

ARTICLE

Treating Uncertainty as Risk: The Next Step in the Evolution of Environmental Regulation

by Kevin L. Fast

Editors' Summary: This Article presents an argument that EPA's use of reference concentrations (RfCs) in risk assessments, if upheld by the courts, will dramatically change the nature of health-based regulation. In a background section, the Article summarizes RfC methodology and its origins. The Article compares the traditional standard-setting approach, in which the Agency first identifies a level of exposure that presents a significant risk of harm and then sets a standard below that level, with EPA's RfC-based approach, which allows the Agency to avoid identifying an exposure level that poses a significant risk. Next, using EPA's regulatory decisions about manganese as a case study, the Article illustrates EPA's use of the methodology in risk assessment. In a discussion and analysis section, the Article focuses on how RfCs can be used to manipulate risk-assessment results, and reviews the courts' reaction to the methodology to date. The Article then recommends that members of the regulated community oppose RfC-based risk-assessment methodology in judicial and legislative forums. The Article concludes that EPA's use of the methodology reflects a move toward a zero-risk regulatory paradigm and is a questionable attempt to assign to the regulated community the burden of disproving the existence of any theoretically possible risk to public health.

This Article examines the use of reference concentrations (RfCs) by the U.S. Environmental Protection Agency (EPA) in risk assessment. The Article's thesis is that use of RfCs in risk assessment has provided EPA with a basis for attempting to justify health-based regulation even where no evidence of risk to public health exists, and thereby, to shift to the regulated community the burden of proving the absence of risk in order to avoid regulation. The Article first provides background on risk assessment, addressing the basic requirements for risk-based regulation as they have developed over the last two decades. It then explores the genesis of the RfC methodology, the shifting boundaries between science and policy reflected in application of this methodology, and the regulatory consequences incident to the establishment and application of RfCs as reflected in a recent series of EPA risk assessments involving manganese. Based on this examination, the Article then makes recommendations to limit the misuse of RfCs in risk assessment.

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Background

From Facts to "Quasi-Facts"

More than 200 years ago, John Adams said that "[f]acts are stubborn things, and whatever may be our wishes, our inclinations, or the dictates of our passions, they cannot alter the state of facts or evidence."¹ Facts as they are commonly understood are susceptible to scientific evaluation and provide a potential nexus for differing perspectives to meet on a common ground. It is largely for this reason that administrative agencies of the federal government, given the task of regulating the nation's day-to-day activities in areas where no clear consensus on a proper regulatory course exists, ultimately must justify their regulatory decisions with reference to facts.² What administrative agencies choose to treat as fact, however, and what the courts ulti-

1. John Adams, Argument in Defense of the [British] Soldiers in the Boston Massacre Trials (1770), reprinted in JOHN BARTLETT, FAMILIAR QUOTATIONS 337 (16th ed. 1992).
2. See *Industrial Union Dep't v. American Petroleum Inst.*, 448 U.S. 607, 653, 10 ELR 20489, 20501 (1980) ("The burden was on the Agency" to justify its regulations "on the basis of substantial evidence.").

mately sanction as fact, has strayed increasingly far from what one traditionally might have called a fact. Nowhere is this more apparent than in the context of environmental regulation and efforts to protect the public health.

The government's authority to regulate to protect public health has evolved substantially over time, with each evolutionary step dramatically expanding the reach of governmental authority and the complexity of governmental regulation. Before the advent of modern environmental law, a government's authority to act to protect public health was constrained by the necessity to marshal facts proving the existence of an actual harm amounting to a public nuisance.³ Today, by contrast, government agencies routinely act to protect public health based on what might be called "quasi-fact"—a mix of suspected but not yet substantiated "facts" linked with precautionary policy judgments.

Central to these modern regulatory activities is the concept of "risk." When implementing the precautionary goals established by Congress in a range of environmental statutes, regulatory authorities need not provide proof of actual harm as a predicate to regulation. Rather, they need only present evidence of "a significant risk of harm."⁴ Further strengthening this "preventative" approach to regulation, courts have routinely deferred to an agency's judgment regarding the existence of risks, recognizing that such judgments occur at the "frontiers of scientific knowledge."⁵

For example, the government might conclude that evidence of a physiological change resulting from exposure to a particular material (e.g., a slight fluctuation in blood chemistry) should be deemed indicative of a risk to public health, even though no clear facts link such changes to specific adverse health effects. Precautionary judgments of this sort—the "quasi-facts" to which this Article refers—provide the foundation for much of existing risk-based regulation.

Another good example is the present approach to carcinogenic materials. The federal government routinely acts to reduce exposures to carcinogenic materials based on the now widely accepted belief that exposure to *any* level of a carcinogen presents some risk of developing cancer. This belief is not fact per se, but rather a judgment inferred from an inconclusive and sometimes conflicting scientific data-

base.⁶ Based largely on quasi-facts of this sort, the government has regulated to protect the air we breathe, the water we drink, and the land on which we live.

Despite the already precautionary nature of this long-standing approach to environmental regulation, EPA now seeks to move beyond the bounds of quasi-fact regulation to what might be called a "non-fact" basis for regulation. Extending even further the evolution away from fact, EPA has sought in recent risk assessments to regulate not only where a significant risk can be identified (relying on quasi-facts), but also where *no evidence of a risk exists at all*. More specifically, EPA has asserted the right to regulate based on a "concern" about *potential* risks where the available facts in EPA's judgment fail to prove the absence of risk. The absence of fact, rather than the existence of fact, has become the basis for regulation.

One regulatory tool for this non-fact approach to regulation is the inhalation RfC, which EPA has begun to employ in risk assessments under the Clean Air Act.⁷ An RfC is defined by EPA to be "[a]n estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious noncancer health effects during a lifetime."⁸ It is an estimate of a "safe" level of exposure to a particular air pollutant developed by applying various "uncertainty factors" (ranging from 1 to 10,000) to account for evidentiary gaps identified in the available scientific data for that pollutant. Under the guise of these uncertainty factors—applied in the Agency's discretion as a question of "scientific judgment"—EPA has crafted a methodology that, if ultimately sanctioned as legal, will allow potentially unbounded authority to implement regulatory policies unrelated to real risk.

The Nature of Risk-Based Regulation

Under the typical grant of congressional authority to regulate to protect public health, federal agencies such as EPA must be able to demonstrate as a precondition to regulation that pollution "endanger[s]" the public health.⁹ To show

3. See, e.g., *Georgia v. Tennessee Copper Co.*, 206 U.S. 230 (1907) (court granted injunction on behalf of the state of Georgia where it was "satisfied, by a preponderance of evidence, that the sulphurous fumes cause and threaten danger" to the land, air, and inhabitants of Georgia). For an excellent discussion of the relationship between the common-law "nuisance" doctrine and the evolution of environmental law, see WILLIAM H. RODGERS JR., *ENVIRONMENTAL LAW: AIR AND WATER*, vol. 1, ch. 2 (1986).

4. See, e.g., *Industrial Union Dep't*, 448 U.S. at 641, 10 ELR at 20498; see also *Ethyl Corp. v. U.S. Environmental Protection Agency*, 541 F.2d 1, 12-13, 6 ELR 20267, 20272-73 (D.C. Cir. 1976); *Reserve Mining Co. v. U.S. Environmental Protection Agency*, 514 F.2d 492, 519-20, 5 ELR 20596, 20606-07 (8th Cir. 1975).

5. *Ethyl Corp.*, 541 F.2d at 29, 6 ELR at 20282. See also *Environmental Defense Fund v. U.S. Environmental Protection Agency*, 598 F.2d 62, 80, 8 ELR 20765, 20775 (D.C. Cir. 1978) (EPA has the "latitude to protect against risks that are incompletely understood"); *Natural Resources Defense Council v. U.S. Environmental Protection Agency*, 824 F.2d 1146, 1153, 17 ELR 21032, 21036 (D.C. Cir. 1987) ("Congress chose . . . to deal with the pervasive nature of scientific uncertainty and the inherent limitations of scientific knowledge by vesting in the Administrator the discretion to deal with uncertainty in each case"); *Ausimont U.S.A., Inc. v. U.S. Environmental Protection Agency*, 838 F.2d 93, 96, 18 ELR 20456, 20458 (3d Cir. 1988) ("These questions broaching the frontiers of scientific knowledge highlight the need for testing.").

6. See EPA, *A Descriptive Guide to Risk Assessment Methodologies for Toxic Air Pollutants*, EPA-453/R-93-038, at 3-7 (Sept. 1993) [hereinafter *EPA Guide to Risk Assessment*] ("It was initially believed that a single exposure to a carcinogen, regardless of magnitude, could start the carcinogenic process. This 'nonthreshold' concept of carcinogenesis stated that there is no 'zero-risk' dose in terms of carcinogen exposure. Recent advances in molecular biology, however, indicate cellular mechanisms exist that are capable of repairing the early damage of a carcinogen. The determination of whether a carcinogen exhibits a threshold or not should be based on the latest available biological evidence and be a case-by-case decision. As a practical matter, however, humans are exposed to many carcinogens and the ability to repair damage will vary among individuals within a population. Therefore, unless there is clear evidence to the contrary, cancer dose-response assessments usually assume no threshold.").

7. See EPA, *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry*, EPA/600/8-90/066F (Oct. 1994) [hereinafter *RfC Guidance*].

8. *RfC Guidance*, *supra* note 7, at xxviii ("Glossary").

9. *Ethyl Corp.*, 541 F.2d at 16, 6 ELR at 20274 ("[A]ir pollution must endanger the public health before regulation is justified" under the Clean Air Act). There are, of course, exceptions to this general rule. Section 5(e)(1) of the Toxic Substances Control Act, for example, gives EPA authority to address certain chemicals for which insufficient information exists to allow the Agency to quantify risk. See

endangerment, the Agency is required to present evidence of an exposure level at which there is a risk of specific adverse effects, and to evaluate the magnitude of that risk.¹⁰ As the U.S. Supreme Court has stated with respect to workplace regulation:

[B]efore promulgating any standard, the Secretary must make a finding that the workplaces in question are not safe. But "safe" is not the equivalent of "risk-free." . . . a workplace can hardly be considered "unsafe" unless it threatens the workers with a significant risk of harm.

Therefore, . . . the Secretary is required to make a threshold finding . . . that significant risks are present and can be eliminated or lessened by a change in practices.¹¹

Stated another way, an agency's authority to regulate to protect public health "must be based exclusively upon the [agency's] determination of the risk to health at a particular emission level."¹²

These concepts have been repeatedly upheld by the courts. For example, in the seminal 1976 decision *Ethyl Corp. v. U.S. Environmental Protection Agency*, the U.S. Court of Appeals for the District of Columbia upheld EPA regulations adopted under the Clean Air Act that reduced permissible lead levels in gasoline based on evidence that a blood lead level of 40 micrograms per deciliter ($\mu\text{g}/\text{dl}$) is "indicative of danger to health."¹³ The fundamental issue presented by the petitioners in *Ethyl Corp.* was whether the Agency's authority to regulate to protect public health extended to cases where EPA could not provide "proof of actual harm" to public health from then-existing lead concentrations in gasoline.¹⁴ The D.C. Circuit held that it did.

The specific danger identified by EPA at a blood lead level of 40 $\mu\text{g}/\text{dl}$ was evidence of "increased frequency of hyperactivity among children" and "metabolic interference with heme synthesis in the bone marrow."¹⁵ EPA's regulations requiring reductions in lead concentrations were designed to keep blood lead levels below this threshold, in order to address this risk.¹⁶ As the dissent in *Ethyl Corp.* pointed out, however, whether a blood lead level of 40 $\mu\text{g}/\text{dl}$ represented a level of lead exposure at which adverse health effects first occurred, based on the factual record before the court, was not a known fact. Rather, in the words of the dissent, it "represent[ed] a level above which it would be 'prudent' to prevent further lead exposure."¹⁷

15 U.S.C. §2603(e)(1), ELR STAT. TSCA §5(e)(1). Under such circumstances, however, the oversight role of the judiciary is enhanced. See *id.* §2603(e)(2)(A) & (B), ELR STAT. TSCA §5(e)(2)(A) & (B) (EPA must seek an injunction in order to act where no specific risk can yet be quantified).

10. See, e.g., *Chemical Mfrs. Ass'n v. U.S. Environmental Protection Agency*, 859 F.2d 977, 983, 19 ELR 20001, 20004 (D.C. Cir. 1988) ("[T]he degree to which a particular substance presents a risk to public health is a function of two factors: (a) human exposure to the substance, and (b) the toxicity of the substance.").

11. *Industrial Union Dep't v. American Petroleum Inst.*, 448 U.S. 607, 642, 10 ELR 20489, 20498 (1980).

12. *Natural Resources Defense Council v. U.S. Environmental Protection Agency*, 824 F.2d 1146, 1164, 17 ELR 21032, 21043 (D.C. Cir. 1987).

13. *Ethyl Corp.*, 541 F.2d at 38-40, 6 ELR at 20287-88.

14. *Id.* at 12, 6 ELR at 20272.

15. *Id.* at 39 n.85, 6 ELR at 20288 n.85.

16. *Id.* at 38-43, 6 ELR at 20287-90.

17. *Id.* at 101, 6 ELR at 20324.

