) person drinking 2 liters of water per day containing 10 pg/L would receive a dose of 0.29 pg/kg/day. This figure is more than an order of magnitude (over forty-fold) higher than EPA's 1985 one in a million cancer risk-specific dose of 0.006 pg/kg/day.

This approach is conceptually preferable to the simpler point-estimate approach, but it remains unclear how to ascertain the precise form of the distribution of pollutant concentrations below the analytical detection limits.

Table A-4. Estimated AWQC Exceedances - Number of Streams Below Plants Exceeding AWQC by Industry Subsector

Subsector	Priority Pollutant	Baseline	Proposed BAT
Bleached papergrade kraft and soda	TCDD	80	71
	TCDF	59	15
	Chloroform	24	0
	Pentachlorophenol	19	2
	2,4,6-Trichlorophenol	5	0
Dissolving sulfite	TCDD	5	5
	TCDF	4	3
	Chloroform	3	2
	Pentachlorophenol	3	1
Dissolving kraft	TCDD	3	2
	TCDF	1	1
	Chloroform	1	0
	Pentachlorophenol	1	1
	2,4,6-Trichlorophenol	1	0
Papergrade sulfite	TCDD	9	0
	TCDF	5	0
	Chloroform	1	0
	Pentachlorophenol	1	0

Baseline - estimated current AWQC exceedances.
Proposed BAT - estimated AWQC exceedances after implementation of proposed BAT.
[a] In addition to TCDD and TCDF, of the organochlorines listed, chloroform, pentachlorophenol, and 2,4,6-trichlorophenol are regulated as priority pollutants.
Sources: *Fed. Reg.*, Vol. 58, pp. 66078-66216; EPA (1993a, Attachment A-12).

Alternatively, all chlorinated organic compounds in an effluent sample can be measured collectively at the end of the pipe using an indicator such as the concentration of adsorbable organic halides (AOX). AOX is a test measure used by Swedish researchers in studies conducted between 1977 and 1985 to evaluate some dramatic effects on fish populations located near bleached kraft paper mills in that country (e.g., fishkills). The Swedish Environmental Protection Agency (SEPA) began regulatory action in 1986 to reduce the total organochlorine discharges from pulp and paper mill. In its regulation, Sweden relied on AOX because it is a relatively inexpensive and reliable measurement technique and because essentially all of the halides emitted from pulp and paper mills are chlorinated compounds (Thompson and Graham 1997). The SEPA established an AOX discharge limit of 1.5-2.0 kg/air-dried ton for Swedish mills (Berry *et al*. 1991).

According to EPA (1993a), however, the distribution of observed effects in fish populations does not appear to correlate well with AOX measurements, and there is no statistically significant relationship between the level of AOX and specific chlorinated

organic compounds, such as TCDD and TCDF. Berry *et al*. (1991) concluded that AOX is essentially linearly related to the amount of chlorine used in bleaching while the polychlorinated organic materials (dioxin, furan, and polychlorinated phenols) show a greater rate of reduction than AOX as the chlorine charge is reduced. Furthermore, according to an industry official, unlike pulp and paper mills in the U.S., Swedish mills do not employ secondary wastewater treatment that can biologically degrade some organochlorines. As a result, a Swedish mill would discharge a larger amount of organics (which deplete dissolved oxygen in water that fish require for respiration) and a different mix of pollutants.[30] On the basis of the Five Mill Study and the integrated risk assessment of dioxin from pulp and paper mills required under the consent decree, EPA (1993a) concludes that "although AOX concentrations can be used to determine the removal of chlorinated organics to assess loading reductions, they do not provide information on the potential toxicity of the effluent."

An environmentalist argues that because discharges of dioxin and pentachlorophenol below the analytical detection limits will not meet stringent, federal health-based ambient water quality criteria, it is justifiable to use reductions achieved in AOX loadings as a surrogate for comparing the efficacy of alternative pollution control technologies. The preference for AOX is also motivated by concerns that only a few of the numerous organochlorines discharged by bleaching mills have been identified or toxicologically tested and that many of the uncharacterized compounds could be environmentally hazardous. An industry official, on the other hand, focuses on the lack of correlation between AOX and effluent toxicity and argues that if alternative pollution control technologies yield similar concentrations of dioxin and furan, it is invalid to compare them on the basis of reductions achieved in AOX. The reductions in AOX, says this industry representative, do not achieve any measurable or monetizable environmental benefits. In addition, the estimated AWQC exceedances for dioxin are based on non-binding federal criteria that are substantially more stringent than the binding ambient criteria established by some States and approved by EPA. According to an academic, industry has demonstrated that it can meet the dioxin AWQC of states like Maryland without adopting all of the measures proposed by EPA in 1993.

[30] Secondary biological treatment can have a strongly reduce polychlorinated phenol discharge levels. However, the efficiency of chlorophenol removal by secondary treatment varies greatly. Secondary treatment has a weak effect on dioxin and furan levels (Berry *et al*. 1991).

*In terms of organochlorines, the main **measurable** difference between requiring 100% chlorine dioxide alone and requiring it in combination with oxygen delignification is a substantial reduction in AOX, a water quality indicator that is not statistically related to levels of dioxin or any particular organochlorine. Environmentalists justify using AOX because discharges of dioxin below the analytical detection limits will not meet stringent federal health-based criteria and because AOX is a surrogate for a "soup" of chlorinated organics about which little is known. Industry counters that reductions in AOX do not achieve any measurable or monetizable environmental benefits.*

This seemingly arcane disagreement over the use of AOX will perhaps be the pivotal issue in finalizing EPA's pulp and paper effluent regulations. As would be required under EPA's regulatory proposals, the pulp and paper industry has already begun to convert many of its bleaching plants from elemental chlorine to chlorine dioxide. As discussed above, in some plants where complete substitution of ClO_2 for elemental chlorine is currently being used, the result has been TCDD and TCDF concentrations in effluents that are below the limits of detectability. Dioxin and furan have been detected in other plants employing both complete substitution and oxygen delignification (AET 1994; ERG 1996). This information was unavailable at the time of the proposal because no pulp bleaching plants were operating using 100% chlorine dioxide prior to the proposal. (See discussion below.)

As originally proposed in 1993, the regulations would require large segments of the industry to employ OD (or extended delignification). Based on the new environmental performance data for mills employing complete substitution, the agency announced in 1996 that for the bleached papergrade kraft mills, two BAT options were being considered: complete substitution with and without additional delignification (*Fed. Reg.*, Vol. 61, pp. 36835-36858). In terms of organochlorines, the main *measurable* difference between the alternative technologies is a substantial reduction in AOX achieved by adding delignification.[31] Because there is no discernible threshold chlorine charge for dioxin formation in the pulp bleaching process, some imperceptibly small reduction in the formation of dioxin-like compounds may be associated with a reduction in AOX. How much reduction there would be in concentrations below the detection limit is speculative.

[31] EPA has estimated that bleached papergrade kraft mills using complete substitution and OD can achieve undetectable levels of TCDD and AOX concentrations of approximately 0.25 kg/kkg. In its 1996 notice, EPA proposed to set the daily maximum limitation for bleached papergrade kraft mills using complete substitution at 0.769 kg/kkg (*Fed. Reg.*, Vol. 61, p. 36842). According to an industry official, however, using complete substitution alone yields AOX concentrations of approximately 0.5 kg/kkg. Therefore, the incremental reduction in AOX appears to be on the order of 50-70%. The reductions in AOX are also associated with reductions in conventional pollutants such as chemical and biological oxygen demand. NRDC (1996) reports that EPA has estimated that requiring OD in addition to complete substitution would reduce the cumulative loading of chlorinated phenolic compounds by 2,000 kg per year. In EPA's 1996 notice, however, the agency indicated that both options were expected to achieve undetectable daily maximum bleach plant limits for specified chlorinated phenolics (*Fed. Reg.*, Vol. 61, p. 36841).

3. The Process within EPA

Setting the Agenda

According to an environmentalist, the inadvertent detection of high levels of dioxin in fish downstream of pulp and paper mills in 1983 was primarily responsible for getting the effluent regulations on the agency's agenda. An EPA official observes, "Even in the absence of [detecting] dioxin, there was an internal schedule within the water program that would have had them [the water office] review the effluent guidelines for pulp and paper mills. But the dioxin issue changed the pollutant of concern and added new impetus to that exercise."[32] Harrison and Hoberg (1991) conclude, however, that while the preliminary findings in 1983 were sufficient to get pulp mill dioxins quickly onto EPA's *research* agenda, when exposure of the findings to a wider audience occurred as a result of the 1987 *New York Times* article by Philip Shabecoff, pulp mill dioxins were elevated to the *regulatory* agenda.[33] Prominent press reports in 1989-90 concerning dioxin in milk from bleached paper cartons and dioxin-related fish advisories below pulp and paper mills kept up the pressure (Thompson and Graham 1997).

Sources from EPA and industry agree that the Reilly administration made the decision to combine the hazardous air pollutant and clean water rulemaking for the pulp and paper industrial "cluster" for administrative reasons, rather than as an attempt to take an integrated look at reducing environmental risks across environmental media from the sector as a whole.

Dioxin Formation Disclosed: The 5 Mill Study

Initially, EPA suspected that the source of dioxins detected in the 1983 national survey reference streams was use of dioxin-contaminated chlorophenols as "slimicides" on pulp mill machinery, rather than formation of dioxins during the process (Harrison and Hoberg 1991). In 1985, the Environmental Defense Fund and the National Wildlife Federation filed suit against EPA for denying the environmentalists' petition to regulate dioxins and furans under TSCA. Meanwhile, EPA tested wastewater treatment sludge from pulp and paper mills and found that dioxin levels were highest in the sludges of bleached kraft pulp mills. This suggested that dioxin was probably being formed as a by-product during the bleaching of wood pulp with chlorine. (*Fed. Reg*., Vol. 58, p. 66092). In 1986, EPA, the American Paper Institute (API), and NCASI (National Council of the Paper Industry for Air and Stream Improvement, the industry's research arm) agreed to undertake the "5 Mills Study." The study results detected TCDD and TCDF in effluents

---

[32] CWA Sec. 307 (a)(3) requires effluent standards to be reviewed every three years.

[33] Shabecoff and his successor at the *NYT*, Keith Schneider, have been prominent figures in the recent debate over the appropriate role of environmental journalists. Many viewed Shabecoff and other environmental journalists as being too sympathetic to environmental groups, and Schneider, who wrote a highly-publicized series of articles on EPA's over-estimation of the cancer risks of dioxin, went on to become the leading voice of a revisionist camp of environmental journalism.

of four of five mills, pulps of all five mills, and wastewater treatment plant sludges of all five mills.

Environmentalists learned of the agreement between EPA and the paper industry to conduct the 5 Mill Study, and in December of 1986, Greenpeace initiated a Freedom of Information Act (FOIA) request seeking all available information on the pulp mill dioxin problem. In January of 1987, a letter from an EPA official to API was leaked which suggested that EPA had agreed to notify the industry immediately of receipt of any FOIA requests and that, barring such requests or results indicating a potential threat to human health, the agency did not intend to release any results until publication of the final 5 Mills Study report. The following August, Greenpeace released a report alleging an EPA cover-up (Harrison and Hoberg 1991). In September, based on the 5 Mills Study and analyses of dioxin in paper products that were later added to the study, the *New York Times* ran a front-page story reporting that traces of dioxin had been detected in household paper products (Shabecoff 1987). Until this point, dioxin was primarily associated in the public mind with pesticides and combustion processes. The prominent disclosure that dioxin was formed by pulp bleaching and was present in common household products undoubtedly provided EPA with leverage to gain industry's cooperation to support additional research.

Integrated Assessment, the Consent Agreement, and the 104 Mill Study

According to an EPA official, when the agency's water program first realized that dioxin was being formed by pulp and paper bleaching, it immediately recognized that the problem went beyond its jurisdiction and approached the toxic substances program saying, "we have a problem to share with you." After a quick review, the agency decided that the issue spilled over into the risk management jurisdictions of other agencies. An interagency workgroup including EPA, FDA, the Consumer Products Safety Commission (CPSC), and the Occupational Safety and Health Administration (OSHA) was formed to identify the data needs to determine the extent of the problem. The interagency group met with API to lay out its plans for a multi-media, multi-pathway assessment of effluents, pulp, sludge, occupational, and consumer risks and requesting industry to bear the burden of the costs. An industry official says that EPA proposed using its TSCA (Sec. 4) authority to require the pulp and paper sector to provide the data, and noted that the authority supplied the agency "with an arrow in their quiver" during the negotiations. The American Forest and Paper Association (AF&PA, a newly consolidated industry trade association) "hit the roof" because of the TSCA threat, says this source. "It was a question of trust." EPA did not exercise its TSCA authority but retained it as a negotiating point. Thus began the "104 Mill Study." The assessment officially began in 1989, and an EPA official noted that the forest products industry spent $3-$4 million on the study.