Recognizing that the identification of a risk to public health often occurs on the "frontiers of science," where insufficient information exists to demonstrate conclusively that certain activities will endanger the public health, the D.C. Circuit deferred to EPA's judgment regarding the existence of a risk and its magnitude, but stopped short of allowing EPA to act in the absence of fact:

The Administrator may apply his expertise to draw conclusions from suspected, but not completely substantiated, relationships between facts, from trends among facts, from theoretical projections from imperfect data, from probative preliminary data not yet certifiable as "fact," and the like. We believe that a conclusion so drawn—a risk assessment—may, if rational, form the basis for health-related regulations . . .¹⁸

In other words, while recognizing that to protect public health, an agency "must act . . . largely 'on choices of policy, on an assessment of risks, [and] on predictions dealing with matters on the frontiers of scientific knowledge,'" the court held that EPA must also act at least "in part on 'factual issues.'"¹⁹

A similar result occurred several years later in *Lead Industries Ass'n v. U.S. Environmental Protection Agency*,²⁰ where the D.C. Circuit upheld EPA's ambient air quality standard for lead based on evidence presented by EPA of specific "blood lead levels" at which "subclinical effects" suggestive of adverse effects would occur.²¹ EPA determined, for example, that "the lead-related elevation of erythrocyte protoporphyrin" was related to health "impairment," and further that "only when blood lead concentration reaches a level of 30 $\mu\text{g Pb}/\text{dl}$ is this effect significant enough to be considered adverse to health."²² Then, "in order to provide an adequate margin of safety, and to protect special high risk sub-groups" against the risk of these adverse effects, EPA established an ambient level of lead based on a blood lead level of 15 $\mu\text{g}/\text{dl}$, thereby ensuring

18. *Id.* at 28, 6 ELR at 20281.

19. *Id.* at 29, 6 ELR at 20282 (quoting *Amoco Oil Co. v. U.S. Environmental Protection Agency*, 501 F.2d 722, 740-41, 4 ELR 20397, 20406 (D.C. Cir. 1974)). In a scathing dissent to the *Ethyl Corp.* decision, Judge Wilkey wrote:

All true risk assessment is based on facts and nothing else. Those professional risk-assessors, the professional sports gambling fraternity, would smile at any other theory. . . .

Our colleagues apparently find it necessary to legitimize the Administrator playing hunches. They assert, "Danger is a risk, and so must be decided by assessment of risk as well as by proof of facts." Of course the Administrator assesses risk—from the facts as he knows them. *The question here is how much he knows.* To the extent the agency found it necessary to make an "assessment of risk as well as [rely on] proof of facts," the agency was frankly just speculating. . . . It is precisely a devotion to *facts*, not hunches, that distinguishes the professionals from the amateurs in assessing risks; we deem the Administrator to have been intended by Congress to be a "professional."

Id. at 95-96, 6 ELR at 20321 (second emphasis added) (citations omitted). Judge Wilkey ultimately concluded that no "causal connection" had been demonstrated between lead emissions and harm. *Id.* at 110-12, 6 ELR at 20329-30.

20. 647 F.2d 1130, 10 ELR 20643 (D.C. Cir.), *cert. denied*, 101 S. Ct. 621 (1980).

21. *Id.* at 1138-41, 10 ELR at 20645-47.

22. *Id.* at 1144, 10 ELR at 20649.

that blood lead levels for 99.5 percent of the population would remain below 30 µg/dl.²³

The same approach is reflected in the D.C. Circuit's decision in *American Petroleum Institute v. Costle*.²⁴ In this case, the court upheld EPA's promulgation under Clean Air Act §109 of an ambient air quality standard for ozone that was based on evidence that "the 'probable level for adverse effects in sensitive persons is in the range of 0.15-0.25 ppm.'"²⁵ The evidence presented by EPA showed that exposure to ozone at these levels may "impair mechanical function of the lung" and "induce respiratory and related symptoms in sensitive segments of the population."²⁶ According to the court, EPA properly concluded that an ozone standard of 0.12 ppm was sufficiently below the "probable level for adverse effects" that it incorporates "an adequate margin of safety."²⁷ In the court's words, because the EPA Administrator presented "evidence of risk" at these exposure levels, this decision was reasonable.²⁸

These decisions illustrate that the principal check on the Agency's authority to identify risk over the past 20 years has been evidentiary in nature. Courts have ruled, for example, that health-based regulation cannot be supported by mere conjecture or speculation, i.e., that a material "may or may not" cause a risk.²⁹ Rather, an agency must present evidence showing that it has "a more-than-theoretical basis for suspecting that some amount of exposure occurs and that the substance is sufficiently toxic at that exposure level to present an 'unreasonable risk of injury to health.'"³⁰ This was the case in *Ethyl Corp.* (evidence of increased frequency of hyperactivity among children and metabolic interference with heme synthesis in the bone marrow), *Lead Industries Ass'n* (evidence of lead-related elevation of erythrocyte protoporphyrin), and *American Petroleum Institute* (evidence of impairment of the mechanical function of the lung).

EPA's recent use of RfCs in risk assessment is designed to sidestep this evidentiary requirement. Rather than supporting regulation by presenting evidence of a specific level of exposure at which health effects deemed by EPA to be "adverse" might occur, under RfC methodology the Agency instead identifies a conservative "safe" level of exposure derived by applying uncertainty factors whenever EPA is unable to locate sufficient evidence to rule out any theoretically possible effects. Once it has established this "safe" level of exposure (the lower the level, of course, the "safer" it will be), EPA compares the safe exposure level to measured or predicted exposure levels to see if, in the absence of regulation, exposure levels might exceed the safe level of exposure. If they do (or even if they are lower than, but

close to, the safe level), EPA has asserted the right to regulate, even if it cannot identify a specific adverse effect that will or even might occur at the measured or predicted exposure level.³¹

In this way, reliance on RfCs in risk assessment fundamentally alters the nature of the assessment. It shifts to the regulated entity the burden of disproving any theoretically possible public health impact in order to avoid regulation, even where no evidence of such an impact exists. Thus, it dramatically expands the potential reach of EPA's regulatory authority.

To better understand this new EPA approach to public health-based regulation, the following sections discuss the RfC methodology in greater detail, providing information on the genesis of the RfC methodology, the derivation of RfCs, and application of an RfC for manganese in risk assessment.

The Genesis of the RfC Methodology

In 1990, Congress substantially increased EPA's responsibility to control the emission of hazardous air pollutants under the Clean Air Act. Most notably, Clean Air Act §112, as amended, requires EPA to implement a two-tiered regulatory program to control the emission of 189 listed "hazardous air pollutants."³² The list of hazardous air pollutants under §112 includes both "threshold" and "nonthreshold" pollutants. "Threshold" pollutants are those presumed to be safe at or below some level of exposure. "Nonthreshold" pollutants are those, such as carcinogens, for which any level of exposure is presumed to present a risk to health.

In the first tier of regulation, EPA must require for certain stationary sources the installation of technology that reduces emissions of the listed substances to the "maximum degree" that EPA determines is "achievable," i.e., to employ maximum achievable control technology (MACT).³³ The second tier of regulation addresses any "residual" risks to public health remaining after compliance with the technology-based standards. For substances that are known, probable, or possible human carcinogens, Congress directed that additional standards must be issued under this second tier of regulation if the residual risks to public health exceed one in one million for the "individual most exposed" to emissions of the pollutant.³⁴ By contrast, for threshold pollutants such as manganese, Congress simply directed that EPA establish standards that protect public health with an "ample margin of safety."³⁵

EPA originally proposed the RfC methodology for the primary purpose of identifying "residual risk" for threshold pollutants under Clean Air Act §112.³⁶ When EPA's Science Advisory Board (SAB) reviewed the RfC methodology in 1990, it indicated that

23. *Id.* at 1143-45, 10 ELR at 20648-49.

24. 665 F.2d 1176, 11 ELR 20916 (D.C. Cir. 1981).

25. *Id.* at 1187, 11 ELR at 20920.

26. *Id.* at 1182-83, 11 ELR at 20917-18.

27. *Id.* at 1187, 11 ELR at 20920.

28. *Id.*

29. *Chemical Mfrs. Ass'n v. U.S. Environmental Protection Agency*, 859 F.2d 977, 986, 19 ELR 20001, 20005 (D.C. Cir. 1988); see also *Corrosion Proof Fittings v. U.S. Environmental Protection Agency*, 947 F.2d 1201, 1215 n.14, 22 ELR 20037, 20042 n.14 (5th Cir. 1991); *American Petroleum Institute*, 665 F.2d at 1186-87, 11 ELR at 20920 ("[T]he Administrator's conclusion must be supported by the record, and he may not engage in sheer guesswork.").

30. *Chemical Mfrs. Ass'n*, 859 F.2d at 988, 19 ELR at 20007 (emphasis added).

31. See, e.g., EPA, *Fuels and Fuel Additives Registration Regulations*, Notice of Proposed Rulemaking, 57 Fed. Reg. 13168, 13211 (Apr. 15, 1992) (EPA notes that "significant concern might persist even if preliminary estimates of the exposure levels were low relative to the RfC.").

32. 42 U.S.C. §7412, ELR STAT. CAA §112.

33. *Id.* §7412(d), ELR STAT. CAA §112(d).

34. *Id.* §7412(f), ELR STAT. CAA §112(f).

35. *Id.*

36. 55 Fed. Reg. 39321 (Sept. 26, 1990).

Inhalation Reference Concentrations (RfCs) have been developed to serve as baseline health risk estimates for non-cancer effects . . . resulting from exposure to airborne pollutants. . . . It is anticipated that RfCs will be used for [Clean Air Act] regulatory activities as a part of the determination of listing/delisting decisions, lesser quantity cutoffs, and residual risk for non-cancer health effects of air toxics.³⁷

Thus, EPA originally proposed the RfC methodology as a screening tool for purposes of deciding when risks clearly do not exist; the methodology was *not* designed to identify the existence of actual risks or their magnitude. Indeed, EPA scientists have acknowledged that while the RfC can be applied to identify situations unlikely to present a risk, it cannot be applied for estimating "the risk at doses or concentrations between the . . . RfC and the toxicity data" which is available for a particular substance (i.e., the lowest exposure levels at which effects have been observed) because "to date no scientifically validated method for estimating this risk has been found."³⁸

Indeed, the Agency itself has recognized that "[e]xceeding the RfC does not necessarily indicate that a public health risk will occur."³⁹ EPA has stated, for example, that "to estimate a level [of exposure] at which public health risks could be potentially significant . . . it [is] appropriate to consider exposure levels one order of magnitude [10 times] higher than the reference concentration or dose."⁴⁰

What an exceedance of the RfC does indicate, according to EPA, is "the possibility of [] a risk."⁴¹ This "possibility of [] a risk" creates, in turn, "uncertainty," or what EPA has called a "reasonable basis for concern"⁴² and therefore, in EPA's view, a valid basis for regulation.

The Derivation of RfCs

The derivation of RfCs involves two basic components, one substantive and one procedural. The substantive component addresses the methodology by which an RfC is developed from the existing scientific literature for a particular air pollutant. The procedural component addresses who has the responsibility for developing RfCs generally, and the gen-

eral framework in which the RfC development process unfolds.

The Substantive Component

As noted above, the RfC is designed to reflect a "safe" level of exposure. It is, in EPA's words, designed to be a "protective" level of exposure,⁴³ or, stated another way,

the exposure level below which no deleterious effects are expected to occur over a lifetime of exposure. That is, the reference concentration . . . is a level at which the potential for public health effects would be considered negligible.⁴⁴

EPA develops RfCs in two stages. First, EPA undertakes a review of the scientific literature for a candidate substance. Based on that review, EPA identifies the most appropriate animal or human health studies for defining a lowest-observed-adverse-effect-level (LOAEL) or a no-observed-adverse-effect-level (NOAEL) for the substance in question.⁴⁵ These LOAELs or NOAELs are based on an evaluation of physiological changes measured in studies involving exposures of humans or animals to the substance in question. Although considered to be a largely scientific inquiry, EPA's RfC guidance acknowledges that "the distinction between adverse effects and nonadverse effects has been and remains problematic . . . and usually contains an element of scientific judgment."⁴⁶ These effect levels, whether established from animal or human health data, are then adjusted to human equivalent concentrations that reflect exposures to the air pollutant 24 hours a day, 7 days a week for a lifetime.⁴⁷

Second, "uncertainty factors" are applied to reduce the LOAEL or NOAEL to account for "recognized uncertainties in the extrapolations from the experimental data conditions to an estimate appropriate to the assumed human scenario."⁴⁸ As EPA has explained, "[t]he magnitude of the [uncertainty] factors used to determine [RfCs] . . . are based more on conservative estimates than on actual data," and are ultimately "arbitrary."⁴⁹

EPA applies uncertainty factors (1) to convert a LOAEL into a NOAEL (where a NOAEL cannot be established directly from health studies), (2) to account for potentially sensitive subpopulations (e.g., children and the elderly), (3) to account for potential interspecies variability (e.g., establishing a threshold for humans based on animal testing), and (4) to account for limitations in the scientific database

43. *Id.* at 42250.

44. 56 Fed. Reg. at 27363 (emphasis added).