According to an EPA official, to get a handle on the extent and magnitude of the problem, the interagency group insisted on representative samples from all 104 mills that used chlorine in bleaching. EPA assumed the coordinating role and concentrated on the risks from effluents, sludge, and occupational exposures. The EPA coordinator was a

Dwain Winters, an analyst in the Office of Toxic Substances (OTS). The FDA focused on food contact papers and medical devices. The CPSC looked at writing papers, diapers, and other consumer products. Overall, the group identified more than 153 separate dioxin exposure pathways to be analyzed. The interagency group developed a Quality Assurance/Quality Control protocol for conducting samples, and NCASI conducted the study.

In securing broad coverage to provide a strong analysis for the purposes of scoping and identifying the dioxin problem from bleaching mills, the interagency group traded off in-depth analysis at individual plants that would have been more useful in formulating a technological remedy. An academic observes that this investigative strategy also permitted the agency to avoid having to generalize between different mills using similar processes. Industry, on the other hand, wanted to do an intensive study of particular types of bleaching mills in order to better evaluate processes which caused dioxin formation and to identify the key steps that were responsible. Late in 1988, the industry designed and conducted an intensive study of 22 bleaching plants independent of the 104 Mill Study (Thompson and Graham 1997).

While the negotiations with industry were ongoing, the agency began separate negotiations with EDF and NWF regarding their suit by describing the planned study. In addition to writing the 104 Mill Study into the consent decree, EPA agreed to make determinations whether regulatory actions were required for the pulp, sludge, and effluent and, if so, to identify the information needed for regulatory decisionmaking. These decisions were subject to judicial review. An academic notes that by involving EDF and NWF in negotiating the plan for the 104 Mill Study, the scope of the study was broadened and the process was somewhat delayed.

According to an EPA official, as a result of the 104 Mill Study, dioxin in pulp and paper wastewater was identified as the route of major health concern, with land disposal of sludge being a secondary health concern. Agency analysts, however, flagged land disposal of sludge as the primary route of ecological concern. The agency did not view dioxin in paper products as a major risk, but it was “on the borderline of concern,” so the agency referred food contact papers to FDA under TSCA (Sec. 9). FDA accepted the referral and pursued voluntary reductions, according to this source. To arrive at these conclusions, the agency scientists combined the results of the 104 Mill Study with information regarding the health hazards of dioxin and analyses of various exposure pathways. Some of this information was taken as given (e.g., the estimated carcinogenic potency of dioxin supplied by the 1985 EPA hazard assessment), some of it represented departures from EPA’s normal assessment procedures for water quality criteria (e.g., assumptions made regarding fish consumption), and some of it resulted from original research (e.g., a study was conducted on uptake of dioxin through the skin using cadaver tissue samples).

Hazard Assessment

EPA had established its official position on the environmental and health effects of dioxin before the integrated assessment got underway. The agency issued its Ambient Water Quality Criteria Document for dioxin in 1984.[34] According to the hazard assessment issued by EPA in 1985, dioxin was to be regarded by all agency programs as a probable human carcinogen on the basis of adequate animal data and limited human data. The dose associated with an increased cancer risk of up to 10^{-6} was officially 0.006 pg/kg body weight/day. The agency's 1983 statement permitting states to use "other scientifically defensible methods" to modify EPA ambient water quality criteria, as well as the agency's initial stab at a dioxin reassessment in 1988, invited the States to exercise some scientific discretion in assessing the hazards of dioxin. For the EPA regulatory programs offices, however, using the results of the 1985 assessment remains non-discretionary. According to an EPA water official, it is clear that the toxicological information on dioxin available through the agency's Integrated Risk Information System (IRIS) was to be used in the program's analyses, "we use whatever ORD (the agency's Office of Research and Development) tells us to use."

The latest dioxin reassessment was underway by 1991, and the water program has acknowledged in its regulatory proposals that new information might become available, but there seems little chance that the agency will reach closure on the review of dioxin's toxicity before it finalizes the pulp and paper effluent guidelines. According to an EPA official, however, the minor adjustment made to the dioxin risk-specific factor in the 1994 draft reassessment (from 0.006 pg/kg/day to 0.01 pg/kg/day for a cancer risk of 10^{-6}) would not have significantly affected decisions about the pulp and paper sector. The existing Reference Dose (RfD) on the IRIS for dioxin non-cancer effects is at about the level where cancer risks approach 10^{-4} (one in ten thousand) Because the agency was concerned with individual risk levels below this (10^{-5} - 10^{-6}), says this source, estimated cancer effects drove the determination that the pulp and paper effluent was the prime human health concern. An EPA water official comments, "At this stage, on the effects of dioxin, as a user of science, I feel somewhat more certain. But for every question we've answered, we've raised new ones."

The scope of EPA's hazard assessment was limited by the complex and variable chemical composition of pulp and paper effluents and by the lack of toxicity data for those substances which were identified. Based on an evaluation of pulp and paper effluent sampling data collected by EPA (both independently and in cooperation with industry), the agency identified 26 organic chemicals (including dioxin and furan) as contaminants of concern. Of these 26 contaminants, 24 are organochlorines and 6 are priority pollutants. Only 11 have RFDs and 6 have cancer potency factors available using EPA's primary toxicological databases, IRIS and HEAST (Health Effects Assessment Summary

[34] The 1984 Criteria Document was prepared jointly by the EPA Offices of Water and Research and Development (ORD). The process was managed by the ORD Environmental Criteria and Assessment Office located in Cincinnati, OH. The health effects chapter acknowledged 44 contributors from EPA and other federal agencies, international institutions, academia, industry, and environmental groups.

Tables).[35] Due to a lack of data on human health toxicity, only 13 of the 26 contaminants could be evaluated for their potential human health impacts (EPA 1993a).

Exposure Assessment

The dioxin exposure assessment was conducted by a consultant (Tetra Tech) under contract to the EPA Office of Water/Office of Science and Technology/Standards and Applied Science Division. To conduct the assessment required estimates of several factors. The factors and the sources of the estimates are provided in Table A-5. An examination of the table reveals a grab bag of types and sources of scientific information: peer reviewed literature, gray literature, default assumptions, *ad hoc* assumptions, and professional judgments produced by all levels of government, industry, and academia.

Table A-5. Sources of the Estimates Used in Dioxin Exposure Assessment.

Exposure Factor	**Source of Estimate**
Dioxin discharges from plants	104 Mill Study
In-stream dioxin concentrations	Site-specific flow data for 68 streams, dilution factors for 17 mills discharging to open waters (e.g., oceans, estuaries, lakes), and two dilution models. Dilution factors provided by Office of Water publication and Regional EPA personnel. One of the dilution models was developed by EPA/Office of Research and Development/Exposure Assessment Group and was still under EPA review at the time.
Dioxin concentrations in fish	Bioconcentration factor (BCF) for TCDD in trout from a laboratory study included in 1991 Banbury Report edited by Gallo *et al.* BCF for TCDF derived from 1988 article in *Environmental Toxicology and Chemistry*. Biota to Suspended Solids Accumulation Factor (BSSAF) of 0.09 derived from EPA Lake Ontario study. BSSAF of 0.02 derived from NCASI study.
Fish consumption rates	For recreational anglers, estimates based on a 1988 New York State survey; a 1981 Tacoma, WA County Health Department Report; and a 1989 University of Michigan Technical Report. For subsistence anglers, 145 g/day assumed and said to be "consistent" with a 1982 U.S. Dept. of Ag. report.
Change in fish consumption due to State fishing advisories	EPA assumed a 20% decrease for recreational anglers and no effect on subsistence anglers based on a 1990 University of Michigan M.S. Thesis and a 1990 paper presented at an American Fisheries Society Meeting.
Size of fish-consuming populations	Number of fishing licenses in a county multiplied by average family size (based on Census Bureau data) of 2.63 to yield fish-consuming population in each county. EPA assumed that 95% of fishing licenses were issued to recreational anglers and 5% to subsistence anglers.
Exposure duration, body weight, etc.	EPA Office of Solid Waste and Emergency Response Standard Default Exposure Factors

[35] The priority pollutants are chloroform, methylene chloride, pentachlorophenol, TCDD, TCDF, and 2,4,6-trichlorophenol.

The exposure assessment for dioxin in pulp and paper effluents marked a departure from EPA's standard operating procedures for developing water quality criteria. Normally, the water office would assume fish consumption levels based on national averages. In this case, however, the agency focused on exposure scenarios it expected would occur near pulp and paper mills, including highly exposed subpopulations--sports anglers and subsistence anglers. According to an EPA official, for the fish consumption rates of highly exposed subpopulations, risk analysts can only provide some reasonable upper-bound estimate that is subject to a high degree of uncertainty due to the lack of data. In contrast, there are good data available on average fish consumption rates by the general population. This source allows that "the way we chose to calculate the risk for fish consumption became complicated. The normal procedure under water quality criteria assumed a certain level of average consumption, but we had to make assumptions that went beyond that to look at specific subpopulations like subsistence anglers. Nobody believed that the average consumption rates would be representative of real exposures, so the solution was to provide a number of consumption rates."

EPA also evaluated the exposure scenarios under two alternative models for estimating how dioxin concentrations would be diluted in receiving water bodies. The first was a simple dilution model, which assumes that all carcinogenic pollutants discharged into a receiving stream are available for uptake by fish. The other--called the DRE model--is a more complex dilution model under development by EPA . It assumes that the uptake of dioxins and furans depends on the levels of suspended solids and the partitioning of the pollutants between fish tissue and sediment. As a result, the partitioning model produces lower exposure estimates.

In EPA's 1996 notice that it was considering two BAT options for the major pulp and paper subsector, the agency also stated that it was considering using only the DRE model for estimating dioxin and furan concentrations in fish for the final rule. However, the agency stated that it would modify the DRE model, however, to reflect ongoing contamination. The modification entailed replacing the Biota to Suspended Solids Accumulation Factor (BSSAF) of 0.09 with a BSSAF factor of 0.2 (*Fed. Reg.*, Vol. 61, p. 36846). This might seem trivial, but the BSSAF is a critical model parameter in predicting the accumulation of dioxin in fish. The BSSAF value of 0.09 represents the agency's default for dioxin based on Lake Ontario data which is primarily from historical sources. To support its conclusion that a BSSAF factor of 0.2 was more appropriate for ecosystems subject to ongoing contamination, the agency cited EPA (1994c), one of the volumes of the agency's draft dioxin reassessment. Although EPA (1994c) indicates that BSSAFs for aquatic systems where contamination is ongoing might be greater than from systems where the contamination is primarily historical, it stops short of endorsing any particular value for all water bodies where contamination is ongoing. EPA (1994, Sec. 7.2.3.6) describes an analysis of the impact of pulp and paper mill effluents on fish tissue concentrations conducted by NCASI using data from the 104-mill study. This study found that *for the 38 mills discharging into smaller water bodies*, using a BSSAF of 0.2 (up from 0.09) improved the predictive performance of the model. According to EPA (1994c, Table 4-1), BSSAFs for dioxin observed in the literature range from 0.009 to 2.94.

Therefore, EPA's assumption that a BSSAF of 0.2 is appropriate for *all* water bodies downstream of discharging mills, regardless of the size of the receiving water body and other factors, appears to have a somewhat tenuous basis. More importantly, however, this analytic anecdote illustrates the problems associated with attempting to conduct an environmental risk assessment for a national rulemaking while taking into account site-specific conditions. On the basis of at least some relevant, empirical data, EPA is adjusting its default model to take into consideration differences between historical and ongoing dioxin contamination. Making a finer distinction could potentially require a large number of field studies to determine the BSSAF for every water body downstream of a bleaching pulp and paper mill. Furthermore, despite the importance of the BSSAF in predicting the dioxin accumulation in fish tissue, it is only one of many exposure factors to be evaluated.

The Regulatory Impact Assessment: The Numbers Behind the Proposal

The Water Office's Regulatory Impact Assessment (RIA) estimated that the proposed BAT for the pulp and paper industry would result in a national annual reduction of 5-35 cancers in recreational and subsistence anglers (EPA 1993b). The lower value in the range is only for TCDD and TCDF and was estimated using the partitioning dilution (DRE) model. The upper value is calculated using the simpler, conservative dilution model and is still dominated (99%) by the effects of TCDD and TCDF. Using a range of $2 to $10 million for the value of a life, EPA (1993b) estimated the benefits of the proposed effluent regulations to be $10 - $350 million per year.[36] Using the simple dilution approach, EPA (1993a) estimated that greater than 99 percent of the noncancer hazard (as indicated by the mills exceeding RfDs for recreational and subsistence angler populations) can be attributed to dioxin and furan.

Senior EPA sources have suggested that the industry has framed the issue narrowly by focusing on cancer effects and failing to consider non-cancer effects of dioxins. An EPA water official, however, notes that the program was unable to monetize the benefits of non-cancer health effects in the Regulatory Impact Assessment (RIA) due to the lack of quantitative dose-response data for non-cancer effects. (This situation is generic to non-cancer effects and not unique to dioxins.) According to this official, "we did a very good job with what science we had." Not surprisingly, an industry official characterizes the RIA as a "poor job." An environmentalist complains that the Office of Water feels compelled to conduct a detailed risk and economic assessment for a "technology-based" rule. However, EPA is required by Executive Order to conduct a Regulatory Impact Analysis for all proposed regulations with an annual economic impact of more than $100 million.