45. Where the scientific literature is not sufficiently robust, EPA will not set an RfC for the chemical until additional studies have been completed. See RfC Guidance, *supra* note 7, §4.1 (discussing minimum data requirements for establishment of an RfC).

46. *Id.* at 4-13 to 4-15. EPA is currently considering a number of innovative techniques for determining LOAELs and NOAELs from the available health literature, including use of the benchmark and Bayesian statistical methodologies. See *id.*, app. A.

47. These adjustments are made in different ways depending on the nature of the study (e.g., animal studies or human studies) from which the LOAEL or NOAEL has been identified. See *id.* §4.3.

48. *Id.* at 4-73.

49. EPA, Principles of Neurotoxicity Risk Assessment, 59 Fed. Reg. 42360, 42397 (Aug. 17, 1994).

37. EPA Science Advisory Board, Review of the Office of Research and Development's Draft Document "Interim Methods for Development of Inhalation Reference Concentrations," Report of the Environmental Health Committee, EPA-SAB-EHC-91-008, at 3 (Apr. 1991). Notably, the "listing/delisting" activities referred to by the SAB arise under Clean Air Act §112(b)(3). That section provides that the EPA Administrator "shall delete a substance from the list [of hazardous substances] upon a showing . . . that there is adequate data on the health and environmental effects of the substance to determine that emissions, ambient concentrations, bioaccumulation or deposition of the substance may not reasonably be anticipated to cause any adverse effects to human health or adverse environmental effects." 42 U.S.C. §7412(b)(3)(C), ELR STAT. CAA §112(b)(3)(C).

38. Michael Dourson, Presentation at EPA Benchmark Dose Workshop (Sept. 28, 1993).

39. EPA, Fuels and Fuel Additives; Waiver Decision/Circuit Court Remand, 59 Fed. Reg. 42227, 42250 (Aug. 17, 1994) [hereinafter Waiver Decision].

40. EPA, National Emission Standards for Hazardous Air Pollutants for Source Categories, 56 Fed. Reg. 27338, 27363 (proposed June 13, 1991) (pmb.).

41. 59 Fed. Reg. at 42259, col. 3.

42. *Id.* at 42260, col. 3.

(e.g., where certain data gaps may exist with respect to a relevant health endpoint).⁵⁰ Uncertainty factors for each of these considerations are selected from a range of one to ten. For example, EPA might apply an uncertainty factor if, in its judgment, a certain health endpoint (e.g., reproduction) has not been adequately evaluated in the scientific literature. EPA might also apply an uncertainty factor to account for the possibility that different species of a metal might have different toxicities. In short, EPA applies uncertainty factors whenever EPA concludes that "definitive" evidence on potential health issues is lacking.

Since one can always perform more studies and further refine the database for a substance, what is "definitive" is ultimately a policy judgment. EPA has cautioned, for example, that in making these "conservative estimates" of safe exposure levels, "[a]ny dose-response assessment, such as the RfC, has inherent uncertainty and imprecision because the process requires some subjective scientific judgment,"⁵¹ and that the multiplication of several uncertainty values of 10 "is likely to yield unrealistically conservative RfCs."⁵² EPA's RfC Guidance also cautions that one must consider the "biological plausibility" of the RfC obtained through the application of uncertainty factors,⁵³ as well as the nature of the effect evaluated in the studies on which the LOAEL or NOAEL is based (i.e., lesser uncertainty factors are applied where a LOAEL or NOAEL reflects a minor or reversible physiological effect, as compared to a physiological effect that is clearly adverse to health).⁵⁴ Once EPA applies these uncertainty factors, however, the resulting value supposedly reflects a safe level of lifetime exposure, even for sensitive subgroups of the population.

As of August 1993, EPA had developed RfCs for 44 different substances.⁵⁵ These RfCs incorporate a wide range of uncertainty factors from one material to the next. Listed below is a breakdown of these RfCs according to the magnitude of the uncertainty factors applied in developing the RfC.

Uncertainty factors	Number of RfCs
1 to 100	10
101 to 999	8
1,000 or higher	26

As reflected in this tabulation, EPA has identified significant levels of uncertainty in the public health database for nearly every chemical it has reviewed.

50. RfC Guidance, *supra* note 7, tbls. 4-8 & 4-9.

51. *Id.* at 4-74.

52. *Id.* at 4-73.

53. *Id.* at 2-44 (directing consideration of whether there is a "[b]iologically plausible relationship between metabolism data, the postulated mechanism of action, and the effect of concern").

54. *Id.* at 2-18 ("Judgments concerning medical or biological significance should be based on the magnitude and class of a particular effect. For example, cough or phlegm production can be considered less important than effects resulting in hospital admissions.").

55. EPA, Regulatory Noncarcinogenic Criteria for the Hazardous Air Pollutants to Be Regulated Under the Air Toxics Program (Aug. 1993).

The Procedural Component

RfCs are developed internally by an EPA workgroup for inclusion in the computer-based information service known as the Integrated Risk Information System (IRIS) database.⁵⁶ The membership of the workgroup is composed exclusively of selected scientists from within EPA's program and regional offices. The only exceptions are scientists from the Agency for Toxic Substances and Disease Registry (ATSDR) and the U.S. Food and Drug Administration who are invited to participate as observers "to assist the Agency in the information-gathering process."⁵⁷

According to EPA, the purpose of the RfC workgroup is "to reach consensus on . . . inhalation RfCs for noncancer chronic human health effects developed by or in support of program offices and the regions."⁵⁸ The "consensus" that the workgroup strives to achieve means that "no member office is aware either of information that would conflict with the . . . RfC, or of analyses that would suggest a different value that is more credible."⁵⁹ To be included on IRIS, the decision of the workgroup on the RfC level must be "unanimous."⁶⁰

The public's involvement in RfC development is limited. For example, the public is provided no direct access to RfC workgroup proceedings. Although EPA has indicated its intention to publish periodically a list of substances for which RfC development activities are either planned or underway,⁶¹ the only mechanism for public input is a cumbersome "three-step process" for submission of information.⁶² Step one involves submission of an "inventory" of all information a submitter wishes to provide to the IRIS "Submission Desk." In step two, EPA identifies from the submission inventory "the information that should be submitted." Step three involves the submission "of the information requested by the Agency."⁶³ Under this procedure, EPA has substantial discretion to shape the nature of the record supporting its actions.⁶⁴

56. See generally EPA, Office of Health and Environmental Assessment, IRIS Background Paper (Feb. 1993) (available from the ELR Document Service, ELR Order No. AD-68) [hereinafter IRIS Background Paper].

57. *Id.* at 8. The ATSDR is an agency of the U.S. Public Health Service, which was created under the Comprehensive Environmental Response, Compensation, and Liability Act, 42 U.S.C. §9604(I), ELR STAT. CERCLA §9604(I).

58. IRIS Background Paper, *supra* note 56, at 8.

59. *Id.* at 9.

60. *Id.*

61. EPA, Integrated Risk Information System [IRIS], 58 Fed. Reg. 11490, 11493-94 (Feb. 25, 1993).

62. See 58 Fed. Reg. at 11492.

63. *Id.* EPA recently reaffirmed use of this information submission process as part of a pilot test program designed to enhance the public's participation in the RfC setting process for a discrete number of existing and candidate RfCs. See EPA, Integrated Risk Information System (IRIS); Announcement of Pilot Program, 61 Fed. Reg. 14570 (Apr. 2, 1996).

64. EPA has taken tentative steps to incorporate a form of external "peer review" for candidate RfCs. The efficacy of this external peer review is open to question, however, since EPA has, in at least one case, paid peer reviewers to review the candidate RfC, and chosen peer reviewers who may have a vested interest in deferring to EPA's judgment on the RfC. In the case of manganese, for example, documents obtained under the Freedom of Information Act show that two of three "external" peer reviewers were the authors of

Because the public is foreclosed from any meaningful participation in the development of RfCs, EPA has recognized that “the entry of a [RfC] in IRIS is not a rulemaking.”⁶⁵ As a result, EPA has also acknowledged that “the entry of a [RfC] on IRIS does *not* make the number legally binding (i.e., the value is *not* entitled to conclusive weight).”⁶⁶ Rather, EPA has explained that EPA itself must justify all aspects of the RfC derivation in each separate regulatory application.⁶⁷ As discussed further below, however, EPA’s public pronouncements on the effect of RfCs does not match the reality of how EPA has attempted to use RfCs.

Use of the RfC as a Risk-Assessment Tool

As originally proposed by EPA, the RfC process can play a valuable screening role for identifying where risks do not exist, and therefore, where regulation is *not* appropriate. Because the RfC is by definition a safe level of exposure (even for potentially sensitive subgroups in the population), exposures at or below the RfC can reasonably be deemed to present negligible risks and can be ignored as a regulatory matter.⁶⁸

Unfortunately, while use as a screening tool might have been the initial purpose of RfCs, this is not how EPA has used them in practice. Rather, EPA has applied RfCs in risk assessment as if they provided a clear delineation between safety and risk, and has concluded that regulation is necessary whenever estimated exposures to the substance fall in the range of, or exceed, the RfC level. This use of RfCs ignores the fact that the identification of an exposure level that entails no appreciable risk and the identification of an exposure level at which risk becomes significant, and therefore warrants a regulatory response, are mutually exclusive tasks. By definition, a single exposure level cannot be both safe and present a significant risk.

Moreover, given the central role of uncertainty factors in the RfC derivation process, it also eliminates what has heretofore been the only check on EPA’s authority to identify risk—namely, the requirement that EPA identify, by reference to evidence of some sort, a specific level of exposure to an air pollutant at which a health effect deemed by EPA to be adverse to some segment of the public (including sensitive subgroups) first occurs. Rather than rely on evidence of an effect at a specific exposure level, EPA

studies EPA relied on in developing the manganese RfC and who were paid by EPA to review the manganese RfC, while the third reviewer was a scientist with another government agency. EPA nevertheless characterized this review as “external” peer review. See ORD’s Response to Public Comments on Ethyl Corporation’s Methylcyclopentadienyl Manganese Tricarbonyl (MMT) Waiver Application, EPA Air Docket A-93-26, No. II-A-15, at 46 [hereinafter EPA Response to Comments].

65. EPA, “Regarding Use of IRIS Values in Superfund Risk Assessment,” OSWER Directive #9285.7-16, at 2 (Dec. 21, 1993).

66. *Id.*; see also Settlement Agreement, General Elec. Co. v. Browner, No. 93-1251 at 2 (D.D.C. Oct. 25, 1993) (“[T]he Agency will consider all credible and relevant information before it” where “an outside party questions IRIS values.”).

67. IRIS Background Paper, *supra* note 56, at 5.

68. Of course, as new information becomes available, an RfC can be refined, potentially triggering different conclusions about the safety of particular exposure levels.

applies uncertainty factors where evidence is lacking on a particular health issue.⁶⁹

Used in this way, the RfC methodology provides EPA with substantial discretion to decide which substances merit regulatory attention and which do not without reference to real risks. This discretion arises in two ways. First, EPA exercises discretion when it establishes an RfC by deciding the magnitude of uncertainty factors to be applied to derive a “safe” level of exposure to a particular substance. While EPA has maintained that application of uncertainty factors is a scientific endeavor, experience suggests that the distinction between science and policy is blurry at best where the question is ultimately, “how safe is safe?”⁷⁰

Second, EPA exercises discretion when it interprets the RfC and applies it to the risk assessment. The Agency may conclude, for example, that exposures at or near the level of the RfC are acceptable in one case, but decide that similar exposures in another case warrant a regulatory response.

A Case Study: Manganese

To appreciate fully the implications of EPA’s use of RfCs in risk assessments, it is helpful to review EPA’s application of RfCs to assessments of the risks posed by manganese. Manganese has been the subject of extensive health studies and repeated regulatory reviews. This attention has been prompted in part by use of manganese as a gasoline additive (known as MMT), and in part by the pervasive nature of manganese emissions in modern industrial societies. The contrast between the conclusions of public health evaluations of manganese conducted well before EPA developed its methodology for deriving RfCs, and EPA’s more recent evaluations, highlights the extent to which use of the RfC as a risk-assessment tool provides EPA with a means to justify public health-related regulatory decisions (ostensibly as a matter of science, not policy) in the absence of any evidence of real risk.

Manganese as a Fuel Additive

MMT is an octane-enhancing fuel additive whose principal ingredient is manganese. It was first developed in the late 1950s, and became a commonly used gasoline additive during the 1970s. Its use became so prevalent that in 1977, EPA determined that beginning in 1978, the fuels used for certification of motor vehicles under the Clean Air Act *must* contain MMT at a concentration greater than 0.1 gram

69. In certain respects, risk assessments involving carcinogens also contain “non-fact” elements. Agencies such as EPA typically extrapolate from high-dose experimental animal data to low-dose human exposure to calculate carcinogenic risk. The process used for this extrapolation is, according to EPA, driven more often by policy considerations than by science. See EPA Guide to Risk Assessment, *supra* note 6, at 3-12. As a result, risk assessments for carcinogens may also be subject to attack on the grounds that they lack a strong factual foundation. The major obstacle to such an attack, however, is that the most widely accepted theory of carcinogenesis provides at least a plausible basis for the “non-fact” presumption that any exposure to a carcinogen presents some degree of risk. See *id.* at 3-6 to 3-9.

70. EPA’s RfC Guidance recognizes that the decision whether to apply uncertainty factors (and to what degree) requires “subjective scientific judgment.” RfC Guidance, *supra* note 7, at 4-74.

manganese per gallon—a level four times higher than the maximum level currently permitted in the United States.⁷¹

In response to this EPA action, the automobile industry approached Congress seeking legislation that would prohibit the use of fuel additives, such as MMT, that were not “substantially similar” to the fuels used in the certification of 1975 motor vehicles—the vehicles that first began operation on unleaded gasoline—unless EPA determined that use of the fuel additive would not cause or contribute to a failure of emission control devices. Responding to this pressure, Congress in 1977 added §211(f) to the Clean Air Act.⁷² As a result, in September 1978, use of MMT in unleaded gasoline was suspended pending approval by EPA for reintroduction into unleaded gasoline under §211(f)(4).⁷³

Despite the exclusion of MMT from the unleaded fuel market, its use in leaded gasoline has been continuous, ultimately peaking at about eight million pounds in 1985. MMT was also used in unleaded gasoline during the 1979 oil crisis under an emergency EPA administrative order,⁷⁴ and has been used continuously as an aftermarket additive in unleaded gasoline.⁷⁵ In total, from 1974 to 1995, nearly 70 million pounds of MMT have been used in gasoline in the United States.

MMT has also been used in Canadian unleaded gasoline for nearly 20 years. Because of its widespread use in Canadian gasoline, Canadian public health authorities have repeatedly reviewed whether use of MMT presents a risk to public health in Canada.⁷⁶ In its most recent assessment completed in late 1994, Health Canada concluded that “all analyses indicate that the combustion products of MMT in gasoline do not represent an added health risk to the Canadian population.”⁷⁷

Manganese as the Subject of Health Testing and Repeated Regulatory Reviews

Manganese is the fifth most abundant metal in the earth's crust, and is naturally present at substantial levels in the ambient air, water, soil, and food.⁷⁸ It is also an indispen-

sible raw material for a wide range of industries in the United States and around the world.⁷⁹

Manganese is an essential nutrient for human health (and for this reason, a common ingredient in vitamin supplements),⁸⁰ but at very high levels of exposure can cause a debilitating neurological disease similar to Parkinson's Disease.⁸¹ The existence of this neurological disease has prompted, in EPA's words, “a wide range of animal studies focused on the neurotoxic effects of this metal,” as well as a “wide range of epidemiological studies” in workers exposed to manganese.⁸² Echoing EPA, the ATSDR noted as recently as September 1995 that “[a] considerable amount of toxicity information is available for manganese, and its major targets of toxicity (i.e., neurologic and reproductive systems) have been identified.”⁸³ With respect to inhalation exposures in particular, this information is, according to the ATSDR, “sufficient to derive a chronic inhalation [minimum risk level for manganese and manganese compounds].”⁸⁴

In addition to the wide range of studies on manganese compounds generally, the manganese emissions resulting from the combustion of MMT have been the subject of considerable study.⁸⁵ Based on the results of studies such as these, EPA conducted numerous evaluations of MMT

71. See EPA, MSAPC Advisory Circular (Jan. 7, 1977) [hereinafter MSAPC Circular].

72. 42 U.S.C. §7545(f), ELR STAT. CAA §211(f).

73. 43 Fed. Reg. 41424 (Sept. 18, 1978). EPA therefore rescinded the requirement that certification fuel contain MMT.

74. EPA, Regulation of Fuels and Fuel Additives, MMT—Suspension of Enforcement, 44 Fed. Reg. 32281 (June 5, 1979).

75. An aftermarket fuel additive is an additive that individual consumers can mix with gasoline.

76. See, e.g., Health and Welfare Canada, Methylcyclopentadienyl Manganese Tricarbonyl (MMT), 78-EHD-21, at 29 (1978). (“[T]here is no evidence to indicate that ambient manganese concentrations from the use of [MMT] as a primary antiknock agent in gasoline . . . would constitute a hazard to human health.”); Health and Welfare Canada, Review of Information on Manganese and the Oxidation Products of MMT Combustion (1988).

77. Health Canada, Risk Assessment for the Combustion Products of Methylcyclopentadienyl Manganese Tricarbonyl (MMT) in Gasoline, at 69 (Nov. 30, 1994).

78. See EPA, Health Assessment Document for Manganese, EPA-600/8-83-013F, at §2.1 (Aug. 1984) [hereinafter HAD]. (“Manganese is a ubiquitous element in the earth's crust, in water and in particulate matter in the atmosphere.”).

79. For a brief overview of the many industrial applications of manganese, see EPA, Locating and Estimating Air Emissions From Sources of Manganese, EPA-450/4-84-007h (Sept. 1985).

80. See Manganese Inhalation Reference Concentration for Chronic Inhalation Exposure (RfC) (Nov. 1, 1993), available in Integrated Risk Information System (IRIS) [hereinafter Manganese RfC] (“Mn is an essential element.”).

81. *Id.*

82. See HAD, *supra* note 78, at 6-24. The HAD contains an overview of the many health studies conducted on manganese, ultimately referencing more than 500 studies. See also Agency for Toxic Substances and Disease Registry, U.S. Public Health Service, Toxicological Profile for Manganese and Compounds, PB93-110781, tpls. 2-1 & 2-2 (July 1992) [hereinafter Manganese Toxicological Profile].

83. Science International, Priority Data Needs for Manganese (Draft), at 39 (Sept. 1995) (report prepared for the U.S. Public Health Service).

84. *Id.* at 23. ATSDR defines a “minimum risk level” as “[a]n estimate of daily human exposure to a chemical that is likely to be without an appreciable risk of deleterious effects (noncancerous) over a specified duration of exposure.” Manganese Toxicological Profile, *supra* note 82, Glossary.

85. See, e.g., Wellington Moore Jr., *Exposure of Laboratory Animals to Atmospheric Manganese From Automobile Emissions*, 9 ENVTL. RES. 274, 282 (1975) (“Exposure of hamsters and rats to automotive emissions containing increased concentrations of Mn particulate resulting from the use of a Mn [gasoline] additive did not produce any histopathological lesions that could be attributed to the increased concentrations of Mn”); Frederick Coulston, *Inhalation Toxicology of Airborne Particulate Manganese in Rhesus Monkeys*, Albany Medical College, EPA Contract 68-02-0710, at 27 (Nov. 1976) (“Clinical data and visual observations of the animals showed that they tolerated the exposure well. In fact, these data and observations did not divulge any effects which would be attributed to manganese.”); Charles Ulrich, et al., *Evaluation of the Chronic Inhalation Toxicity of a Manganese Oxide Aerosol I—Introduction, Experimental Design, and Aerosol Generation Methods*, 40 AM. INDUS. HYGIENE ASS'N J. 238 (Mar. 1979); Charles Ulrich et al., *Evaluation of the Chronic Inhalation Toxicity of a Manganese Oxide Aerosol II—Clinical Observations, Hematology, Clinical Chemistry, and Histopathology*, 40 AM. INDUS. HYGIENE ASS'N J. 322 (Apr. 1979); Charles Ulrich et al., *Evaluation of the Chronic Inhalation Toxicity of a Manganese Oxide Aerosol III—Pulmonary Function, Electromyograms, Limb Tremor, and Tissue Manganese Data*, 40 AM. INDUS. HYGIENE ASS'N J. 449 (May 1979).

and manganese during the 1970s and 1980s. In each of these evaluations, EPA concluded that low-level exposure to manganese from MMT use or from other industrial sources did not present a significant risk to public health.

In 1973, for example, EPA's National Environmental Toxicology Research Laboratory released an evaluation of manganese.⁸⁶ EPA's efforts to remove lead from gasoline prompted the evaluation. As explained by the report,

The toxicity of manganese compounds has received recently renewed attention because of possible widespread use of an organic manganese compound [MMT] as an antiknock additive in gasoline. . . . If lead is removed or reduced considerably in gasoline, the consumption of MMT might increase significantly.⁸⁷

EPA ultimately concluded that the use of MMT in place of lead as an octane-enhancing fuel additive (at concentrations up to 0.25 gram manganese per gallon, or a level eight-fold greater than the current maximum legal level) would *not* present a risk to public health. As explained by EPA, there was "a reasonable margin of safety with use of manganese in gasoline" because "[a]vailable evidence indicates that dosages required to produce . . . adverse effects are several orders of magnitude above those that would be present in the ambient air as a result of even the widespread use of manganese as a gasoline additive."⁸⁸

Then, in 1975, EPA issued two additional manganese evaluations. The purpose of the first report, released in April 1975, was "to summarize the current knowledge of manganese in relation to its effects upon human health and welfare and the environment."⁸⁹ Noting that "[m]anganese (Mn) is among the trace elements least toxic to mammals,"⁹⁰ EPA concluded in the April report that "[t]here is currently no evidence that human exposure to manganese at the levels commonly observed in the ambient atmosphere results in adverse health effects."⁹¹ With respect to the use of MMT in gasoline, the National Environmental Toxicity Research Laboratory concluded in the April report that "[t]here is no evidence that predicted manganese concentrations resulting from the use of [MMT] would result in adverse health effects."⁹²

In October 1975, EPA released a second manganese health evaluation focusing specifically on the use of MMT in gasoline.⁹³ The paper provided a "review [of] both the past and the more recent new information related to an environmental assessment of manganese as a fuel additive."⁹⁴ The findings of this second evaluation mirrored

86. Wellington Moore Jr., *Manganese* (U.S. EPA, National Environmental Toxicology Research Laboratory, June 1973).

87. *Id.* at 1.

88. EPA, *Regulation of Fuels and Fuel Additives: Control of Lead Additives in Gasoline*, 38 Fed. Reg. 33734, 33738 (Dec. 6, 1973).

89. EPA, *Scientific and Technical Assessment Report on Manganese*, PB-242 291, at 1-1 (Apr. 1975).

90. *Id.* at 2-1.

91. *Id.* at 2-3. The April report indicated that "[t]he NASN urban average manganese concentration is less than 0.2 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$), but several cities have annual averages in the 0.5 to 3.3 $\mu\text{g}/\text{m}^3$ range." *Id.* at 2-2.

92. *Id.* at 2-3.

93. See John B. Moran, *The Environmental Implications of Manganese as an Alternative Antiknock*, SAE Paper No. 750926 (Oct. 15, 1975).

94. *Id.* at 3.

those of EPA's April evaluation. The report acknowledged, for example, that with or without MMT in gasoline "[a]mbient air manganese concentrations are likely in all urban and non-urban U.S. areas with higher concentrations evident only near major sources."⁹⁵ With respect to whether manganese emissions would adversely affect public health in this context, the report concluded that "it is likely that the use of manganese in gasoline [does] not pos[e] a direct disbenefit to public health."⁹⁶

Nearly 10 years later, near the time that MMT use in leaded gasoline peaked at eight million pounds annually, EPA initiated a reevaluation of whether to regulate manganese emissions when it issued its Health Assessment Document for manganese.⁹⁷ This document extensively evaluated the health effects of manganese emissions from all anthropogenic sources, including emission from automobiles using MMT.⁹⁸ Among other things, it relied on a 1979 study of the health effects of exposure to MMT combustion products in primates to calculate a NOAEL for humans of 87 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) (or a level nearly 10,000 times higher than EPA's current manganese RfC).⁹⁹ Noting that "the toxicity of numerous manganese compounds has been tested in animals by all common routes of exposure," EPA determined in 1985 that "present ambient air concentrations of manganese do not pose a significant risk to public health and that no regulation directed specifically at manganese is necessary at this time under the Clean Air Act."¹⁰⁰

In short, in every case where EPA evaluated whether low-level exposure to manganese in air presented a significant risk to public health prior to development of the manganese RfC, the Agency concluded that it did not. Regulatory action directed at manganese occurred only after EPA began to rely on the manganese RfC in its risk assessments.

The History of the Manganese RfC and Its Use in Risk Assessment

In 1990, Ethyl Corporation filed for permission from EPA to allow the reintroduction of MMT (under the label "HiTEC@3000 performance additive") as a bulk fuel ad-

95. *Id.* at 7.

96. *Id.* at 52. Although EPA raised questions about the impact of MMT on nonmanganese tailpipe emissions, such as hydrocarbon emissions, EPA conceded that these other collateral impacts of MMT, "are insufficient to constitute a 'significant' adverse effect within the context of Section 211 of the 1970 Clean Air Act Amendments." *Id.*

Notably, a little more than one year after EPA completed its 1975 report on MMT health effects, EPA issued a mobile source advisory circular which mandated that gasoline used for the testing necessary to certify that vehicles complied with emission standards under the Clean Air Act must contain between 0.115 to 0.135 grams manganese per gallon. See MSAPC Circular, *supra* note 71.

97. See HAD, *supra* note 78.

98. See, e.g., *id.* at 2-2, 3-94 ("MMT continues to be used at [approximately] 0.05 g Mn/gal in [approximately] 20% of leaded gasoline" and "[a]bout 15-30% of manganese combusted in MMT-containing gasoline is emitted from the tailpipe").

99. *Id.*, app. at A-3.