[36] According to Viscusi (1996), on average, workers receive $3 - $7 million in compensation per statistical life lost.

Communicating the Science to Decisionmakers

Because Assistant Administrator for Water Robert Perciasepe was confirmed only days before the 1993 proposed effluent regulations were issued in compliance with the consent decree, he was unable to engage in the decisionmaking. According to an EPA water official, the key decisionmakers included Acting Assistant Administrator Martha Prothro and Tudor Davies, Director of the Water Office of Science and Technology (then acting Deputy Asst. Adm.). Reportedly, EPA Administrator Carol Browner was also briefed. The issue was of sufficient importance that it was not completely delegated to an Acting Assistant Administrator (AA). "The Cluster Rule was high stakes, high visibility," says this source, adding "this involved the 'D-word' [dioxin]. It had everybody's attention." There were presentations for Prothro from NCASI, AF&PA, and academics. "The AA attended mill [plant site] visits--that's unusual." Due to the high level of decisionmaker interest, according to this source, it was "not difficult to get their attention." In addition, because dioxin is regarded as an important multi-media pollutant, the decisionmakers "knew a lot about it; they were sensitized and could mesh things together about dioxin other than what they just heard about in the context of pulp and paper."

4. The Proposal, Industry's Response, and a New Framework

In announcing the 1993 proposal, Assistant Administrator Perciasepe estimated that dioxin discharges from the pulp and paper sector into surface waters would be reduced from over 300 grams to less than 30 grams per year (*Environment Reporter*, 11/5/93, pp. 1227-1228). The BAT effluent regulations proposed in 1993 for four pulp and paper subsectors organized by production process are summarized in Table A-6.

In 1993, NCASI reported data on the pulp and paper industry's progress in reducing the dioxin content of effluents, pulps, and wastewater treatment sludges (NCASI 1993). In 1994, a study prepared for a supplier of chlorine dioxide technology, the Alliance for Environmental Technology (AET), reported that substitution of chlorine dioxide for elemental chlorine reduces the formation of chemicals like dioxin that accumulate in fatty tissues to the limits of detectability (AET 1994). Among the study's authors was Robert Huggett, a marine biologist at William and Mary College, and soon-to-be-EPA Assistant Administrator for ORD.[37]

[37] Huggett recused himself from involvement in the pulp and paper dioxin rule.

Table A-6. 1993 Proposed BAT and Estimated Impact for Pulp and Paper Subsectors.

Subsector	Principal Products	Proposed BAT	Size/Est. Cost
Bleached papergrade kraft and soda	papergrade kraft market pulp, paperboard, coarse papers, tissue papers, and fine papers for business, writing, and printing	OD* and 100% ClO_2	78 mills $260 annualized 1-3 plant closures 500-4,000 jobs
Dissolving sulfite	pulps used for rayon, cellophane, and cellulose products	OD and 100% ClO_2	5 mills $5 annualized 1 plant closure
Dissolving kraft	pulps used for rayon, acetate, and other cellulose products	OD and 70% ClO_2	3 mills $11.9 annualized No plant closures
Papergrade sulfite	tissue paper, fine papers, newsprint	TCF	10 mills $25 annualized 2 plant closures

OD* - oxygen delignification or extended cooking of pulp prior to bleaching.
OD - oxygen delignification prior to pulp bleaching.
100% ClO_2 - complete substitution of elemental chlorine (Cl_2) for chlorine dioxide (ClO_2) in the bleaching process.
70% ClO_2 - seventy percent substitution of Cl_2 for ClO_2.
TCF - Totally Chlorine Free bleaching using peroxide (H_2O_2) or ozone (O_3).
Costs in millions of 1991 dollars for all the mills in the subsector, as estimated by EPA.[38]

The new data on the performance of alternative technologies made it clear to EPA sources that *human health* risks resulting from land disposal of pulp and paper sludge were moot. Agency officials felt confident that whatever the final outcome of the Cluster Rule, it would simultaneously control human health risks associated with sludge land disposal. EPA (1990) estimated that TCDD soil concentrations of 0.12 ppt and 12.0 ppt would produce human cancer risks of 10^{-6} and 10^{-4}, respectively. The new performance data suggested that alternative technologies achieved overall decreases in the formation of dioxins. This meant that the dioxins were not simply being shifted from the wastewater to the sludge.

Some EPA official, however, remained concerned about possible *ecological* effects of sludge land disposal. EPA (1990) estimated that if sludge were to be land applied, dioxin concentrations as low as 0.03 ppt would be necessary to protect the "most sensitive" terrestrial species, identified as the American woodcock (*Scolopax minor*). The bird would be highly exposed to dioxin in soils because the contaminant accumulates in

[38] According to an industry official, EPA underestimated the capital costs of the proposed BAT by more than 40% because OD, in combination with some of the best management practices (BMPs) EPA has proposed (particularly spill controls) may require plants to install larger recovery furnaces (costing approximately $100 million per plant).

the woodcock's primary food source, worms.[39] Some EPA staff felt that the agency's decision to retract the proposed regulation of sludge land disposal did not give due consideration to ecological risks. They also felt that environmental advocates who had been engaged in the issue lost interest once human health risks appeared under control. According to an industry official, however, the AF&PA insisted upon an SAB review of the ecological risk assessment. The SAB determined that while the agency had identified a hazard to the woodcock, it had not substantiated that population-level effects would result from land disposal of dioxin-tainted pulp and paper sludge.[40] In 1994, EPA negotiated a voluntary agreement with the pulp and paper industry (Gilman and EPA 1994) that places no land disposal restrictions on sludges containing less than 10 ppt dioxin and serves primarily to formalize best management practices.

In 1995, EPA announced the availability of sampling data characterizing the performance of bleached papergrade kraft and papergrade sulfite mills employing complete substitution of elemental chlorine with ClO_2 with and without oxygen delignification (*Fed. Reg.*, Vol. 60, pp. 34938-24940). In July 1996, EPA announced that its review of new data on the performance of chlorine dioxide bleaching indicated that dioxins and furans in wastewater discharges from bleached papergrade kraft and soda mills could be reduced by 95 percent and 99 percent, respectively (EPA 1996). The agency also announced a "new framework" for the pulp and paper effluent guidelines. The primary elements of this new regulatory framework included: a) consideration of two BAT options for two pulp and paper subsectors: bleached papergrade kraft and papergrade sulfite; b) deferral of a final decision for the dissolving sulfite and dissolving kraft subsectors; and c) a voluntary incentives program to reward mills that exceeded regulatory requirements in reducing discharges (*Fed. Reg.*, Vol. 61, pp. 36835-36858).

For the bleached papergrade kraft subsector, EPA announced that it was considering complete (100%) substitution of ClO_2 for elemental chlorine (Option A) and complete substitution with OD (or extended delignification) (Option B). EPA stated that both options appear to reduce dioxins and furans in wastewaters to concentrations at or below the current analytical detection limits. The incremental environmental benefits that EPA attributed to the use of OD (or extended delignification) included reduced chronic toxicity to some aquatic species. EPA stated that the reduced chronic toxicity is probably attributable to a reduction in chemical oxygen demand. (COD is a conventional water pollutant, the technological control of which is subject to a test of economic reasonableness, as discussed above.) The agency also stated that the reduced chronic toxicity may reflect an incremental reduction in the potential formation of dioxin and furan

[39] The woodcock is a favored game bird. Adults woodcocks are approximately 1 foot long and weigh half a pound and can eat their weight in worms daily. In response to the drop in woodcock numbers over the last 20 years in some parts of its range (from the Georgia to the Canadian maritime provinces and west to Minnesota), the U.S. Fish and Wildlife Service announced the North American Woodcock Management Plan in 1990. Because the woodcock's breeding habitat is successional scrub forest, the principal cause of their decline appears to be the maturation of hardwood forests growing on abandoned farmlands in the northeast (http://alloutdoors.com; //www.im.nbs.gov).

[40] It should be noted that estimating population-level effects is extremely uncertain and can require lengthy and costly site-specific studies.

below the analytical detection limits as well as a reduction in all chlorinated compounds loadings, as measured at the end-of-the-pipe by AOX. (Recall that dioxin and furan are priority pollutants, the technological control of which is subject to the less demanding economically achievable standard.) The notice further states:

> Although statistically significant relationships between AOX and a broad range of specific chlorinated organic compounds have not been established, trends in concentrations changes have, however [sic], been observed between AOX and specific pollutants, including dioxin, furan, and chlorinated phenolic compounds. Even though dioxin and furan are no longer measurable at the end-of-pipe at many mills, the potential for formation of these pollutants continues to exist at pulp and paper mills as long as any chlorine-containing compounds (including chlorine dioxide) are used in the bleaching process...EPA expects that [reductions in] AOX discharges...will in turn further reduce the likelihood of the formation and discharge of these chlorinated organic pollutants.

Thus, EPA appears to reject the claim that there is a threshold level of chlorine below which no dioxin formation occurs in bleaching pulp. The agency also implicitly suggests that it is considering requiring reductions in priority pollutants beyond the point of diminishing returns because such reductions are achievable. (Recall that the determination of BAT involves consideration of both technological and economic achievability.)

Table A-7 summarizes the proposed effluent limits under the 1996 Options A and B for the Bleached Papergrade Kraft and Soda subsector. Table A-8 summarizes EPA's estimates of the economic impacts under Options A and B (*Fed. Reg.*, Vol. 61, pp. 36840-36841).

Table A-7. Bleached Papergrade Kraft and Soda Plant (Daily Maximum) Limitations

Pollutant	Option B 1993 OD* and 100% ClO_2	Option A 1996 100% ClO_2	Option B 1996 OD* and 100% ClO_2
TCDD	ND	ND	ND
TCDF[41]	359 (ng/kkg)	24.1 (pg/l)	24.1 (pg/l)
Chlorinated Phenolics[42]	ND	ND	ND
Chloroform (g/kkg)	5.06	5.33	5.33
AOX (kg/kkg)[43]	0.267	0.769	0.236

OD* - oxygen delignification or extended cooking of pulp prior to bleaching.
ND - No detect.

Table A-8. Estimated Impacts for the Bleached Papergrade Kraft and Soda Subsector under 1996 Options A and B.[44]

Option B 1993 OD* and 100% ClO_2	Option A 1996 100% ClO_2	Option B 1996 OD* and 100% ClO_2
78 mills $223.2 Annualized 1-3 plant closures 500-4,000 jobs	85 mills $140 Annualized 1 plant closure 500 jobs	85 mills $155 Annualized 3 plant closures 4,100 jobs

OD* - oxygen delignification or extended cooking of pulp prior to bleaching.
Costs in millions of 1995 dollars, as estimated by EPA.

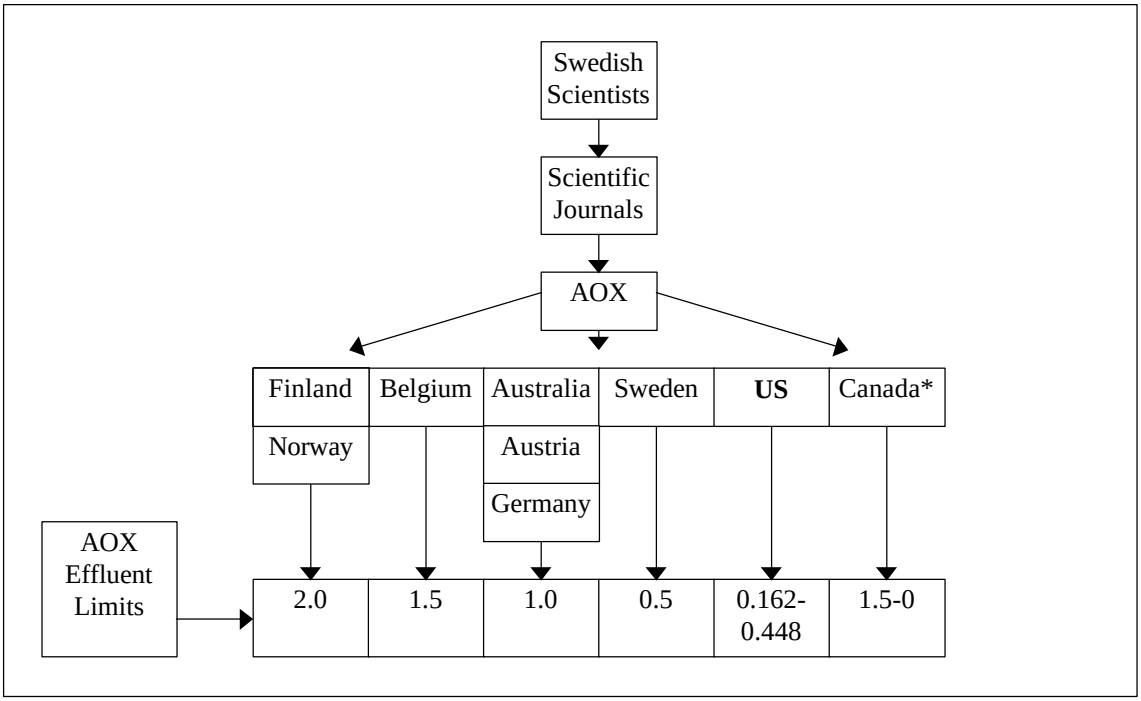
5. Concluding Observations

Sources interviewed for this case study suggest that the treatment of AOX is the principal outstanding science policy issue regarding the final BAT determination for the pulp and paper effluent guidelines. In terms of a fate and transport analogy, the information provided by AOX was first transported to EPA from Sweden in the early 1980s via international scientific journals. In 1986, the use of AOX as a regulatory parameter achieved a measure of international legitimacy when the Swedish EPA adopted it. Australia, Austria, Belgium, Finland, and Germany have followed, and AOX was thoroughly assimilated by EPA into the 1993 proposal. However, the fate of the information provided by AOX varied considerably across countries. The AOX limits currently being considered by EPA (0.448 kg/ton or .162 kg/ton based on a monthly

[41] The difference in units for TCDF between the 1993 proposal and the 1996 options reflects a change from a production-normalized bleach plant limitation to an effluent concentration-based limitation.
[42] ND for two pollutants (trichlorosyringol and 2,4,6-trichlorophenol)(mg/kkg).
[43] Whereas the other pollutant limits are measured at the bleach plant, that for AOX is an end-of-pipe measurement.
[44] Differences in estimated impacts between the 1993 proposal and Option B in 1996 in Table A-7 reflect a revised economic impact analysis. The difference between Tables A-6 and A-7 in the annualized cost estimate for the 1993 proposal is due to expressing the costs in different base year dollars (1991 or 1995). As indicated above, industry sources believe that EPA has substantially underestimated the incremental capital costs of OD.

average limitation) are slightly to 3 times lower Sweden's, currently the strictest national-level standard for softwood pulp mills (EKA 1996; Thompson and Graham 1997).[45] The fate and transport of AOX in international pulp and paper effluent limits is illustrated in Figure A-1.