100. 50 Fed. Reg. 32627 (Aug. 13, 1985).

ditive in unleaded gasoline in the United States.¹⁰¹ In the ensuing administrative and judicial proceedings, EPA consistently opposed the reintroduction of MMT for use in unleaded gasoline in the United States. The Agency raised one obstacle after another in an ultimately unsuccessful effort to foreclose MMT's use.¹⁰² The final obstacle raised by EPA, and the one EPA may have believed to be least open to judicial restraint, was the Agency's concern that manganese emissions from use of MMT might present some level of risk to public health. As ultimately described by EPA Administrator Browner, her "concern" was based on Ethyl's failure to "rule[] out" the mere "possibility" of risk.¹⁰³

During the four-year administrative proceeding that followed submission of the 1990 waiver application for MMT, EPA exhaustively evaluated all issues pertaining to the additive, including whether manganese emissions from use of MMT might adversely impact public health. The public health evaluation is contained in three separate manganese risk assessments conducted by EPA over the course of the proceeding.

□ *The 1990 Risk Assessment.* In November 1990, EPA released the first manganese risk assessment conducted in connection with Ethyl's waiver application for MMT.¹⁰⁴ In this risk assessment, EPA reported that it had developed, for the first time, an RfC for manganese at a level of 0.4 $\mu\text{g}/\text{m}^3$.¹⁰⁵ Notably, at no time during the 180-day review period for the initial waiver application did EPA provide notice to affected parties or the public in general that it was developing the manganese RfC. Instead, EPA developed the manganese RfC behind closed doors and without public input.

□ *Derivation of the Manganese RfC.* EPA derived the 0.4 $\mu\text{g}/\text{m}^3$ manganese RfC as follows. First, EPA chose to base the RfC on a 1987 study by Dr. Harry Roels evaluating the effects of occupational exposures to manganese.¹⁰⁶ The Roels study examined the effects of manganese exposure at a manganese oxide and salt producing plant in 141 workers exposed to a median manganese exposure level of 970 $\mu\text{g}/\text{m}^3$ for an average of 7.1 years. From the Roels study, EPA identified a LOAEL for manganese in humans of 340 $\mu\text{g}/\text{m}^3$.¹⁰⁷ This

LOAEL reflected small, but statistically significant, differences in the results from a battery of psychomotor tests.

The psychomotor tests used in the Roels study addressed subtle differences in eye-hand coordination, visual reaction time, and hand-steadiness—physiological effects that EPA has concluded elsewhere "may or may not accurately reflect the capabilities of the nervous system and may lead to inaccurate conclusions."¹⁰⁸ In one test, for example, subjects were asked to insert a stylus in holes of various sizes and to hold it steady. Examiners counted the number of times the stylus touched the sides of the holes. If the "hits" for the exposed groups outnumbered those for the control group, then an "effect" was demonstrated.

As EPA has explained in its neurotoxicity assessment guidelines, these "functional neurobehavioral tests are *not* well equipped to distinguish between impairment from neurotoxicity and from non-chemical variables."¹⁰⁹ Among the nonchemical variables recognized by EPA to affect the results of psychomotor testing are age, education, gender, economic status, and the effects of alcohol, drug, or tobacco use.

Notwithstanding the uncertainty as to whether the LOAEL identified by EPA in fact reflected an "adverse effect" of manganese exposure, EPA then applied a 300-fold uncertainty factor, and a three-fold modifying factor (for a total adjustment factor of 900) to reduce the LOAEL to the "safe" exposure level of 0.4 $\mu\text{g}/\text{m}^3$. The uncertainty factors included a 10-fold factor for adjusting the LOAEL of 340 $\mu\text{g}/\text{m}^3$ to a NOAEL of 34 $\mu\text{g}/\text{m}^3$; a 10-fold factor to account for potentially sensitive subpopulations (reducing the RfC to 3.4 $\mu\text{g}/\text{m}^3$); and a three-fold factor to account for the lack of chronic exposure durations in the chosen studies and uncertainty regarding bioaccumulation of manganese (further reducing the RfC to 1.1 $\mu\text{g}/\text{m}^3$).¹¹⁰ The final modifying factor of three (reducing the RfC to 0.4 $\mu\text{g}/\text{m}^3$) was applied to account for the possibility that manganese exposures for the study population in the 1987 Roels study were actually lower than reported in the study, since the manganese processing facility which was the subject of the study had grown in size over the study period.¹¹¹

In short, EPA concluded that a lifetime of exposure at or below 0.4 $\mu\text{g}/\text{m}^3$ of manganese would *not* present an appreciable health risk, even for sensitive subgroups (including children or the elderly) in the population. Having established this "safe" level of exposure, EPA then applied the RfC in the first of its three risk assessments for manganese.

□ *The Focus on Manganese Exposures.* To determine whether use of manganese in gasoline might present a significant risk to public health, EPA estimated likely exposures to manganese with use of MMT. (While abundant data existed showing ambient levels of manganese in the United States and elsewhere, data showing the distribution of personal exposures to manganese for a typical urban population with use of MMT were not then available.) To make these estimates, EPA relied on existing exposure studies for gaseous emissions of carbon

101. EPA grants or denies permission for use of fuel additives such as MMT under authority of §211(f)(4) of the Clean Air Act. 42 U.S.C. §7545(f)(4), ELR STAT. CAA §211(f)(4). The MMT waiver proceedings are contained in three EPA air dockets: A-90-16; A-91-46; and A-93-26.

102. A description of EPA's efforts to block use of MMT is contained in three D.C. Circuit opinions, each of which overturns an EPA decision concerning MMT. See *Ethyl Corp. v. Browner*, 989 F.2d 522, 23 ELR 20689 (D.C. Cir. 1993); *Ethyl Corp. v. U.S. Environmental Protection Agency*, 51 F.3d 1053, 25 ELR 20817 (D.C. Cir. 1995); and *Ethyl Corp. v. Browner*, 67 F.3d 941, 26 ELR 20002 (D.C. Cir. 1995).

103. Waiver Decision, *supra* note 39, at 42227, 42259.

104. EPA, ORD's Comments on the Use of Methylcyclopentadienyl Manganese Tricarbonyl (MMT) in Unleaded Gasoline, EPA Air Docket A-90-16, No. IV-A-1 [hereinafter 1990 Risk Assessment].

105. *Id.* at 6.

106. Harry Roels, *Epidemiological Survey Among Workers Exposed to Manganese: Effects on Lung, Central Nervous System, and Some Biological Indices*, 11 AM. J. INDUS. MED. 307 (1987).

107. 1990 Risk Assessment, *supra* note 104, at 6. The LOAEL included adjustments to reflect continuous 24-hour exposure to manganese for the general population.

108. EPA, *Principles of Neurotoxicity Risk Assessment*, 59 Fed. Reg. 42360, 42382 (Aug. 17, 1994).

109. *Id.* (emphasis added).

110. 1990 Risk Assessment, *supra* note 104, at 6.

111. *Id.*

monoxide (CO) from automobiles and assumed that the distribution of manganese emissions with use of MMT would parallel those for CO.¹¹²

Using this approach, EPA estimated that from 1 to 10 percent of the population would be exposed to a level of manganese exceeding the RfC of 0.4 $\mu\text{g}/\text{m}^3$ (depending on the amount of manganese emitted from vehicles using MMT), with worst-case exposures approaching a level in excess of 1.0 $\mu\text{g}/\text{m}^3$.¹¹³ Combining the exposure estimates with the RfC of 0.4 $\mu\text{g}/\text{m}^3$, EPA ultimately concluded that it could not determine definitively whether use of MMT "will (or will not)" present a risk to public health.¹¹⁴

Of controlling importance to EPA was the fact that its estimate of exposures exceeded the "safe" RfC level of 0.4 $\mu\text{g}/\text{m}^3$ for a small portion of the population when one postulated manganese emissions from vehicles greater than 30 percent of the manganese in the fuel.¹¹⁵ By contrast, EPA's first risk assessment acknowledged that "[d]oses at the RfD/RfC or less are *not* likely to be associated with any significant health risks, and are therefore likely to be protective and of little concern."¹¹⁶ In EPA's view, these "overlapping boundaries" between the exposure estimates and the RfC precluded quantitative risk characterization, which in turn created uncertainty regarding the risks from MMT.¹¹⁷

In 1990, Ethyl voluntarily withdrew its waiver application for MMT to allow EPA and Ethyl to evaluate further the impact of MMT on regulated vehicle emissions, among other issues.¹¹⁸ EPA, therefore, did not have to decide what role the first manganese risk assessment would play in a regulatory decision on MMT. EPA observed, however, that the results of the risk assessment warranted "a discussion within the scientific community . . . [to] achiev[e] a consensus on the uncertainties and data needed to better understand the implications of adding MMT to unleaded gasoline."¹¹⁹

□ *The 1991 Risk Assessment.* In March 1991, EPA convened an international symposium to obtain the "discussion within the scientific community" regarding what information might be necessary to address the uncertainties that had led to application of the 900-fold uncertainty factor.¹²⁰ After three days of meetings, the symposium identified testing that could be done to refine the 900-fold uncertainty factor, but concluded that the need for further studies would be influenced by the extent of exposure to manganese associated with use of MMT.¹²¹

112. *Id.* at 10.

113. *Id.* at 11 & tbl. 2.

114. *Id.* at 15-16.

115. *Id.* at 11. Ethyl Corporation had indicated in the waiver proceeding that a plausible upper bound on manganese emissions from vehicles was 30 percent. EPA Docket A-90-16, No. IV-D-58.

116. *Id.*, attachment 1 at 6 (emphasis added).

117. 1990 Risk Assessment, *supra* note 104, at 11-16.

118. Letter from Ethyl to EPA, EPA Air Docket A-90-16, No. IV-D-212 (Nov. 1, 1990).

119. 1990 Risk Assessment, *supra* note 104, at 16.

120. EPA, Information Needed to Improve the Risk Characterization of Manganese Tetraoxide and Methylcyclopentadienyl Manganese Tricarbonyl, EPA Air Docket A-93-26, No. II-D-58 at 2 (Sept. 11, 1991) [hereinafter EPA Information Needs Document].

121. *Id.* at 15 ("[I]f new exposure studies result in decreasing the exposure assessment to a major degree, further health studies may not be essential.").

This conclusion was the result of additional exposure analyses conducted by Ethyl and its consultants prior to the symposium to refine EPA's initial manganese exposure estimates. The first analysis took advantage of the fact that MMT has been used in unleaded gasoline in Canada for nearly two decades.¹²² Ethyl conducted a study designed to measure manganese exposures over a two-week period for cab drivers (a subpopulation likely, given their work environment, to have elevated exposures to manganese from MMT) and office workers in Toronto, Ontario, during February 1991. The study participants wore personal exposure samplers to allow direct measurement of manganese exposures.

The measured manganese exposures from this study were far lower than EPA's estimates of exposure. For example, the cab drivers, whom EPA agreed represented the upper 4 percent of exposures in Toronto,¹²³ had measured personal manganese exposures ranging from 0.015 to 0.049 $\mu\text{g}/\text{m}^3$, and an average exposure of 0.035 $\mu\text{g}/\text{m}^3$. For the office workers, personal manganese exposures were considerably lower, ranging from 0.002 to 0.048 $\mu\text{g}/\text{m}^3$, and averaging 0.013 $\mu\text{g}/\text{m}^3$.¹²⁴

A second exposure analysis conducted by Ethyl's technical consultants relied on the extensive database for lead exposures from automobile emissions as a basis for predicting manganese exposures. Relying on a study of personal exposures to lead in Los Angeles conducted in 1975,¹²⁵ this analysis assumed that the distribution of manganese exposures from use of MMT was more likely to be similar to the distribution for another metallic fuel additive, lead, than to a gaseous pollutant such as CO, which was the basis for EPA's exposure model. Like the results from the Toronto study, the "lead model" estimates of manganese exposures were far lower than estimates reflected in EPA's 1990 risk assessment. The 99th percentile of manganese exposures using the lead model did not exceed 0.14 $\mu\text{g}/\text{m}^3$, or a level nearly three-fold lower than the manganese RfC.¹²⁶

Finally, another modeling approach known as the South Coast Risk Exposure and Assessment Model produced similar manganese exposure estimates. This model predicted that the maximum manganese exposure in Los Angeles, the urban area having the highest density of automobile traffic in the United States, would not exceed 0.2 $\mu\text{g}/\text{m}^3$, or a level two-fold lower than the manganese RfC.¹²⁷

Based on these refinements to the exposure estimates

122. See Grace Woods, Health Canada, Risk Assessment for the Combustion Products of Methylcyclopentadienyl Manganese Tricarbonyl (MMT) in Gasoline (Nov. 30, 1994).

123. See EPA, Memorandum Regarding "ORD's Comments on Ethyl Corporation's Resubmittal of a Waiver Application for Methylcyclopentadienyl Manganese Tricarbonyl," EPA Air Docket A-91-46, No. IV-B-8, at 4-6 (Dec. 12, 1991) [hereinafter 1991 Risk Assessment].