Figure A-1. Fate and Transport of AOX in International Pulp and Paper Effluent Limits (kg/ton for softwood pulp mills).



*There is no national Canadian AOX effluent limit. It varies by province.

Although the formation of dioxin and other organochlorines from pulp bleaching seems plausible at even minimal levels of chlorine input, the effect that AOX has on EPA's comparison of dioxin control alternatives is impeded by the agency's inability to transform AOX into toxicological equivalent units. According to one EPA official, "on AOX, we have a lot more information, and some specific questions have been addressed, but I don't think we've moved too far in being more certain about its environmental significance. While the uncertainty has gone down some, the level of controversy may have heightened. As the analysis now stands, we would continue to use AOX as a regulatory parameter." This is a case, add this source, where perfect information might make the decision for the decisionmaker.

An environmentalist suggests that the impact of science has been very high--but negative--in this case. The Clean Water Act "is a technology-based statute, and EPA has

[45] Softwoods are evergreens. Hardwoods are deciduous. In the US, most pulp is produced from softwood. Sweden has set an AOX limit of 0.3 kg/ton for hardwood pulp mills to be achieved in 2000-2005 (EKA 1996).

digressed from the technology basis." This source points out that requiring OD in addition to complete substitution is superior in preventing organochlorine discharges into the environment and complains that the "health effects analysis has tied [EPA] up in knots." This source blames industry and OMB for pressuring EPA to emphasize cost-effectiveness criteria and to stray too far from the congressional intent to rely on technology-based standards to achieve environmental results in the face of scientific uncertainty. This source also points out that the Natural Resources Defense Council still has a 1993 petition pending with EPA to ban all discharges of dioxin from the pulp and paper industry under CWA Sec. 307a. The NRDC's position is that due to the technological inability to detect dioxins at concentrations believed to be toxic, the ban can only be achieved by prohibiting the use of chlorine in pulp bleaching.

An EPA official appreciates that when one evaluates a technology-based standard in terms of its adequacy to protect health and safety, it may be seen as falling short of the mark. "For some people, *any* exposure to dioxin is unacceptable." The pulp and paper industry, once widely perceived as environmentally recalcitrant, has taken some voluntary and negotiated steps to demonstrate how it could dramatically reduce dioxin discharges. These steps were taken in an attempt to get out ahead of the regulatory curve and in response to consumer demand for environmentally-friendly products and potential tort liability concerns.[46] The industry may have also sensed that the ongoing dioxin reassessment would not produce substantial regulatory relief.

In February 1997, Assistant Administrator Perciasepe announced that the pulp and paper effluent rulemaking would not be released for several months (*Environment Reporter*, 2/12/97, p. 2142). An April 1997 article appearing in *The Washington Post* suggested that the "Pulp Friction" saga had spilled over into the halls of Congress (Skrzycki 1997). In light of the performance of newly adopted pulp bleaching technologies, it remains to be seen whether EPA will judge the substantial and *measurable* reductions in discharges of dioxins and other organochlorines as sufficient to scale back from its 1993 proposal to impose more stringent and costly pollution control technologies. To a considerable extent, the decision hinges on the marginal "unmonetizable" benefits of technologically imperceptible reductions in the formation of dioxin, which, in the eyes of some, remains the Darth Vader of chemicals.

[46] See Thompson and Graham (1996) for further discussion of the possible role of tort liability risk in the pulp and paper industry's decision to adopt advanced bleaching technologies.

REFERENCES

AET (The Alliance for Environmental Technology). 1994. A Review and Assessment of the Ecological Risks Associated with the Use of Chlorine Dioxide for the Bleaching of Pulp.

AET (The Alliance for Environmental Technology). 1995. Five Great Reasons Why We Care: The Pulp and Paper Industry's Virtual Elimination Strategy. September. (http://aet.org/science/5reasons.html.)

Berry, R., C. Luthe, R. Voss, *et al*. 1991. "The effects of recent changes in beached softwood kraft mill technology on organochlorine emissions: An international perspective," *Pulp and Paper Canada*, Vol. 92, No. 6, pp. 43-55.

Colborn, T., D. Dumanoski, and J. Peterson Myers. 1996. *Our Stolen Future: Are We Threatening Our Fertility, Intelligence, and Survival?* Dutton: NY.

Copeland, C. 1993. Toxic Pollutants and the Clean Water Act: Current Issues. Congressional Research Service Report for Congress (93-849 ENR).

CRS (Congressional Research Service). 1993. Toxic Pollutants and the Clean Water Act: Current Issues. CRS Report 93-849 ENR.

EKA (EKA Chemicals, Inc.). 1996. EKA Chemicals Comments on Cluster Rule Proposal. August 12, 1996. EKA Chemicals, Inc., Marietta, GA.

EPA (U.S. Environmental Protection Agency). 1989. Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated dibenzo-p-dioxins and dibenzofurans (CDDs and CDFs) and 1989 Update. Risk Assessment Forum.

EPA (U.S. Environmental Protection Agency). 1990. Assessment of Risks from Exposure of Humans, Terrestrial and Avian Wildlife, and Aquatic Life to Dioxins and Furans from Disposal and Use of Sludge from Bleached Kraft and Sulfite Pulp and Paper Mills. Office of Pesticides and Toxic Substances, July.

EPA (U.S. Environmental Protection Agency). 1993a. Water Quality Assessment of Proposed Effluent Guidelines for the Pulp, Paper, and Paperboard Industry. Office of Water (EPA-821-R-93-022).

EPA (U.S. Environmental Protection Agency). 1993b. Regulatory Impact Assessment of proposed Effluent Guidelines and NESHAP for the Pulp, Paper, and Paperboard Industry. Office of Water.

EPA (U.S. Environmental Protection Agency). 1994a. Estimating Exposure to Dioxin-Like Compounds. Volume 1: Executive Summary. External Review Draft. Office of Research and Development.

EPA (U.S. Environmental Protection Agency). 1994b. Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Vol. III of III. External Review Draft.

EPA (U.S. Environmental Protection Agency). 1994c. Estimating Exposure to Dioxin-Like Compounds. Volume III: Site-Specific Assessment Procedures. External Review Draft.

EPA (U.S. Environmental Protection Agency) 1996. EPA releases new data on pulp and paper discharges; considers regulatory and voluntary options. July 3 press release.

EPA-V (U.S. Environmental Protection Agency, Region V). 1988. USEPA Bench Scale Wastewater Treatability Study, Proposed Interim Control Measures, Interim NPDES Permit Strategy.

EPA/SAB (EPA/Science Advisory Board). 1995. *A Second Look at Dioxin*.

ERG (Eastern Research Group, Inc.). 1996. Pulp and Paper Mill Data Available for BAT Limitations Development. Prepared under contract for USEPA Office of Water. July 10.

Executive Enterprises. 1984. *Clean Water Act Permit Guidance Manual*. Executive Enterprises Publications Co., Inc.: Wash., DC.

Fingerhut, M., W. Halperin, B. Marlow, *et al*. 1991. "Cancer Mortality in Workers Exposed to 2,3,7,8-Tetrachlorodibenzo-p-Dioxin," *New England Journal of Medicine*, Vol. 342, pp. 212-218.

Finkel, A. 1988. "Dioxin: Are We Safer Now than Before?," *Risk Analysis*, Vol. 8, No. 2, pp. 161-165.

Fogarty, J. 1991. "A Short History of Federal Water Pollution Control Law," *Clean Water Deskbook*, 2nd ed. Environmental Law Institute: Wash., DC, pp. 5-20.

Gilman and EPA. 1994. Memorandum of Understanding between Gilman Paper Company and the U.S. Environmental Protection Agency Regarding the Land Application of Pulp and Paper Mill Materials.

Hanmer, R. 1988. Memorandum from Rebecca W. Hanmer, Acting Asst. Adm. for Water to Water Management Division Directors, "Interim Strategy for the Regulation of Pulp and Paper Mill Dioxin Discharges to the Waters of the United States, August 9.

Harrison, K. and G. Hoberg. 1991. "Setting the Environmental Agenda in Canada and the United States: *Canadian Journal of Political Science*, March, pp. 3-27.

Hirshfield, A., M. Hirshfield, and J. Flaws. 1996. "Problems Beyond Pesticides," *Science*, Vol. 272, pp. 1444-1445.

Jasanoff, S. 1990. *The Fifth Branch: Science Advisors as Policymakers*. Harvard Univ. Press: Cambridge, MA.

Kociba, R. D. Keyes, J. Beyer, *et al*. 1978. "Results of a Two Year Chronic Toxicity and Oncogenicity Study of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin in Rats," *Toxicology and Applied Pharmacology*, Vol. 46, pp. 279-303.

Lucier, G., G. Clark, C. Hiremath, *et al*. 1993. "Carcinogenicity of TCDD in Animals," *Toxicology and Industrial Health*, Vol. 9, pp. 631-668.

Moore, J. R. Kimbrough, and M. Gough. 1993. "The Dioxin TCDD: A Selective Study of Science and in M. Uman, ed. *Keeping Pace with Science and Engineering*. National Academy of Engineering: Wash., DC, pp. 221-242.

NCASI (National Council of the Paper Industry for Air and Stream Improvement). 1993. Summary of data reflective of pulp and paper industry progress in reducing the TCDD/TCDF content of effluents, pulps, and wastewater treatment sludges, NY: NCASI, June.

NRDC (Natural Resources Defense Council). 1996. "Option B" Has Significant Environmental Benefits. NRDC: Wash., DC.

NTP (National Toxicology Program). 1982. Bioassay of 2,3,7,8-Tetrachlorodibenzo-p-dioxin for Possible Carcinogenicity (Gavage Study), Technical Report Series Number 102. NTP: Research Triangle Park, NC.

OTA. (U.S. Congress, Office of Technology Assessment). 1989. *Technologies for Reducing Dioxin in the Manufacture of Bleached Wood Pulp*. OTA-BP-0-54.

OTA (U.S. Congress, Office of Technology Assessment). 1991. *Dioxin Treatment Technologies*. OTA-BP-0-93. GPO: Wash., DC.

Powell, M. 1996. 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. RFF Discussion Paper 97-05. Resources for the Future, Wash., DC.

Roberts, L. 1991. "Dioxin Risks Revisited," *Science*, Vol. 251, pp. 624-626.

Shabecoff, P. 1987. "Traces of Dioxin Found in Range of Paper Products," *New York Times*, Sept. 24, p. A1.

Skrzycki, C. 1997. "Pulp Friction: The EPA's Tussle Over Paper Pollutant Rules," *The Washington Post*, April 11, pp. G1-G2.

Smith, B. 1992. *The Advisers: Scientists in the Policy Process*. Brookings Inst.: Wash., DC.

Thompson, K.M. and Graham, J.D. 1997. "Producing Paper without Dioxin Pollution," *The Greening of Industry: A Risk Management Approach*, pp. 203-268. Harvard Center for Risk Analysis: Cambridge, MA.

Viscusi, K. 1996. "Alternative Institutional Responses to Asbestos," *Journal of Risk and Uncertainty*, Vol. 12, pp. 147-170.

Whelan, E.M. 1985. *Toxic Terror*. Jameson Books: Ottawa, IL.

LIST OF ABBREVIATIONS

AET	Alliance for Environmental Technology
AF&PA	American Forest and Paper Association
AOX	adsorbable organic halide indicator
APA	American Paper Institute
AWQC	ambient water quality criteria
BAT	best available technology
BCF	bioconcentration factor
BCT	best conventional (pollution control) technology
BMP	best management practice
CDC	Centers for Disease Control and Prevention
CPSC	Consumer Product Safety Commission
CWA	Clean Water Act
EDF	Environmental Defense Fund
EPA	Environmental Protection Agency
FDA	Food and Drug Administration
FOIA	Freedom of Information Act
FWPCA	Federal Water Pollution Control Act
IARC	International Agency for Research on Cancer
IJC	International Joint Commission
IRIS	Integrated Risk Information System
NCASI	National Council of the Paper Industry for Air Stream Improvement
NRDC	Natural Resources Defense Council
NWF	National Wildlife Federation
OD	oxygen delignification
OMB	Office of Management and Budget
OTA	Office of Technology Assessment
SAB	Science Advisory Board, EPA
TCDD	tetrachlorodibenzo-para-dioxin
TCDF	tetrachlorodibenzofuran
TEF	toxic equivalent factor
TSCA	Toxic Substances Control Act of 1976
USDA	United States Department of Agriculture

B. Lead in Soil at Superfund Mining Sites

1. Background

This case study deals with an environmental hazard, lead in soil, for which EPA has no universally-applicable standard that regional and state officials can take from a "look-up table" and "plug in" to determine the "Preliminary Remediation Goal" (PRG) for contaminated site remediation. Establishing the PRG is the first step that Superfund remediation project managers take in the remedy selection process to begin to address the question of "how clean is clean?" Although technical feasibility, cost, the permanence of the treatment, applicable or relevant and appropriate requirements (ARARs), and other factors are considered by remediation project managers in negotiating the final remedy selection with the PRPs, community, and other agencies, the PRG can serve as an important benchmark in the negotiations. Because there is no discrete number indicating "safe" levels of lead in soil, this case study does not describe the typical process by which EPA uses scientific information in site-specific decisionmaking. Instead, it provides some insight into the challenges and opportunities facing contaminated site programs if they were to more broadly and consistently develop and use site-specific scientific information.

The Comprehensive Environmental Response, Compensation, and Liability Act, (CERCLA) of 1980 created the EPA Superfund program. CERCLA had two primary objectives: to identify and clean up sites contaminated with hazardous substances throughout the U.S. and to assign the costs of cleanup directly to those parties--called responsible parties--who had something to do with the sites (Probst *et al*. 1995). Although Superfund is not solely limited to closed or abandoned sites, the program has primarily focused on these types of sites. The number of old mineral extraction, milling, and processing sites within the U.S. is unknown, but large, with estimates ranging between 100,000 and 400,000 sites. As of 1990, the Superfund National Priorities List (NPL) contained about 1,200 sites, of which sixty were mining sites.[47] An additional 220 mining sites are listed by the Comprehensive Environmental Response Compensation and Liability Information System (CERCLIS) (Tilton 1994).[48] Lead in soil is a contaminant of concern at over 400 Superfund sites (Ryan and Zhang 1996).

The Resource Conservation and Recovery Act (RCRA) of 1976 deals broadly with hazardous waste produced at operating industrial sites. Mineral extraction and ore milling generate two billion tons of solid waste a year, representing nearly 40% of the country's total solid waste, and dwarfing the 270 million tons of hazardous waste generated outside

---

[47] As used here, the generic term "mining sites" refers to locations where various stages in the mining process occurs. It includes, but is not limited to, sites where mineral-bearing rock called ore is extracted from underground or surface mines. During milling (or beneficiation), ore is processed (or upgraded), usually at or near the site of extraction, into concentrates which are further processed in a smelter or refinery. Final processing sometimes occurs at a considerable distance from the site of extraction.

[48] CERCLIS contains information on over 30,000 potentially hazardous sites (Tilton 1994). According to a report by the Society of Environmental Geochemistry and Health, as of 1993, soil lead had been found at 922 of the 1,300 sites on the NPL (*Environmental Health Perspectives*, 11/94, pp. 912-913).

the mineral sector. Responding to concerns of the mining industry, however, Congress passed the "Bevill Amendment" in 1980 (RCRA Sec. 3001(b)(3)(A)(i-iii)) to exempt "high-volume, low hazard" solid wastes from RCRA Subtitle C hazardous waste federal regulation. Currently, wastes generated from extraction and milling are classified as non-hazardous wastes and subject to state regulation under RCRA Subtitle D, while many (but not all) mineral processing wastes fall under Subtitle C (Tilton 1994). While much of the ongoing discussion concerning lead in soil regulation occurs in the context of the Superfund program, the potential liability for future RCRA "Corrective Action" motivates the owners of active industrial operations to engage in the debate.

Many old mining sites are relatively small and isolated, but the contamination at some mining and processing sites (including areas in the "fallout zone" downwind of smelters) is extensive. The sheer magnitude of many mining sites, relative to most contaminated sites, presents EPA with an extraordinary risk management challenge. EPA has identified approximately 50 "Large Area Lead Sites." The Jasper County, MO Superfund Site, for example, includes 10 different areas covering a total of 7,000 acres.[49] At a Superfund site located in Midvale, Utah, the old tailings pond alone covers 260 acres and contains 14 million tons of material in uncovered piles.[50] In some cases, these large sites overlap residential areas. At the California Gulch Superfund Site in Leadville, CO, mountainous tailings piles lie in close proximity to residences in the small, remote mining

[49] Jasper County is an historic lead and zinc mining, milling, smelting area located in southwest Missouri. It is located in EPA Region 7, headquartered in Kansas City, KS. Mining activities in Jasper County began around 1850 and continued through the 1950's, and millions of tons of mining, milling, and smelting waste materials are dispersed throughout the county, including residential areas. The site was added to the Superfund National Priority List in 1990 (Baysinger-Daniel 1995).

[50] Tailings are the fine waste particles that are produced during ore milling and typically suspended in water. Under current practices, tailings from surface mines are deposited in a tailings (or settling) pond, while those from underground mines are deposited in the mine itself. Historically, tailings may have simply been piled in waste yards or placed in a settling pond. At abandoned sites, however, uncovered tailings piles often remain after settling ponds have dried up.

The Sharon Steel site is located in Midvale, Utah, a small town twelve miles south of Salt Lake City. Around 1906, the US Smelting, Refining, and Mining Company began processing ores at Midvale, and in the mid-1920s, the company built a mill to process lead-zinc ores and a lead smelter. During World War II, Utah was a major lead-producing region. ASARCO (American Smelting and Refining Company) also operated a lead smelter in Murray, and the International Smelting and Refining Company, a subsidiary of the Anaconda Company, had a lead mill and smelter at Tooele. After the war, lead-zinc mining production in Utah declined. In 1958, US Smelting and International Smelting agreed to share facilities. US Smelting closed its Midvale smelter and shipped its concentrates to Tooele for smelting and International Smelting closed its mill and shipped its ore to Midvale for milling. In 1971, US Smelting closed its Midvale mill and changed its name to UV Industries, and in 1979, a year before passage of CERCLA, the company went bankrupt. US Smelting sold the Midvale site to Sharon Steel, which planned to use the site for commercial purposes. The mill and smelter had been torn down, but the tailings pond and slag pile from the smelter remained. The tailings pond contained 14 million tons of uncovered piles up to 50 feet deep and covered 260 acres. The fine-grained material blew off-site, and 44,000 people lived within 2 miles of the site. Health effects were first suspected in 1982, and the site was proposed for the NPL in 1984 (Tilton 1994). Site characterization is an important element in assessing what specific wastes (e.g., lead carbonate or lead sulfide) and proportions of different wastes (e.g., milling wastes vs. smelter emissions) may be present on a particular site. Due to the complex history of many mining sites, this can be difficult.

town. According to Murray (1995), current or historical mining areas of the inter-mountain west often encompass tens or hundreds of square miles.

Although there are a variety of risk management measures that can be taken to limit exposure to lead-contaminated soil for extended periods (e.g., capping small sites with asphalt[51]), the cleanup standards provisions of the 1986 Superfund Amendments and Reauthorization Act (SARA, Sec. 121) explicitly state a congressional preference for permanent treatment. Currently, the only proven, permanent remedial alternative for lead-contaminated soil is excavation and disposal.[52] The cost and extent of the cleanup at Large Area Lead Sites depends on the level or concentration of lead in soil that is considered hazardous (i.e., the lower the level of soil lead that EPA considers a health concern, the greater the extent and cost of the cleanup). Given the enormous volume of material involved, excavation and disposal could cost liable parties hundreds of millions of dollars for some Large Area Lead Sites.[53]

Opposition to EPA's proposed remedies for these sites has not come exclusively from the Potentially Responsible Parties (PRPs), however. In some cases, local governments and citizens have objected. The city of Midvale, for example, objected to EPA's proposed solution because it would have precluded future commercial use of the site. Leadville residents voiced concerns about property values and jobs.[54] At the Smuggler Mountain site near Aspen, CO,[55] local residents challenged the need for EPA's proposed remedy by questioning the basic scientific model that EPA uses for assessing the health risks from soil lead (Tilton 1994). (The agency's "IEUBK Model for Lead in Children" is discussed in greater detail below.) SCAM (Superfund Coalition Against Mismanagement), an umbrella group of citizens representing 45 communities around the country, filed suit in 1994 against EPA for issuing *de facto* soil lead cleanup regulations

---

[51] According to an EPA official, the agency has decided to initiate soil excavation at the Jasper County Site, with the lead-contaminated soils being deposited in the roadbed of a new highway construction project. Due to their areal extent, capping Large Area Lead Sites with asphalt is not regarded as a feasible alternative.

[52] In this context, disposal means deposition in a facility designed to permanently contain the contaminated soil. Federal scientists and academics are researching alternative remediation methods involving the application of phosphate, iron oxide, and sewage sludges intended to permanently render soil lead less soluble. See, e.g., Ryan and Zhang (1996). An independent toxicologist comments, however, that while phosphate may temporarily immobilize lead, it can mobilizes arsenic, which is commonly associated with lead on sites contaminated by extractive mining.

[53] According to an EPA official, the initial remediation cost estimate for the Sharon Steel-Midvale Site was in excess of $200 million. All of the sites involved in EPA's Phase II bioavailability study (discussed below) had projected cleanup costs of more than $100 million. Probst *et al*. (1995) suggests that for the mining industrial sector as a whole, the annual financial burden of Superfund cleanup and transaction costs, estimated at $220.5 million under the current law, is likely to be quite large in relation to the industry's profitability.

[54] According to Murray (1995), Leadville homeowners have suffered a substantial drop in property values. An EPA official estimates that 80% of the employment in Leadville remains mining-related and suggests that residents "were parroting whatever the mining company said."

[55] The Smuggler Mountain Superfund Site covers approximately 75 acres and is located about 1 mile northeast of Aspen, CO, in Pitkin County. A mobile home park and two condominium developments are located a few hundred yards from the site (Lagoy *et al*. 1989).

for CERCLA and RCRA sites as OSWER guidance without seeking public input (*Environment Reporter*, 12/16/94, p. 1608).[56]

A principal basis for the OSWER soil lead cleanup guidance is the IEUBK model. The agency's default assumption imbedded in its IEUBK model about how much lead in soil ingested by children would be absorbed into their bloodstream is commonly regarded as driving the cost of the proposed cleanups and has been hotly contested in these cases. In general, the higher the rate of lead absorption, the lower the soil lead Preliminary Remediation Goal indicated by the IEUBK model. As a result of the standoff over the assumed rate of lead absorption, any cleanup decision regarding the Large Area Lead Sites throughout the country was deferred for several years (Tilton 1994) while EPA Region 8 (centered in Denver, CO) directed a study on the lead absorbed by juvenile swine after digesting soil samples from 8 different sites. Based on previous laboratory studies using rodents, the presumption by many was that EPA's model generally overstated the amount of lead that would be absorbed from soil on mining sites, and that it especially overestimated it for mining sites without significant historical milling or smelting activity. The juvenile swine experiment results, however, indicated considerable variability among the sites tested, with some higher, some lower, and some about the same as the agency's default assumption, according to an EPA scientist. For at least one site, lead absorption from soils downwind of the smelter was unexpectedly *lower* than that from soils contaminated with milling wastes. Consequently, it appears that EPA cannot generalize across sites where similar mining activities occurred or even draw any general distinctions between different types of mining sites.

According to an EPA official, the total cost (excluding EPA staff time) of the lead absorption studies directed by EPA on 8 Large Area Lead Sites was $1.4 million, a small sum compared to the potential liability associated with the sites. However, soils from more than 40 sites currently identified as Large Area Lead Sites remain untested, and many active or old mining sites could become subject to future regulation. Furthermore, once the site-specific lead absorption rate is no longer contested, a new variable in the complex risk equation (e.g., local children's blood lead levels or activity patterns) can replace it as the disputed factor that drives remediation costs.[57]

---

[56] CERCLA does not require EPA to conduct national rulemaking. As a result, EPA relies on administrative guidance to implement Superfund. However, the 1992 Residential Lead-Based Paint Hazard Reduction Act (Title X of the Housing and Community Development Act) required EPA to develop standards defining hazardous levels of lead in lead-based paint, household dust, and soil (adding Title IV to the Toxic Substances Control Act) by 1994. In July 1994, EPA issued guidance for CERCLA and RCRA (OSWER Directive # 9355.4-12), as well as interim guidance under TSCA. The agency has not yet promulgated a formal standard for lead in soil under TSCA. According to press reports, EPA issued TSCA guidance in lieu of a formal rule because existing scientific data could not justify the standards the agency was considering and suitable data may not be available for five years (*Inside EPA*, 12/16/94, p. 14).