124. Personal Exposure Sampling—Toronto, EPA Air Docket A-91-46, No. II-D-3, app. 13, attachment 5.

125. ALEX AZAR ET AL., AN EPIDEMIOLOGICAL APPROACH TO COMMUNITY AIR LEAD EXPOSURE USING PERSONAL AIR SAMPLERS, ENVIRONMENTAL QUALITY AND SAFETY, 254-90 (Supp. 1975).

126. Technical Note: Estimating Exposures to Manganese From the Use of HiTEC 3000 in Unleaded Gasoline, EPA Air Docket A-91-46, No. II-D-3, app. 13, attachment 4.

127. Modeling of Manganese Exposure in Mobile Populations, EPA Air Docket A-91-46, No. II-D-2, app. 13, attachment 2.

from EPA's 1990 risk assessment, EPA explained in its first report on the manganese symposium that,

[w]hen viewed in total, . . . [the] results available prior to and as a consequence of the conference on Mn/MMT are sufficient [given inherent and unavoidable uncertainties] to provide quantitative estimates of the inhalation exposure levels that the urban population would experience if MMT were used in unleaded gasoline.¹²⁸

This conclusion reflected the observations of several EPA scientists.¹²⁹

Following the completion of the manganese symposium, EPA completed the second manganese risk assessment in late 1991 as part of Ethyl's second waiver application (filed in July 1991). This new risk assessment accepted the findings of the manganese symposium that EPA's initial exposure estimates for manganese were far too high, and that maximum manganese exposures would not likely exceed one-half of the "safe" level of manganese exposure reflected in the manganese RfC.¹³⁰ This reevaluation, however, did not change EPA's fundamental public health conclusion. EPA's 1991 risk assessment retained the view that it was not possible to conclude "definitively" whether use of MMT "will (or will not)" create a risk to public health.¹³¹ This was despite EPA's statement in the 1990 risk assessment that exposures *below* the RfC "were of little concern."¹³²

Retaining the "we're not sure" conclusion required EPA to modify the focus of the manganese risk assessment. In the first risk assessment, EPA focused on the fact that its estimate of the distribution of manganese exposures showed that a small percentage of the population would be exposed to manganese at levels somewhat in excess of the RfC (or what EPA had termed the problem of "overlapping boundaries").¹³³ Because the new exposure information demonstrated that EPA's initial conclusion regarding "overlapping boundaries" was no longer valid, EPA simply reinterpreted the manganese RfC.

128. EPA Information Needs Document, *supra* note 120. A separate information needs document dated three months later (shortly before EPA's 1992 denial decision) omits any qualitative discussion of the exposure issues contained in the September health recommendations document. See EPA Air Docket A-93-26, No. II-A-16.

129. For example, Ernest Falke, Chief of the EPA Toxics Effects Branch, observed that "[y]ou should . . . cite current ambient exposures to provide perspective. This is especially important in light of the fact that the exposure scientists [at the manganese symposium] estimated that MMT would add minimally to . . . inhalation exposure." EPA Air Docket A-93-26, No. II-D-42, app. 7 (Falke Memorandum). Similarly, J. Michael Davis, a staff scientist with EPA's Office of Research and Development, observed shortly before the symposium that "[e]valuations by EPA . . . suggest that ambient levels of [manganese] may not be increased very much by the introduction of MMT-gasoline." EPA Air Docket A-93-26, No. II-D-58 (Davis MMT Paper). Finally, John Irwin, the EPA staff scientist responsible for the manganese exposure assessment, concluded that use of MMT would "not chang[e] the numbers a great deal over what's there in ambient. This is a very small incremental increase. We aren't talking about doubling what's there already or tripling or something. We're talking about a very small amount of material to something that's already heavily burdened by crustal materials in the atmosphere." EPA Air Docket A-91-46, No. II-D-2, app. 10 (Transcript of Symposium) at 20.

130. 1991 Risk Assessment, *supra* note 123, at 4-6.

131. *Id.* at 7.

132. 1990 Risk Assessment, *supra* note 104, attachment 1, at 6.

133. See *supra* notes 115-17 and accompanying text.

According to this second risk assessment, the manganese RfC is an inexact estimate which contains, by definition, an order of magnitude of uncertainty around the RfC.¹³⁴ This meant, according to EPA, that the "true" RfC might range to a level as low as approximately 0.12 $\mu\text{g}/\text{m}^3$ (or a level 2,700 times lower than the concentrations at which questionable physiological effects had been observed in the Roels study).¹³⁵ At this "lower bound" RfC level, the exposure estimates, even as revised, showed a slight overlap, which EPA proceeded to rely on to maintain its conclusion that a "quantitative" risk assessment was not possible. (According to EPA, those individuals exposed to a level of manganese as low as 0.12 $\mu\text{g}/\text{m}^3$ could potentially be at "some" risk, even though the RfC of 0.4 $\mu\text{g}/\text{m}^3$ was by definition a "safe" level even for sensitive subpopulations.)¹³⁶ EPA also noted that substantial uncertainties existed in the level of likely exposures (notwithstanding the conclusions of the EPA-sponsored symposium), which further contributed to EPA's continuing inability to "quantify" whether a risk would or would not result from use of MMT.¹³⁷

EPA ultimately denied Ethyl's second waiver application on grounds other than public health,¹³⁸ but noted in its decision that "the data needed are unavailable to make a reasonable judgment as to MMT's manganese health effects."¹³⁹ The Agency, therefore, was able once again to avoid a decision regarding how the risk assessment would affect a regulatory decision on MMT use. When Ethyl challenged EPA's waiver denial in court, the court remanded the waiver to EPA to allow the Agency to "cure its own mistake," finding that the basis for the denial was in error.¹⁴⁰

□ *The Revised Manganese RfC.* The remand proceeding commenced in June 1993 and was to result in a new decision in November 1993. With respect to public health, Ethyl argued that EPA's 0.4 $\mu\text{g}/\text{m}^3$ manganese RfC was too low by at least a factor of three. Based on new evidence provided by the production plant, Sedema, that was the location of the 1987 Roels study on which the manganese RfC was based, Ethyl established that the final modifying factor of three applied by EPA lacked justification, and that the RfC should therefore be set at a level no lower than 1.2 $\mu\text{g}/\text{m}^3$.¹⁴¹ Among other

134. 1991 Risk Assessment, *supra* note 123, at 3 & fig. 1.

135. *Id.* fig. 1. EPA noted that, conversely, the RfC might range as high as 1.2 $\mu\text{g}/\text{m}^3$. *Id.*

136. *Id.* at 7.

137. *Id.* at 6-7.

138. EPA denied the waiver based on a concern that the impact of MMT use on regulated tailpipe emissions might vary depending on the manner in which a vehicle was operated. See EPA, Fuels and Fuel Additives; Waiver Application, 57 Fed. Reg. 2535 (Jan. 22, 1992).

139. *Id.* at 2547.

140. Ethyl Corp. v. Browner, 989 F.2d 522, 524, 23 ELR 20689, 20690 (D.C. Cir. 1993).

141. EPA applied the modifying factor of three to account for the possibility that past exposures at the Sedema plant had been lower than the exposures measured during the 1987 Roels study, since production of manganese ore at the plant had increased significantly over the time span covered by the Roels study. 1990 Risk Assessment, *supra* note 104, at 6. The new evidence provided by Sedema showed that production increases occurred in tandem with plant expansions, not as a result of processing additional manganese ore in the same production facilities. Comments on the EPA/ORD Risk Assessment for MMT Use in Unleaded Gasoline, EPA Air Docket A-91-46, No. II-D-3, app. 13, at 7.

things, Ethyl suggested that this higher RfC would be more consistent with other manganese health standards, such as the 1.0 $\mu\text{g}/\text{m}^3$ ambient guideline established by the World Health Organization.¹⁴²

Ethyl noted that at this revised level, there were no "overlapping boundaries" between the manganese RfC and estimated exposures, even when applying the order of magnitude uncertainty factor around the RfC that EPA had applied in the 1991 risk assessment.¹⁴³ The lower bound of an RfC of 1.2 $\mu\text{g}/\text{m}^3$ was 0.3 $\mu\text{g}/\text{m}^3$, or a level significantly higher than the highest modeled estimate of manganese exposures with use of MMT, and nearly 10-fold higher than the highest measured personal exposures to manganese in Toronto. Based on this new information, Ethyl believed that EPA's concerns about public health impacts had been adequately addressed. Indeed, EPA had stated in 1991 in connection with a separate regulatory proceeding that "[t]o estimate a level [of exposure] at which public health risks could be potentially significant . . . it [is] appropriate to consider exposure levels one order of magnitude higher than the reference concentration or dose."¹⁴⁴

The Agency, however, had not yet exhausted its ability to revise its RfC-based risk assessment for MMT. Thirty days prior to the deadline for a new decision on the remanded waiver, EPA notified Ethyl that it had revised the manganese RfC, once again without notice to Ethyl or the public at large.¹⁴⁵ Rather than revising the RfC upward, however, EPA lowered the RfC by nearly a factor of 10 from 0.4 $\mu\text{g}/\text{m}^3$ to 0.05 $\mu\text{g}/\text{m}^3$.¹⁴⁶

To achieve this result, EPA relied on a second study, conducted by Dr. Roels and his colleagues in 1992.¹⁴⁷ The 1992 Roels study evaluated the effects of manganese exposure in 92 workers at a battery production plant who on average were exposed to 948 $\mu\text{g}/\text{m}^3$ of manganese for an average period of 5.3 years. EPA established the new manganese RfC by (1) reducing the LOAEL for manganese in humans from 340 $\mu\text{g}/\text{m}^3$ (based on the 1987 Roels study) to 50 $\mu\text{g}/\text{m}^3$ (based on the 1992 Roels study),¹⁴⁸ and (2) increasing the overall uncertainty factor from 300 to 1,000.¹⁴⁹ Although EPA agreed that the modifying factor

of three to account for potentially lower past exposures at the Sedema plant in the 1987 study was no longer appropriate,¹⁵⁰ the Agency determined that additional "data gaps" had been identified with respect to manganese which required application of additional uncertainty factors for calculating the RfC.

In particular, EPA hypothesized that (1) "longer exposure and/or testing later in life might result in the detection of effects at lower concentrations than is possible after shorter periods of exposure and/or younger workers;"¹⁵¹ (2) "different forms of metals may have different toxic properties (due to different oxidation states, different solubilities, and possibly other factors);" ¹⁵² and (3) "definitive data suggesting that developmental and reproductive effects do not occur at ambient exposure levels was not available."¹⁵³ Exactly why these "data gaps" warranted greater emphasis in 1993 than in 1990 (they existed when EPA first established the manganese RfC) was left unexplained by EPA.

□ *The 1994 Risk Assessment.* After EPA provided notice that it had revised the manganese RfC, a number of industries, including the ferroalloy industry, the iron and steel industry, and the coal industry, complained that the new manganese RfC had not been properly justified in light of available scientific data, and as a result was too low.¹⁵⁴ These interests also complained that EPA had revised the manganese RfC without notice or opportunity for public comment, and urged EPA to withdraw the RfC.

In response, EPA indicated that it would reconsider the manganese RfC as part of its evaluation of Ethyl's waiver application for use of MMT, which Ethyl had withdrawn and refiled for a third time on November 30, 1993.¹⁵⁵ EPA stated that it would consider "any additional data or information pertaining to the health effects of manganese" and, based on that information, "determine whether it is appropriate to make any . . . revisions of the RfC."¹⁵⁶ Moreover, EPA said that it would "decide how the RfC should be used in assessing health effects."¹⁵⁷

The ensuing administrative proceeding focused on three general issues. The first issue addressed the use of the RfC as a risk assessment tool. Recognizing that an RfC is defined as a level of exposure to a chemical that presents no appre-

LOAEL was based on occupational studies involving healthy male workers, EPA applied another 10-fold uncertainty factor to account for potentially sensitive subpopulations (e.g., the elderly or children). The result was to reduce the 5 $\mu\text{g}/\text{m}^3$ level to 0.5 $\mu\text{g}/\text{m}^3$. Third, EPA applied another 10-fold uncertainty factor to address what it called "gaps" in the health data for manganese. This final uncertainty factor reduced the RfC to 0.05 $\mu\text{g}/\text{m}^3$. See Manganese RfC, *supra* note 80.

142. EPA Air Docket A-93-26, No. II-D-8 at 15 n. 37.

143. *Id.* at 15-16.

144. EPA, National Emission Standards for Hazardous Air Pollutants for Source Categories, 56 Fed. Reg. 27338, 27363 (June 13, 1991) (proposed June 13, 1991) (pmbi.).

145. EPA Air Docket A-93-26, No. II-D-57 at 1.

146. Manganese RfC, *supra* note 80. The revised RfC covered "respirable" manganese five microns or less in size.

147. Harry Roels et al., *Assessment of the Permissible Exposure Level to Manganese in Workers Exposed to Manganese Dioxide Dust*, 49 BRIT. J. INDUS. MED. 25 (1992).