[57] For example, in the absence of mining activity, background levels of children's blood lead would tend to be higher than normal in areas with naturally high lead content in the rock and soils. Therefore, once the site-specific lead absorption rate is determined, the debate could shift to ascertaining the relative contribution of the contaminated site to children's measured blood lead levels. Alternatively, in a probabilistic analysis of the IEUBK model funded by the Asarco Corporation, Lee *et al.* (1994) concluded

Many Superfund critics have characterized the program's approach to risk assessment as following a "cookbook" and have criticized the program for ignoring site-specific risk assessment information. Under Superfund, EPA, state agencies, or environmental consultants often conduct site-specific exposure assessments of varying scope, depth, and complexity, but the program typically adopts the substance-specific cancer risk values (slope factors), Maximum Contaminant Levels (MCLs), and Reference Doses (RfDs) supplied by EPA's Integrated Risk Information System (IRIS) in a wholesale fashion. Superfund generally uses slope factors to assess whether a site's cancer risks lie in the 10^{-4} - 10^{-6} "action" range. In accordance with the ARARs provision of the 1986 SARA, MCLs often determine groundwater remedial objectives. RfD's are used to evaluate potential non-cancer health effects from soil ingestion.

However, EPA's current goal for reducing lead health risks is to ensure that children's blood lead (PbB) levels do not exceed 10 μg/dL (micrograms per deciliter). (Over the past decade the PbB level commonly associated with impairment has decreased from 25 μg/dL to 10 μg/dL). There is no numerical MCL for lead in drinking water, nor is there a single numerical RfD for ingested lead.[58] Consequently, the Superfund program has not had the *option* of following its standard operating procedures for evaluating risks and determining Preliminary Remediation Goals for lead-contaminated sites.[59] This Superfund case study, therefore, illuminates the challenges and opportunities posed by developing and using rigorous site-specific scientific information. Table B-1 provides a summary background on Large Area Lead Sites.

---

that the driving variables include bioavailability of lead and dietary intake of lead. Therefore, once the site-specific lead absorption rate is determined, the debate could shift to estimating the actual dietary intake of lead.

[58] See Powell (1996) for discussion of the development of EPA's lead policy and regarding EPA's unusual RfD for arsenic. EPA Region 8 is also directing juvenile swine studies to determine the bioavailability of arsenic-contaminated soils.

[59] In principle, Superfund PRGs are developed without consideration of technical feasibility, cost, and public acceptance.

Table B-1. Background on Large Area Lead Sites.

1976	RCRA enacted.
1980	CERCLA enacted. Congress passes Bevill amendment to RCRA.
1985	EPA concludes that some wastes associated with mineral processing meet the hazardous criteria for regulation under RCRA Subtitle C, but that the high volumes of waste from extraction and milling do not. US Centers for Disease Control (CDC) sets screening level for children's PbB at 25µg/dL, suggests lead in soil or dust appears to be responsible for blood lead levels in children increasing above background levels when the concentration in the soil or dust exceeds 500-1000 ppm.
1986	SARA requires the Dept. of Health and Human Services' Agency for Toxic Substances and Disease Registry (ATSDR) to prepare a study of lead poisoning in children. After EPA fails to determine which mineral processing wastes would come under the jurisdiction of RCRA Subtitle C, the Environmental Defense Fund and the Hazardous Waste Treatment Council sue the agency.
1988	ATSDR suggests a potential risk of developmental toxicity from lead exposure at PbB levels of 10-15 µg/dL or lower; identifies paint and contaminated soil as the principal sources of lead for children most at risk (ATSDR 1988). DC Circuit Court of Appeals orders EPA to use the high-volume, low-hazard criteria to narrow the scope of the RCRA Bevill Amendment exemption for mining wastes.
1989	EPA Office of Solid Waste and Emergency Response (OSWER) guidance recommends a soil lead cleanup level of 500-1000 ppm at residential Superfund sites. EPA issues final rules during 1989 and 1990 making most mineral processing wastes subject to RCRA Subtitle C; however, slag from lead and zinc processing is among 20 mineral processing wastes which remain exempt from federal regulation. Extraction and milling wastes remain classified as non-hazardous wastes and thus fall under RCRA Subtitle D.
1990	US Department of Justice files a Superfund suit against Sharon Steel, UV Industries Liquidating Trust, and Atlantic Richfield Company (ARCO) regarding a closed lead smelting facility in Midvale, Utah. ARCO claims that soil lead on the Midvale site poses no hazard. EPA Region 8 directs study rejecting ARCO's claim. The companies eventually agree to pay the government $63 million.
1990	EPA Clean Air Science Advisory Committee (CASAC) recommends a maximum safe PbB level for children of 10 µg/dL. OSWER issues RCRA program guidance on soil lead cleanup describing three alternative methods for setting cleanup levels: 1) use preliminary results of the IEUBK model, 2) use 500-1000 ppm range, or 3) use "background" levels at the facility.
1991	EPA formalizes its multi-media Strategy for Reducing Lead Exposures, adopting the 10µg/dL PbB level goal. EPA Science Advisory Board (SAB) weakly endorses IEUBK model for developing soil lead cleanup levels at CERCLA and RCRA sites.
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. OSWER circulates draft guidance setting 500 ppm lead in soil as the PRGs for Superfund remediations and media cleanup standards (MCSs) for RCRA corrective actions.

Table B-1. Background on Large Area Lead Sites (cont'd).

1994	Urban Soil Lead Abatement Demonstration Project (the 5-year, $15 million "3 Cities Study") finds a link between soil-lead levels and blood-lead levels in children. However, the study suggests that soil abatement alone (without lead-based paint stabilization and/or household dust abatement) will have little or no effect on reducing exposure to lead unless there is a substantial amount of lead in soil (as in Boston test sites, where soil lead levels averaged 2,400 ppm) and unless soil lead is the primary source of lead in house dust (*Science*, 10/15/93, p. 323; EPA (1995a)). OSWER issues "revised interim guidance" under CERCLA and RCRA for residential soil lead setting a "screening level" of 400 ppm. The screening level is based on the IEUBK model using national average inputs and on a goal of limiting exposure to soil lead levels such that a child would have an estimated risk of no more than 5% of exceeding the 10 µg/dL PbB level. The screening level may be selected as the soil cleanup goal (PRG/MCS), or a site-specific assessment using the IEUBK model can be used to develop soil cleanup goals. The guidance recommends remedial action when the goal is exceeded, but adds that soil excavation may not be necessary. Research to determine the bioavailability of soil lead is encouraged for mining sites without significant past milling/smelting activity. Citizens' group sues EPA for issuing *de facto* regulation as guidance without public comment.
1995	House Commerce Committee Chair Thomas Bliley (R-VA) expresses concern about EPA's soil lead cleanup guidance and objects to the agency's reliance on the IEUBK model to justify the lead policy (*Environmental Executive Report*, 10/30/95). EPA proposes to tighten controls of many mineral processing wastes previously exempt from RCRA Subtitle C.
1996	Results of juvenile swine studies directed by EPA Region 8 indicate considerable variability in soil lead bioavailability among Large Area Lead Sites.

2. Scientific Issues

NRC (1993) reports that the health effects of lead at approximately 10 µg/dL in blood include:

- impaired cognitive function and behavior in young children
- increases in blood pressure in adults, including pregnant women
- impaired fetal development
- impaired calcium function and homeostasis[60] 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 transport, and energy metabolism) and cautions that some cognitive and behavioral effects may be irreversible.

[60] This refers to maintaining calcium at appropriate levels in the body.

The primary commercial use for lead is making batteries.[61] In 1990, the U.S. produced (excluding recycling) approximately 500 thousand tons of lead (Young 1992). Environmental releases of lead from the minerals industry results not only from mining lead and lead-zinc ores, but more generally from mining and smelting of non-ferrous (noniron) metals. Copper mining, for example, can release lead into the environment. Like all heavy metals, lead accumulates in the environment because it does not degrade. In soils, lead tends to accumulate in surface organic matter (Kabata-Pendias and Pendias 1984). In contrast to EPA's soil lead screening level of 400 ppm, the lead content of agricultural soils ranges from 1 to 135 ppm with a typical value of 10 ppm (Ryan and Zhang 1996). As a result of industrial and automotive emissions and exterior lead paint, inner-city neighborhoods in many of our major cities have elevated accumulations of lead in soil.[62] Because lead is reported to be least mobile in soils among the heavy metals (Kabata-Pendias and Pendias 1984), the leaching of lead into groundwater is generally of less concern than is direct ingestion of soil lead, particularly since children frequently engage in hand-to-mouth activity.[63] According to an EPA scientist, lead and arsenic are generally the principal contaminants of concern to human health at mining sites.

During the mid-1980s, EPA began developing a risk assessment model that strives to take into account simultaneously the various pathways of lead exposure--inhaled auto and industrial emissions and ingested soil, dust, food, and drinking water.[64] The model, referred to as the Integrated Exposure Uptake Biokinetic Model for Lead in Children (or IEUBK Model), was developed by the Office of Air and Radiation Office of Air Quality Planning and Standards (OAR/OAQPS), first in the context of reviewing the National Ambient Air Quality Standard for lead. The model was later used for the Lead/Copper drinking water rulemaking (see Powell (1996)), and is currently managed by the EPA Office of Research and Development's National Center for Environmental Assessment (ORD/NCEA). The model predicts a distribution of children's blood lead levels as a function of existing blood lead levels, inputs from the various sources, and different lead "bioavailability" rates. Bioavailability refers to the rate and extent of absorption of a substance. For ingested substances, it is measured by the fraction of the orally

---

[61] Lead is also used for radiation shielding, cable covering, ammunition, chemical reaction equipment, fusible alloys, type metal, vibration damping in heavy construction, foil, and bearing alloys (Hawley 1981). Leaded gasoline, new lead plumbing, and the pesticide lead arsenate have been phased out.

[62] Ryan and Zhang (1996) report that many inner-city neighborhoods have mean or median soil lead concentrations in excess of 1000 ppm, with values as high as 50,000 ppm being reported. However, an OSWER official questions whether these values are overestimates. By comparison, the Urban Soil Lead Abatement Demonstration Project was performed in areas with median soil lead concentrations ranging from 237 - 2,396 ppm (EPA 1995a). Suffice to say that levels of lead in soil in many urban areas, particularly in older neighborhoods, are considerably higher than background levels due to accumulated anthropogenic releases of lead into the urban environment.

[63] However, lead does become more soluble in acid soils (Kabata-Pendias and Pendias 1984), and sulfides (which can produce sulfuric acid) make up more than a third of many nonferrous mineral ores (Young 1992).

[64] According to EPA officials, the IEUBK model builds on research conducted in the late 1970s at New York University by Harley and Kneip, who developed a pharmacokinetic model for lead (describing the absorption, movement, storage, and excretion of lead in various compartments of the body) by conducting studies with juvenile baboons and by analyzing human cadavers. The IEUBK added exposure components (levels of lead in air, food, water, soil, and dust) to the pharmacokinetic model.

administered dose that is absorbed by the body.[65] The default assumption for the bioavailability of ingested lead in soil and dust under the IEUBK Model is 30%.[66] According to Murray (1995), site-specific risk estimates for contaminated mining areas are sensitive to variability in assumed bioavailability parameter values. Other things being equal, the higher the rate of lead absorption, the lower the level of soil lead that presents a health concern (and, potentially, the greater the extent and cost of cleanup).

Bioavailability of lead is a function of a number of factors, including the solubility of the chemical form of lead ingested, the soil particle size and surface area-to-weight ratio, the pH of the digestive tract, and nutritional status. The physical and chemical characteristics of soil lead vary from site to site. Based largely on the argument that the soil lead at the Smuggler Mountain Superfund Site "is almost certainly less bioavailable" than the "lead present in soils in the vicinity of smelters and in urban areas where most of the studies relating the concentration in soil to body burdens have been conducted," a group of environmental consultants estimated that soil lead levels of 1,000 ppm would be "safe" (Lagoy *et al*. 1989).[67] Findings reported by Steele *et al*. (1990) and Hemphill *et al*. (1991) suggested that lower-than-default soil lead bioavailability values may be appropriate generally for mining sites, particularly where the predominant source of lead contamination was not late-stage processing activities such as smelting and milling that produce very fine particle sizes.