148. EPA concluded that both the 1987 and 1992 Roels study supported the new manganese RfC because the new RfC was based on inhaling manganese five microns or less in size, whereas the 1990 RfC was based on inhaling all sizes of airborne manganese. See Manganese RfC, *supra* note 80. EPA estimated that approximately 20 to 25 percent of the manganese exposures in the 1987 Roels study occurred in the size range of five microns or less, which would entail a corresponding LOAEL for the study in that size range from 60 to 80 $\mu\text{g}/\text{m}^3$, or roughly the same LOAEL resulting from the 1992 Roels study. *Id.*

149. Based on its evaluation of psychomotor tests conducted in the 1992 Roels study, EPA identified a LOAEL of 50 $\mu\text{g}/\text{m}^3$ for the general population. Then, because EPA had calculated only a LOAEL from the Roels study, it applied an uncertainty factor of 10 to convert the LOAEL (50 $\mu\text{g}/\text{m}^3$) into a NOAEL (5 $\mu\text{g}/\text{m}^3$). Second, because the

150. ORD's "Reevaluation of Inhalation Health Risks Associated With Methylcyclopentadienyl Manganese Tricarbonyl (MMT) in Gasoline," EPA Air Docket A-93-26, No. II-A-17, at 4 (July 1, 1994) [hereinafter 1994 Risk Assessment].

151. Waiver Decision, *supra* note 39, at 42227, 42241.

152. *Id.* at 42242.

153. *Id.* at 42241.

154. See, e.g., EPA Air Docket A-93-26, No. II-D-99 (Letters from the noted industries).

155. EPA, Fuels and Fuel Additives; Extension of Time and Finding Concerning Fuel Additive Waiver Application, 58 Fed. Reg. 64761 (Dec. 9, 1993).

156. *Id.* at 64764.

157. *Id.*

cial risk of adverse health effects for a lifetime of exposure (even for sensitive subpopulations), a number of commenters argued that the various RfCs identified by EPA could not, standing alone, be applied as a basis for identifying the existence of a significant risk of harm to public health.¹⁵⁸ These commenters explained that two elements are essential to demonstrate public health risk: (1) there must be some meaningful level of exposure to the substance in question as a result of its use, and (2) the substance must be sufficiently toxic at the predicted exposure levels to present a risk to public health.¹⁵⁹ From this perspective, the commenters explained that manganese, as an essential nutrient, had not been shown to be in any way threatening to public health at the extremely low levels of exposure likely to occur with use of Ethyl's additive. More specifically, these commenters observed that even maximum predicted manganese exposures—including those derived by EPA—either fell below or within the range of the RfCs identified by EPA to present no appreciable risk, and therefore could not by definition present a significant public health risk.

Second, commenters questioned the biological plausibility of relying on an RfC for manganese as low as $0.05 \mu\text{g}/\text{m}^3$ as an indicator of potential risk, explaining in particular that manganese is pervasive in the human diet. Under EPA's existing reference dose (RfD) for manganese,¹⁶⁰ a person's diet can include up to approximately 9,800 μg of manganese per day without appreciable risk (normal dietary consumption of manganese varies from 2,000-9,000 μg per day).¹⁶¹ In fact, the Food and Drug Administration (FDA) recommends a *minimum* daily intake of manganese of 3,500 μg .¹⁶²

With normal dietary consumption of 2,000-9,000 μg per day, manganese actually absorbed into the body on a daily basis ranges from 60-900 μg .¹⁶³ Stated in other terms, the amount of manganese taken up by a person's body in the diet can safely double, triple, or even quadruple from day to day, as that person consumes different quantities of foods or beverages which naturally contain high levels of manganese (e.g., nuts, bananas, tea, vitamin pills). These variations of manganese in the body have no known adverse effect on human health.¹⁶⁴

Since the human body is accustomed to processing large amounts of manganese (and indeed depends on manganese for survival), commenters explained that systemic uptake of manganese from the inhalation pathway even at levels above the RfC would be no cause for concern. For example,

158. See, e.g., Comments of Ethyl Corporation, EPA Docket A-93-26, No. II-D-98, at 4.

159. See *Chemical Mfrs. Ass'n v. U.S. Environmental Protection Agency*, 859 F.2d 977, 983, 19 ELR 20001, 20004 (D.C. Cir. 1988).

160. An RfD is a level of oral consumption of a chemical which is without appreciable risk over a lifetime.

161. See Manganese Reference Dose for Chronic Oral Exposure (RfD), available in IRIS [hereinafter Manganese RfD].

162. Food and Drug Administration, Food Labeling: Reference Daily Intakes, 59 Fed. Reg. 427 (Jan. 4, 1994).

163. This is based on EPA's conclusion that "[i]n the normal adult, between 3 and 10% of dietary Mn is absorbed." Manganese RfD, *supra* note 161.

164. See Agency for Toxic Substances and Disease Registry, U.S. Public Health Service, Toxicological Profile for Manganese and Compounds at 3 (July 1992) ("The amount of manganese in normal diet (about 2,000-9,000 $\mu\text{g}/\text{day}$) seems to be enough to meet your daily need, and no cases of illness from eating too little manganese have been reported in humans.")

at an exposure level of $0.2 \mu\text{g}/\text{m}^3$ (four times the level of the $0.05 \mu\text{g}/\text{m}^3$ RfC), systemic uptake from the inhalation pathway would be only about 1 microgram per day.¹⁶⁵ These commenters explained that there was simply no evidence to suggest that inhaled manganese is hundreds of times more toxic than ingested manganese. Indeed, this observation was seconded by a scientist from the FDA, who explained that "[t]he 1000-fold difference between the RfD and the RfC [exposure levels] is not consistent with current bioavailability estimates."¹⁶⁶

The third area of debate focused on the level of the RfC itself. Scientists from ICF Kaiser argued, for example, that EPA had been too aggressive in applying uncertainty factors for the manganese RfC and urged EPA to revise the RfC upward to a level in the 3 to 5 $\mu\text{g}/\text{m}^3$ range.¹⁶⁷ Other commenters, including the Environmental Defense Fund, suggested that EPA had been too lax in developing the manganese RfC and urged EPA to drop the RfC even lower to assure a truly "safe" level of exposure to manganese.¹⁶⁸

When EPA ultimately released its third manganese risk assessment, it agreed that an alternative manganese RfC at a level two- to four-fold higher than the 1993 manganese RfC (or an RfC in the 0.09 to $0.2 \mu\text{g}/\text{m}^3$ range) had "greater scientific strengths" than the 1993 RfC since it was based on "[t]he techniques judged by EPA scientists to be most appropriate."¹⁶⁹ Nevertheless, EPA's 1994 risk assessment continued to rely on the "official" $0.05 \mu\text{g}/\text{m}^3$ RfC level since it had been "verified" by EPA's scientists and placed on IRIS.¹⁷⁰ And as further justification for its continued reliance on the $0.05 \mu\text{g}/\text{m}^3$ RfC, EPA asserted that the difference between an RfC of 0.05 and $0.2 \mu\text{g}/\text{m}^3$ was "not scientifically meaningful."¹⁷¹

As to the comments on the biological plausibility of the manganese RfC and the application of the uncertainty factors, EPA rejected them in their entirety. According to EPA, nothing in the information provided by Ethyl or other commenters eliminated the "possib[ility] that a significant fraction of even small amounts of inhaled manganese would be able to reach target sites in the brain."¹⁷² More specifically, EPA explained that Ethyl had not proven the implausibility of the manganese RfC because the studies cited by Ethyl and others "provide[] little or no useful information on a comparison of inhalation and ingestion routes of Mn exposure."¹⁷³

In rejecting these comments, however, EPA made no

165. Based on an RfC of $0.05 \mu\text{g}/\text{m}^3$, the systemic uptake of manganese would be 0.3 μg per day, assuming a respiration rate of 20 cubic meters of inhaled air per day, and assuming that 30 percent of inhaled particles would be absorbed from the lung. See Report of ICF Kaiser, EPA Air Docket A-93-26, No. II-D-85, at 20-21.

166. See Letter from Clark Carrington to Michael Davis, EPA Air Docket A-93-26, No. II-D-58 (July 8, 1991).

167. Report of ICF Kaiser, EPA Air Docket A-93-26, No. II-D-85, at 53-57.

168. *Id.*, No. II-D-68 (Comments of the Environmental Defense Fund) at 20-21 ("[T]he 1993 RfC is more than an order of magnitude too high.")

169. Waiver Decision, *supra* note 39, at 42227, 42253 n.62, 42259.

170. *Id.* at 42259.

171. EPA Response to Comments, *supra* note 64, at 3; see also 1994 Risk Assessment, *supra* note 150, at 29.

172. EPA Response to Comments, *supra* note 64, at 16.

173. *Id.* at 17.

attempt to explain how transport of small amounts of inhaled manganese to the vicinity of the brain could have an adverse effect on the brain due to inhalation, when far larger amounts of manganese will already be in the blood in the vicinity of the brain as a result of the normal dietary intake of manganese. Nor did EPA explain how the extremely small "delivered doses" to the brain that might be possible at the level of the RfC (i.e., less than 1 μg per day) could create a risk of adverse health effects given that the analogous delivered doses to the brain from inhalation would be on the order of 160-190 μg per day for EPA's range of LOAEL values (26-32 $\mu\text{g}/\text{m}^3$) and 60-120 μg per day for the range of NOAEL values (10-20 $\mu\text{g}/\text{m}^3$). Instead, EPA simply reassured that manganese "may" be more toxic when inhaled than when ingested.¹⁷⁴

Having reduced the RfC by nearly a factor of 10, EPA's 1994 risk assessment for MMT predictably produced the "overlapping boundaries" between the RfC and estimated exposures originally identified by EPA in 1990. As part of the 1994 risk assessment, EPA conducted a new manganese exposure assessment relying on new information derived from the PTEAM study conducted in Riverside, California, in 1990.¹⁷⁵ On the basis of this new exposure assessment, EPA concluded that maximum long-term exposures to manganese with use of MMT could reach a level as high as 0.2 $\mu\text{g}/\text{m}^3$ in high-density urban traffic areas.¹⁷⁶ (Notably, this is approximately the same maximum exposure level which resulted from Ethyl's refinements of EPA's initial exposure assessment.)¹⁷⁷

Comparing the new manganese RfC to these revised manganese exposure estimates, EPA explained that:

If MMT were utilized in unleaded gasoline at the specified concentration in [worst-case] urban areas . . . , the Agency's exposure assessment predicts that the exposures of forty to fifty percent of the population in such areas to airborne manganese levels would exceed the present verified RfC of 0.05 $\mu\text{g}/\text{m}^3$, and the exposures of five to ten percent would exceed manganese levels exceeding a potential alternative RfC of 0.1 $\mu\text{g}/\text{m}^3$. *Although it is impossible to state whether a health risk would definitely exist at the projected exposure levels, neither can the possibility of such a risk be ruled out.*¹⁷⁸

On this basis, EPA concluded that it had a "reasonable basis for concern" about MMT and that "the burden of resolving [these] uncertainties should fall on the waiver applicant

rather than the public."¹⁷⁹ EPA therefore prohibited the use of manganese as a fuel additive pending resolution of these "uncertainties"—a prohibition that the D.C. Circuit ultimately overturned on other grounds.¹⁸⁰

Discussion and Analysis

EPA's use of the manganese RfC in the risk assessments for MMT is instructive for three reasons. First, it illustrates how an RfC-based risk assessment has been misused by EPA to avoid presenting, as a predicate to regulation, evidence of an identifiable risk to some segment of the population at some defined level of predicted exposures. Second, it shows how the RfC methodology can be manipulated by EPA to achieve a particular regulatory goal through arbitrary application of uncertainty factors in response to less-than-complete knowledge. This manipulation is made possible, in part, by the exclusion of the public from the RfC-setting process. Third, it raises concerns regarding whether regulation based on the RfC process will receive meaningful judicial review, given the complex, technical, and judgmental characteristics of the process. Each of these issues is addressed more fully below.

Uncertainty as a Basis for Regulation

Reliance on the RfC as a risk-assessment tool shifts the focus of the risk assessment away from an assessment of what is or may be known about a particular air pollutant to what is *not* known about the air pollutant. By relying on the RfC in risk assessment, EPA seeks to shift the burden of justifying proposed regulatory action away from itself and to place that burden on the regulated entity—as a burden to demonstrate why regulation is *not* necessary. Under an RfC-based risk assessment, if the regulated entity cannot demonstrate why regulation is unnecessary by proving the absence of risk, the result is regulation by default. This in turn allows EPA to present a purportedly "rational" case for regulation at much lower exposure levels.¹⁸¹

179. *Id.* at 42260.

180. *Id.* at 42261. The D.C. Circuit acted in *Ethyl Corp. v. U.S. Environmental Protection Agency*, 51 F.3d 1053, 25 ELR 20817 (D.C. Cir. 1995).

181. In connection with recent efforts to develop sediment quality criteria under the Clean Water Act, which are conceptually similar to RfCs, EPA commented that:

Responding to environmental problems with corrective action frequently requires proving that something negative has occurred. "Innocent until proven guilty," the standard of the American judicial system, works well for many legal activities; but applying this logic to environmental protection efforts (an activity is environmentally acceptable until it is proven unacceptable) presents a unique set of strengths and limitations. Regulatory agencies frequently are called upon to prove environmental or human health degradation has or could occur prior to taking any corrective or preventive action. Scientifically sound and legally defensible measures that demonstrate potential or actual impacts are imperative. Fundamental to any effort to ensure environmental protection is to define the party upon whom the burden of proof lies. Chemical specific criteria are one tool developed by regulatory and non-regulatory agencies that is frequently used to meet the burden of proof requirements. Criteria define when a release of a substance into the environment is acceptable and when the release is causing or has potential to cause adverse impacts on aquatic life, wildlife, or human health.