Lead absorption also varies with age. It is higher for children than for adults because calcium and lead absorption are linked. Children absorb calcium at high rates because their bones are growing, but the human metabolism does not distinguish well between lead and calcium, so lead unfortunately "goes along for the ride." As a result, the most common laboratory animals, mature rodents, are not the best animal model for assessing lead bioavailability if physiological similarity to children is the primary evaluative criterion.[68] Emphasizing this criterion, EPA Region 8 selected juvenile swine as the appropriate animal model for testing soil lead bioavailability on Large Area Lead Sites.

Swine, however, have some distinct disadvantages as laboratory animals. Notably, they are large (approximately 35 lbs.) and expensive. In contrast to the abundance of

---

[65] The absorption rate can be a function of administered dose. For example, according to an independent toxicologist, the relationship between blood lead and dose becomes curvilinear at higher doses due to either attenuated uptake or more efficient excretion.

[66] By contrast, the IEUBK default bioavailability for the generally more soluble forms of lead present in food and water is 50%.

[67] Lagoy *et al*.. (1989) based their determination partially on 1985 CDC guidance which suggested that increased blood lead levels were associated with soil lead levels of 500-1000 ppm. Lagoy *et al*.. (1989) also indicate that various approaches to establishing a "safe" level of lead in the soil yield widely varying results ranging from about 100 ppm to 8000 ppm.

[68] According to a federal scientist, juvenile swine have been found to be better animals to model human uptake of nutrients and carbohydrates. An EPA scientist notes that rodents mature at 6-10 weeks, whereas swine mature more slowly. Other problems with the rodent model include a low "residence time" for food (ingested substances pass through the animals quickly, leaving little time for lead absorption) and the fact that rats eat their feces, resulting in a confusing "redosing" of the animals with excreted lead.

laboratories certified to conduct tests with small animals (e.g., rodents, rabbits, and fish), there are very few facilities across the country that can provide the necessary quality assurance to conduct reliable laboratory studies using large animals.[69] A soils scientist notes that rodent studies cost 10 times less, on a per animal basis, than juvenile swine studies (the cost per soil sample is $5,000 and $50,000, respectively). Consequently, for a given sum of money, researchers using rodents can conduct a larger, more statistically powerful study (i.e., one able to detect smaller differences between treatment levels).[70] However, the rodent studies may systematically underestimate soil lead bioavailability in children. According to an EPA scientist, Superfund PRPs promoted the rodent model, because "conveniently for them, it showed low absorption, but that's expected because

To a considerable extent, selection of the most appropriate animal model involves tradeoffs between cost, experimental power and control, fidelity to children's physiology, and the value of information for decisionmaking. Determination of the "optimal" animal model may depend on which evaluative criterion is being used. According to a soils scientist, when the US Department of Agriculture (USDA) conducts nutritional research, it begins with rodent studies to contain research costs, and then, if deemed necessary, moves up progressively to "higher," more costly animals, such as swine or primates. Underscoring the subjectivity involved, this source remarks that in the future, using the results of rodent studies calibrated by the available swine data "is good enough for me. With all of the lead and zinc mine wastes [that need to be tested], it will save millions [of dollars]."

Currently, researchers are working to develop cheaper *in vitro* laboratory models to study lead bioavailability.[71] Most parties seem to agree that *in vitro* methods are ultimately needed to get around the need for animal research altogether. But until a cheap, reliable, and valid *in vitro* model is developed, disputes over selection of the most appropriate animal model will linger. Disagreement among scientists about the *physiological* suitability of the competing animal models has largely subsided, and opinion among toxicologists has apparently converged on the juvenile swine as an appropriate test animal for high stakes Superfund decisions. But in the early 1990s, when EPA Region 8 initiated the swine studies, the bulk of previous work on the bioavailability of heavy metals in soils had been conducted with rodents. According to an EPA scientist, "other investigators in the field were challenged professionally by [EPA's] decision to use a large animal model." Selection of the appropriate animal model was hotly contested not only by scientists, but also by PRPs and their environmental consultants.

---

[69] The juvenile swine studies were conducted at the University of Missouri under contract with EPA.
[70] An EPA scientist suggests that the precision of the swine studies has been greater than expected.
[71] Scientists at the University of Colorado Geochemistry Department and the EPA/ORD/ECAO laboratory in Cincinnati, OH are working to develop a "fake GI [gastrointestinal] tract," says an EPA scientist. The benchtop model consists of a series of chambers with flow-thorough mechanisms, and preliminary results have been "pretty consistent" with those from the juvenile swine model.

3. Process Within EPA

Setting the Agenda

According to an OSWER official, the program was first introduced to the IEUBK model by Jeff Cohen, who was responsible for developing the model as a member of the OAQPS staff and later directed the Office of Drinking Water's Lead Task Force during finalization of the 1991 Lead/Copper Drinking Water Rule. "Our usual approach," says the OSWER official, "is to use RAGS [the Risk Assessment Guidelines for Superfund], site-specific exposure assessments, and then go to IRIS [the Integrated Risk Information System] for cancer slope factors, MCLs, and RfDs. For lead, those were not available to us in IRIS, and we needed some other way to evaluate the sites."

EPA Region 8 first applied the IEUBK model in a Superfund context in the late 1980s on a site in E. Helena, MT where there was a closed lead smelter.[72] According to an EPA scientist, data collected from the site were used to develop agency assumptions in the soil/dust ingestion component of the IEUBK model. For the purposes of the E. Helena remedy selection, the results of the IEUBK model were not used to generate the final numerical soil cleanup level. Instead, "the model was used as a means of technical negotiations with the PRP, ASARCO (American Smelting and Refining Company)," says an EPA official. "EPA was quite successful with it under those circumstances," in part because one of the ASARCO technical consultants had worked academically in development of the model.

Phase I

In 1989, OSWER issued guidance (OSWER Directive #9355.4-02) recommending a soil lead cleanup level of 500-1000 ppm[73] at Superfund sites where the land was currently in residential use or where remediation project managers believe future residential use is possible.[74] Late in the same year, the federal government was preparing for litigation on the Sharon Steel Superfund Site in Midvale, Utah. The site historically had been a lead smelting operation, and there were also mining wastes present on the location. One of the PRPs, the Atlantic Richfield Company (ARCO), argued that the

---

[72] In 1986, the US Centers for Disease Control and the EPA issued a report, *East Helena, Montana, Child Lead Study, Summer 1983*.

[73] This range is identical to that in the 1985 CDC guidance to which Lagoy *et al*.. (1989) referred.

[74] The role of landuse in Superfund cleanups has been a source of considerable controversy. EPA/OSWER (1989, p. 6-7) directs personnel to "assume future residential land use if it seems possible based on the evaluation of the available information." The presumption of residential land use affects risk assessment exposure assumptions regarding levels of on-site soil ingestion, and it may also affect assumptions regarding the quality of drinking water drawn from groundwater. According to EPA (1994), residential landuse is the fourth most common current landuse at Superfund sites and the second most common expected future landuse. According to EPA (1995b), 15% of Superfund sites have people living on site, 80% have residence adjacent to them, and about 20% of the time, when a commercial/industrial site has residences nearby, EPA will assume a future residential land use. See Probst, Wernstedt, and Hersh (1996) for a discussion of the many definitions of land use and its role in Superfund cleanups.

particular form of soil lead at the site was not hazardous because it had *zero* bioavailability. The Department of Justice, which is responsible for litigating Superfund cases in court, approached a toxicologist with EPA Region 8, Chris Weis, to ascertain whether ARCO could be right. Weis recommended a short-term experiment using juvenile swine to test ARCO's argument. The results of the study (now referred to as Phase I) rejected ARCO's assertion that the soil lead had zero bioavailability, but also suggested it was somewhat lower (20-25%) than EPA's default assumption under the IEUBK model (30%).[75]

Unbeknownst to EPA and the Justice Department, ARCO had already conducted some lead bioavailability tests on soil samples from the Midvale site using rodents, and the PRP felt that it had a rock-ribbed scientific argument for minimizing its liability. According to an EPA scientist, "ARCO was furious that we criticized the studies they had [76] The PRP may have been surprised by EPA's challenge because although EPA Regional Offices are generally staffed with a complement of environmental engineers and hydrogeologists, they typically contain relatively few experts in the health aspects of environmental risk assessment, such as Weis.[77] An EPA official observes, "They [ARCO] were for the first time faced with a trained toxicologist who could challenge their evidence. Now they had to contend not only with lawyers and engineers, but also with trained scientists." The PRPs "brought their checkbook to court" and settled on the first day of the trial for $63 million. Prior to the swine study, the estimated cleanup cost was more than $200 million, according to an EPA official.[78]

The resolution of the Sharon Steel case did not, however, clear up the dispute about EPA's use of the IEUBK model for setting numerical soil lead cleanup goals, either for the national Superfund program or for any particular contaminated site. Instead, the terms of debate shifted to attempts to distinguish among types of Large Area Lead Sites where soil lead was expected to be more or less bioavailable (thus obviating the need for costly and time-consuming site-specific applied research) and to arguments over the appropriate laboratory animal for use in testing soils for lead bioavailability (i.e., rats vs. pigs).

---

[75] OSWER Headquarters and ORD/ECAO each provided $100,000 for Phase I.

[76] According to an independent toxicologist, external scientists such as Herbert Needleman of the University of Pittsburgh, John Drexler of the University of Colorado, and Paul Mushak of PB Associates were also prepared to testify as expert witnesses on behalf of the government in the Sharon Steel case. Mushak had submitted a critique of ARCO's arguments that laboratory solubility tests approximated the behavior of lead tailings in humans. Drexler had performed laboratory analyses of Midvale soil samples indicating that lead was present in very fine particles, and therefore more likely to be bioavailable, and also consisted of toxic chemical forms. See Powell (1996) for further discussion of this case and Needleman's role.

[77] Weis has a Ph.D. in environmental toxicology, is a board certified toxicologist, and conducted post-doctoral research in physiology and biophysics at the University of Virginia Medical School prior to joining EPA Region 8. According to an OSWER official, Regions 1 and 8 have a comparatively large number of health scientists.

[78] Tilton (1994) estimates that the transaction costs of doing the technical work, negotiating and litigating the case accounted for between a fifth and a third of the total funds devoted to the Midvale site by the PRPs and EPA.

Phase II

Although OSWER had issued broad soil lead guidance for Superfund and RCRA in 1989-90, pressure was mounting for EPA to provide more definitive guidance for lead-contaminated soil cleanups. In 1992, the Residential Lead-Based Paint Hazard Reduction Act required that EPA develop national standards for hazardous levels of lead in soil. Many EPA remediation project managers and PRPs were hopeful that the Sharon Steel site test results indicating lower-than-IEUBK-default soil lead bioavailability could be applied to the remainder of the Large Area Lead Sites. Others, including scientists who had conducted previous heavy-metal bioavailability research and some PRPs, continued to defend the application of rodent studies. However, EPA Region 8 officials cautioned that a decision to apply the Sharon Steel site test results (Phase I) to all mining sites could not be supported technically due to the variability in site conditions.[79] According to Murray (1995), the PRPs for the Leadville site submitted the results of rodent studies with lower-than-default bioavailability values. Given the results of Phase I, Region 8 was reluctant to depart from the default on the basis of rodent studies. Subsequently, a second tranche (Phase II) of juvenile swine studies was conducted testing lead bioavailability in soil samples from 7 different Superfund sites across the country.[80]

For some sites, the results indicated bioavailability rates substantially lower than the IEUBK default of 30%. For example, the bioavailability was 19% at the Bingham Creek Superfund Site near Salt Lake City, Utah (Murray 1995). The studies have not, however, consistently yielded bioavailability rates lower than 30%. According to EPA officials, the Leadville site results were not substantially lower than the default. For the Jasper County site, the bioavailability of lead in soils contaminated by smelting emissions was in the 28-31% range. Surprisingly, the bioavailability of soil lead from mining wastes was higher--in the 40-50% range. This finding was contrary to the conventional wisdom that smelter fallout would produce the most highly bioavailable lead form present on mining sites.

The Role of External Scientists in the Process

After holding a 1988 conference on lead in soil, the Society for Environmental Geochemistry and Health (SEGH) formed a "Lead in Soil" Task Force to examine the relationship between blood lead and environmental lead and to develop guidelines for assessing and managing the health risks associated with lead in soil and dust. (For a discussion of the role of SEGH in the arsenic in drinking water case, see Powell (1996).) In 1990, the SEGH, with support from EPA and other sponsors, convened a scientific meeting in Chapel Hill, NC on bioavailability and dietary intake of lead. The SEGH task

[79] Phase I was also designed simply to quickly *test* the hypothesis that the soil lead bioavailability at the Midvale site was zero. A larger, more elaborate test was required to *estimate* the actual bioavailability with much precision.

[80] Phase II studies were supported by regional Superfund program budgets (with cost recovery provisions applicable).

force report (summarized in Wixson and Davies 1994) questioned the use of a single value for lead in soil for use in all cleanup situations. An EPA scientist says, "I got the feeling that it [SEGH] was somewhat dominated by the regulated industry. But to their credit, nobody else was providing a forum for these discussions." The major impact that SEGH had was in focusing EPA's attention on the need to consider differences among sites in soil lead bioavailability.[81]

In 1991, a year after OSWER issued RCRA program guidance permitting the use of the IEUBK model for setting soil lead cleanup levels, the EPA Science Advisory Board (SAB) concluded that the IEUBK model represented an improved methodology for assessing total lead exposure and for developing soil lead cleanup levels at CERCLA and RCRA sites. However, the SAB raised concerns about incorrect application of the model and selection of inappropriate input values for default and site-specific applications. In response, OSWER developed a guidance manual for the IEUBK model.

4. Science in the Remedy Selection

The bioavailability of lead in soil is directly related to the *potential* extent and cost of Large Area Lead Site cleanups. Other factors being equal, the higher the bioavailability, the lower the soil lead level indicated by the IEUBK model for the preliminary remediation goal (PRG). As indicated above, a suite of factors is considered in the remedy selection process, but a lower soil lead PRG suggests that a greater volume of dirt would have to be excavated and disposed of to *permanently* meet the health-based cleanup *goal* using currently *proven* remedial technologies. The juvenile swine studies estimated soil lead bioavailability to provide a site-specific refinement of the IEUBK model. Perhaps not surprisingly, the results indicated considerable variability among the sites tested, with some higher, some lower, and some about the same as the agency's default assumption. In terms of the final remedy selection, however, it appears that none of the results will increase the liability of Large Area Lead Site PRPs because EPA deems the cost of removing the contaminated soil to be excessive.

It appears that regardless of whether site-specific experiments indicate a soil lead bioavailability higher or lower than the agency's default assumption, the results will not increase the liability of Large Area Lead Site PRPs because EPA deems the cost of removing the contaminated soil to be excessive.

According to an EPA official, although agency scientists had expected the swine study for the Midvale site to indicate even lower soil lead bioavailability, the difference between the study results (20-25%) and the default assumption (30%) "was significant in

[81] SEGH task force member Rufus Chaney, a USDA researcher, was a strong proponent of using rodents for testing lead bioavailability. Chaney's bioavailability studies with rodents were supported by Dupont. According to an EPA official, DuPont's interest stems from the company's interest in bioavailability of residuals and alternative remediation technologies. EPA was represented on the task force by Richard Cothern (see Powell (1996) for discussion of Cothern's role in the scientific assessment of lead and arsenic in drinking water).

terms of site liability," causing EPA "to raise the proposed action level" (i.e., the soil lead concentration of concern) for the site. For the Bingham Creek Site near Salt Lake City, the experimentally estimated soil lead bioavailability was 19%. According to Murray (1995), the reduced bioavailability value halved the estimated cost of site cleanup from $8 million to $4 million. According to an EPA official, it is questionable whether the slightly reduced soil lead bioavailability estimate for the Leadville site was big enough to make an impact on the risk management decision.

For the Jasper County site, as a result of the unexpectedly higher bioavailability of mining wastes relative to smelter fallout, the estimated geographic distribution of effective exposures to lead via soil was markedly different than initially suspected, according to an official with the Missouri Department of Natural Resources. Consequently, while the swine study did not affect the soil cleanup level, it altered the geographic emphasis of cleanup activities. According to an EPA official, the agency has decided to initiate soil excavation at the site while studying the feasibility of treating the soil with phosphate to reduce the soil lead solubility. According to a Missouri state health official, public demand for additional swine studies to determine the bioavailability of soil lead after phosphate treatment has helped overcome some bureaucratic reluctance to allocating the necessary funds. Although the juvenile swine study did not dramatically reduce the scale of remedial activity, the EPA official considers the study "money well spent. It settled a lot of disputes and saved a lot of arguing regarding what the real number was." Thus, an important contribution of the swine studies to the Jasper County site has been their capacity as a dispute resolution tool (perhaps lowering transaction costs). The results have been accepted as a means for determining the remedial action plan and for assessing the performance of an alternative treatment.

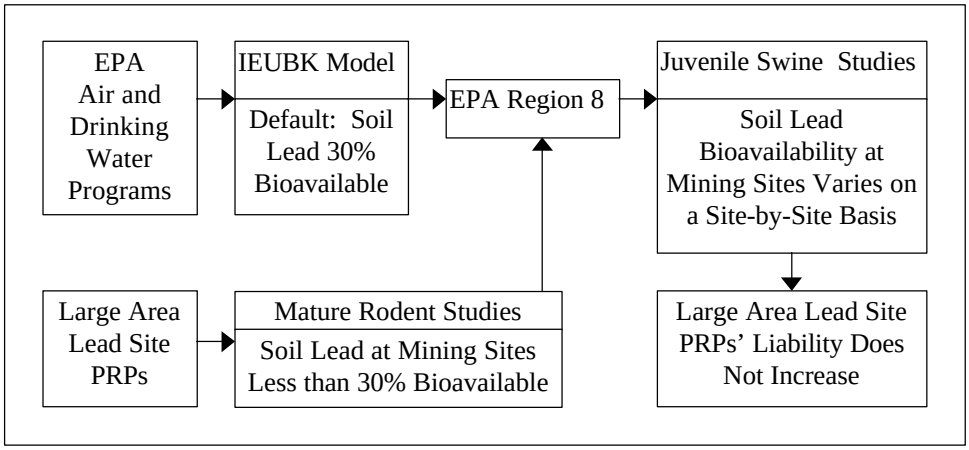
Despite the fact that tests have estimated soil lead bioavailability to be higher than the IEUBK default on some sites, the information has been either beneficial or essentially neutral to Large Area Lead Site PRPs, apparently because EPA feels that the cost of removing the contaminated soil is excessive. Regarding the sites where bioavailability may be higher than the default, an EPA official remarks, "It could potentially lead to higher liability, but I doubt it. All the discussion is about lowering the default." Regarding Large Area Superfund Sites generally, an EPA official concludes, "we will be managing exposure [to environmental hazards] in perpetuity" due to the prohibitive costs of permanently removing the risk. As a result of EPA's implicit policy, the PRPs can take a chance on dramatically reducing their costs by exposing themselves to a relatively modest increase in site liability (EPA can recover the swine study costs), secure in the knowledge that they are not exposing themselves to greater financial risk, even if the science "goes the wrong

5. Concluding Observations

In terms of a fate and transport analogy, this case study again illustrates the mobilization of science (in particular, the IEUBK model) accumulated in different compartments of EPA (the air and drinking water programs) and its assimilation by

another compartment (the CERCLA and RCRA programs). While the soil lead bioavailability studies for Large Area Lead Sites were being done, most of the attention at EPA headquarters at the time was on the high-profile Urban Soil Lead Abatement Demonstration Project (EPA 1995a), according to an OSWER official. Thus, EPA Region 8 also was able to exploit the institutional learning that was occurring in the context of the Urban Soil Lead Abatement study for the purposes of the lead mining sites without the attendant supervision and scrutiny. As in other case studies conducted as part of this project, individual EPA staff (Cohen and Weis) who could bridge media-bound program areas and interface with scientists played key roles in ensuring that the science generated by the agency was put to use in regulatory decisions. Cohen integrated what was known about the effects of lead exposure from various sources (air, drinking water, and soil) that were artificially disaggregated by EPA's organizational structure into three separate programs (the criteria air pollutants, drinking water, and Superfund/RCRA), each with their own parochial concerns. As a trained health scientist on staff at an EPA regional office, Weis was able to design an experiment to *test* the scientific information generated by the regulated community and deposited in the office. These dynamics are illustrated in Figure B-1.

Figure B-1. Fate and Transport Dynamics for Science in the Lead in Soil at Mining Sites Decisions.



Due to the limited number of experts in the health aspects of environmental risk assessment in EPA regional offices, the current management options often may be limited to either absorbing or rejecting such information produced by Potentially Responsible Parties. Relatively few health scientists may be needed if EPA regional offices (and state agencies with delegated responsibility) are expected to uniformly apply the substance-specific toxicological values provided by IRIS and other central databases. If, on the other hand, environmental agencies are expected to more consistently use site-specific scientific information in the contaminated site remedy selection process, they face the challenge of having adequate scientific capacity available (through an appropriate mix of staff and consultants) to manage the production of site-specific data and to critically evaluate the information produced by the regulated community.

REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). 1988. Nature and Extent of Childhood Lead Poisoning in Children in the US: A Report to Congress. US Department of Health and Human Services. July.

Baysinger-Daniel, C. 1995. Cooperation and Coordination among Several Agencies and Private Groups at the Jasper County Superfund Site. Poster Presentation. State and Tribal Forum on Risk-Based Decision Making, St. Louis, MO, November 12-15.

EPA/OSWER (U.S. Environmental Protection Agency/Office of Solid Waste and Emergency Response). 1989. Risk Assessment Guidance for Superfund. Volume I. Human Health Evaluation Manual. EPA/540/1-89/002.

EPA (U.S. Environmental Protection Agency). 1994. Swift/Dingell Q&A Document, Question # 10. Jan. 26. (mimeo).

EPA (U.S. Environmental Protection Agency). 1995a. Urban Soil Lead Abatement Demonstration Project. External Review Draft. ORD/NCEA. EPA/600/R-95/139.

EPA (U.S. Environmental Protection Agency). 1995b. Superfund Administrative Reforms Fact Sheet: May 25.

Hawley, G. 1981. *The Condensed Chemical Dictionary*, 10th ed. Van Nostrand Reinhold Company: NY.

Hemphill, C., M. Ruby, B. Beck, A. Davis, and P. Bergstrom. 1991. "The Bioavailability of lead in Mining Wastes: Physical/Chemical Considerations," *Chemical Speciation and Bioavailability*, vol. 3, pp. 135-148.

Kabata-Pendias, A. and H. Pendias. 1984. *Trace Elements in Soils and Plants*. CRC Press, Inc.: Boca Raton, Fl.

Lagoy, P., I. Nisbet, and C. Schulz. 1989. "The Endangerment Assessment for the Smuggler Mountain in D. Paustenbach, ed. *The Risk Assessment of Environmental and Human Health Hazards: A Textbook of Case Studies*. John Wiley & Sons: NY, pp. 505-525.

Lee, R., W. Wright, and W. Haerer. 1994. Development of a Stochastic Blood Lead Prediction Model. Abstract from 1994 Society for Risk Analysis Annual Meeting.

Murray, B. 1995. Bioavailability of Lead in a Juvenile Swine Model to Support Superfund Mine Site Reclamation. Presentation at the State and Tribal Forum on Risk-Based Decision Making, St. Louis, MO, November 12-15.

NRC (National Research Council). 1993. *Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations*. National Academy Press: Wash., DC.

Powell, M. 1996. The 1991 Lead/Copper Drinking Water Rule and the 1995 Decision Not to Revise the Arsenic Drinking Water Rule: Two Case Studies in EPA's Use of Science. RFF Discussion Paper 97-05. Resources for the Future: Wash., DC.

Probst, K., D. Fullerton, R. Litan, and P. Portney. 1995. *Footing the Bill for Superfund Cleanups: Who Pays and How?* The Brookings Institution and Resources for the Future: Wash., DC.

Probst, K., K. Wernstedt, and R. Hersh. 1996. The Role of Landuse in Superfund Cleanups. Forthcoming report from Resources for the Future: Wash., DC.

Ryan, A. and P. Zhang. 1996. Soil Lead Remediation: Is Removal the Only Option? Research paper by US EPA Risk Reduction Engineering Laboratory, Cincinnati, OH, http://128.6.70.23/html_docs/rrel/ryan.html.

Steele, M., B. Beck, B. Murphy, and H. Strauss. 1990. "Assessing the Contribution from Lead in Mining *Regulatory Toxicology and Pharmacology*, Vol. 11, pp. 158-190.

Tilton, J. 1994. "Mining Waste and the Polluter-Pays Principle in the United States," in R. Eggert, ed. *Mining and the Environment*. Resources for the Future, Wash., DC, pp. 57-84.

Wixson, B. and B. Davies. 1994. "Guidelines for Lead in Soil: Proposal of the Society for Environmental Geochemistry and Health," *Environmental Science and Technology*, Vol. 28, No., 1, pp. 26A31A.

Young, J. 1992. "Mining the Earth," *State of the World 1992*. Worldwatch Institute: Wash., DC, pp. 100-118.

List of Abbreviations

ARCO	Atlantic Richfield Company
ASARCO	American Smelting and Refining Company, Incorporated
CDC	Centers for Disease Control and Prevention
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CERCLIS	Comprehensive Environmental Response Compensation and Liability Information System
ECAO	Environmental Criteria and Assessment Office, EPA
EPA	Environmental Protection Agency
IEUBK	Integrated Exposure Uptake Biokinetic (model for lead in children)
MCL	Maximum Contaminant Level
µg/dL	micrograms per deciliter
NPL	National Priority List
OAQPS	Office of Air Quality Planning and Standards, EPA
OAR	Office of Air and Radiation, EPA
ORD	Office of Research and Development, EPA
OSWER	Office of Solid Waste and Emergency Response, EPA
PbB	blood lead
ppm	parts per million
PRG	preliminary remediation goal
PRP	potentially responsible party
RCRA	Resource Conservation and Recovery Act of 1976
RfD	reference dose
SAB	Science Advisory Board, EPA
SARA	Superfund Amendments Reauthorization Act of 1986
SEGH	Society of Environmental Geochemistry and Health