174. EPA Response to Comments, *supra* note 64, at 16. Notably, as early as August 1990, at least one EPA scientist apparently agreed with Ethyl that manganese toxicity must be assessed in light of typical daily exposures to manganese from dietary sources. In the words of this EPA scientist, "with average adult uptakes [of manganese] on the order of 200 $\mu\text{g}/\text{day}$ from diet, it does not seem likely that MMT will significantly impact [adults]." Memorandum from Joel Schwartz to Richard Morgenstern (Aug. 10, 1990). Until June 1996, EPA refused to release this EPA assessment on the grounds that its release might ultimately "confuse" the public. See *Ethyl Corp. v. Environmental Protection Agency*, 25 F.3d 1241, 24 ELR 21591 (4th Cir. 1995) (litigation arising under the Freedom of Information Act).

175. E.D. Pelizzari et al., Particle Total Exposure Assessment Methodology (PTEAM): Riverside, California Pilot Study—Volume I [Project Summary], EPA Report No. EPA/600/SR-93/050 (Mar. 1992).

176. EPA Response to Comments, *supra* note 64, at 40-41; 1994 Risk Assessment, *supra* note 150, app. B at B-37 to B-38.

177. See *supra* notes 122-127 and accompanying text.

178. Waiver Decision, *supra* note 39, at 42227, 42259 (emphasis added).

Use of the RfC as a risk-assessment tool frees EPA from even the limited evidentiary constraints of the quasi-fact risk-assessment paradigm, in which two initial tasks remain paramount: (1) an evaluation of the available health information for a particular air pollutant to identify the level of exposure that entails, in EPA's judgment, an identifiable adverse impact on health for some segment of the population;¹⁸² and (2) a comparison of the exposure level at which these adverse effects may result with the likely exposures to the pollutant in question.¹⁸³ In the RfC-based risk assessment, the focus is on identification of gaps in the available scientific database and the "uncertainty" that those gaps present. EPA then relies on this uncertainty to establish safe exposure levels that have no direct relationship to what is known about the pollutant at issue, placing the burden on the regulated entity to justify why regulation is not needed whenever exposures exceed (or perhaps even approach) a conservatively derived "safety" threshold.

EPA's regulatory approach to manganese illustrates the implications of this shift in the nature of the risk assessment. Focusing exclusively on facts about manganese, the relative safety of manganese as a fuel additive would seem apparent. Maximum predicted exposures to manganese in the most densely populated urban areas in the United States (which are also the areas most dependent on motor vehicles) are 150- to 250-fold lower than the lowest levels at which effects of any kind have been identified (assuming, as noted above, that these so-called effects are both real and adverse). These predicted exposure levels are also 50- to 100-fold lower than the exposure level at which no observed effects would be expected in the general population.¹⁸⁴ (Notably,

EPA, Briefing Report to the EPA Science Advisory Board on the Equilibrium Partitioning Approach to Predicting Metal Bioavailability in Sediments and the Derivation of Sediment Quality Criteria for Metals, EPA 822-D-94-002, at 1 (Dec. 1994).

182. Natural Resources Defense Council v. U.S. Environmental Protection Agency, 824 F.2d 1146, 1164, 17 ELR 21032, 21042 (D.C. Cir. 1987).

183. See, e.g., Chemical Mfrs. Ass'n v. U.S. Environmental Protection Agency, 859 F.2d 977, 983, 19 ELR 20001, 20004 (D.C. Cir. 1988) ("[T]he degree to which a particular substance presents a risk to public health is a function of two factors: (a) human exposure to the substance, and (b) the toxicity of the substance.").

Although EPA may then apply its judgment to reduce permissible exposure levels below levels at which adverse effects have been identified (i.e., to create a "margin of safety"), the starting point for this effort remains EPA's identification of a level of exposure that presents a significant risk to one or more segments of the population—a risk against which EPA's regulatory program is designed to protect. For example, when EPA established national ambient air quality standards, it first identified "credible scientific evidence suggesting the existence of adverse health effects" within a range of exposure levels and then set standards at the low end of the exposure ranges to provide a safety margin. See, e.g., EPA, Revisions to the National Ambient Air Quality Standards for Particulate Matter, 52 Fed. Reg. 24634, 24641-45 (July 1, 1987); EPA, Review of the National Ambient Air Quality Standards for Carbon Monoxide, 50 Fed. Reg. 37484 (Sept. 13, 1985); EPA, Revisions to the National Ambient Air Quality Standards for Photochemical Oxidants, 44 Fed. Reg. 8220 (Feb. 8, 1979); EPA, National Primary and Secondary Ambient Air Quality Standards [for Sulfur Oxides], 38 Fed. Reg. 25678 (Sept. 14, 1973).

184. As noted above, actual measured exposures to manganese are even lower. EPA has indicated that cab drivers, by virtue of their occupation, experience a level of exposure to automobile pollutants that puts them in the upper 4 percent of manganese exposures for a typical urban population. See *supra* notes 122-24 and accompanying text. Assuming this is so, personal exposures to manganese for this highly exposed subgroup of the population in Toronto, Ontario, where MMT has been in use for nearly 20 years, are more than 850-fold lower than the lowest level of exposures at which a pur-

EPA has stated in other contexts that exposures at the level of the NOAEL for a particular pollutant generally do not present a health risk to the majority of the population.)¹⁸⁵

In contrast to these facts about manganese, EPA's new RfC methodology allowed EPA to assume—without any evidentiary support and despite the conclusions of every EPA risk assessment for manganese conducted prior to development of the RfC process¹⁸⁶—that (1) there is a segment of the population which is sensitive to manganese exposure (despite the essentiality of manganese to human health); and (2) the NOAEL for this sensitive subgroup falls in the 0.5 to 2 $\mu\text{g}/\text{m}^3$ range. EPA further assumed that with respect to this purportedly sensitive subgroup, some or all of the individuals in the group are sensitive to certain unidentified species of manganese compounds, sensitive with respect to unidentified potential reproductive or developmental effects, and might show unidentified effects based on longer periods of manganese exposure. For this subset of the sensitive population, EPA assumes that the NOAEL falls in the 0.05 to 0.2 $\mu\text{g}/\text{m}^3$ range (i.e., the range of RfC values for manganese). These suppositions provide the sole basis for EPA's "concern" about MMT use.

That EPA's concern about manganese lacked a solid factual foundation is also confirmed by a set of EPA briefing documents released by the Agency in June 1996. As might be expected, the briefing documents, which were prepared by EPA staff for review by various senior Agency officials during October 1990 and December 1991, contain arguments both for and against MMT, even on the issue of public health. To support approval of Ethyl's MMT waiver, the briefing documents contain the following factual conclusions:

• *October 1990*: After noting that "[t]he neurological effects of manganese toxicity occur at high-level exposure," the document states that "[a]fter introduction of MMT, manganese levels will still be about one-tenth of one percent of the levels associated with observed adverse effects."¹⁸⁷

• *December 1991*: "Modeling/monitoring data show that exposure to Mn will be below the RfC; no clear evidence exists that MMT will create [a] public health risk."¹⁸⁸

By contrast, EPA's argument supporting denial of the waiver application on public health grounds is based on a policy preference:

• *October 1990*: "While exposure levels are expected to be low, the exposed population is expected to be very large. . . . Given [the] severe adverse health effects [of manganese] at higher levels, widespread

ported effect has been identified, and nearly 300-fold lower than the lowest level of manganese exposures at which no observed effects would be expected in the general population.

185. See EPA, Mercury Study Report to Congress (Draft) 6-1 (Aug. 1995) ("Consumption of mercury equivalent to the NOAEL is predicted to be without harm for the majority of a population.").

186. See *supra* notes 85-100 and accompanying text.

187. EPA, Field Operations and Support Division, MMT in Unleaded Gasoline, Briefing of Deputy Administrator Habicht (Oct. 16, 1990) [hereinafter 1990 EPA Briefing Document] (emphasis added).

188. EPA, Field Operations and Support Division, MMT in Unleaded Gasoline, Briefing of the Administrator (Dec. 1991) [hereinafter 1991 EPA Briefing Document] (emphasis added).

use of MMT even at low levels should not proceed prior to further health and exposure studies."¹⁸⁹

• *December 1991*: The "Agency cannot conclude that airborne Mn from MMT will not cause a public health risk" and, therefore, "a conservative approach is warranted."¹⁹⁰

Of particular note, the arguments supporting denial of the MMT waiver application do not dispute the fact that manganese exposure levels with use of MMT would remain 1,000-fold lower than the lowest exposures at which adverse effects have ever been observed for manganese.

In short, EPA uses RfCs in risk assessment to support regulatory action even where there is no evidence of risk, as in the case of manganese. In RfC-based risk assessments, the concept of uncertainty displaces evidence as the driving force for regulatory action. The ultimate consequence is a shift in the burden of proof for regulatory action. Rather than a requirement that government prove the existence of significant risks as a justification for regulation, regulated entities must prove safety to avoid regulation.

The RfC and Manipulation of Risk Assessments

In addition to focusing on what is *not* known about an air pollutant as a basis for regulatory decisions, use of the RfC as a risk-assessment tool affords EPA broad latitude to respond to changes in the relevant scientific database underlying the risk assessment. The nature of this discretion is readily apparent from a review of the manganese risk assessments. When new information demonstrated that EPA's first risk assessment incorporated unrealistically high estimates of manganese exposure and that lower exposure estimates fell below the then-existing manganese RfC of 0.4 $\mu\text{g}/\text{m}^3$, EPA simply suggested that, by definition, the manganese RfC is not really a single value (i.e., 0.4 $\mu\text{g}/\text{m}^3$), but rather, a range of values (i.e., 0.12 to 1.2 $\mu\text{g}/\text{m}^3$). Recast in this fashion, EPA was able to assert that the full range of exposures might not remain below the RfC (the problem of "overlapping boundaries") and that, therefore, the resulting exposures might not be entirely safe.

When new information demonstrated that EPA's use of a modifying factor of three to lower the manganese RfC from 1.2 $\mu\text{g}/\text{m}^3$ to 0.4 $\mu\text{g}/\text{m}^3$ was inappropriate and that the RfC should be 1.2 $\mu\text{g}/\text{m}^3$, EPA agreed that the modifying factor was inappropriate, but responded by identifying new "data gaps" thereby adding new uncertainty factors in deriving the manganese RfC. This allowed EPA to lower the manganese RfC to 0.05 $\mu\text{g}/\text{m}^3$ rather than to increase the RfC in response to new scientific data addressing the originally identified uncertainty. The problem of "overlapping boundaries" between the RfC and estimated manganese exposures therefore remained.

Finally, where EPA's own analyses subsequently showed that the revised RfC was too low and should be raised (by a factor of two to four), EPA continued to rely on the revised RfC of 0.05 $\mu\text{g}/\text{m}^3$ on the grounds that "there is no significant difference between the verified RfC of 0.05 $\mu\text{g}/\text{m}^3$ and alternative estimates of 0.09 to 0.2 $\mu\text{g}/\text{m}^3$."¹⁹¹

189. 1990 EPA Briefing Document, *supra* note 187.

190. 1991 EPA Briefing Document, *supra* note 188.

191. Waiver Decision, *supra* note 39, at 42250.

The point of these observations is simply that the RfC process as applied by EPA affords EPA ample opportunity to manipulate the assessment to achieve policy objectives unrelated to real risks, using a process which is ultimately policy-based rather than fact-based. By contrast, EPA's discretion to achieve its policy objective (i.e., the identification of a "concern" about MMT) would have been substantially constrained if EPA had employed the traditional quasi-fact approach for risk assessment since that approach requires an evidentiary basis (the identification of a specific risk at a specific exposure level) to support regulatory action.

As noted above, the lowest LOAELS identified by EPA (for an effect which may or may not be real or adverse) fell in the 30 to 50 $\mu\text{g}/\text{m}^3$ range, while the lowest NOAELS fell in the 10 to 20 $\mu\text{g}/\text{m}^3$ range. Had EPA been constrained by the existing evidence on manganese health effects, EPA could not have identified any "overlapping boundaries" between exposures and these effect levels because even the highest estimated manganese exposures developed by EPA over the course of the four-year proceeding remained 50- to 250-fold below those levels.

In sum, EPA's use of RfCs in risk assessment has fundamentally changed the nature of the traditional risk assessment. Use of RfCs shifts the focus of the assessment away from facts and places it on uncertainty and the adjustment factors applied to account for that uncertainty. In this way, EPA greatly expands its discretion to achieve regulatory results consistent with its policy objectives, unhindered by evidentiary constraints. The result is purported authority to regulate far broader than that afforded by the traditional significant risk concept.

The RfC and Judicial Review

The legality of EPA's use of RfCs in risk assessment and as a basis for regulatory decisions has yet to be resolved. Court cases that have involved scrutiny of the application of RfCs in risk assessment have been few and send mixed signals.

For example, when Ethyl challenged EPA's 1994 ban on use of MMT, the D.C. Circuit called EPA's public health assessment of manganese "a bizarre departure from existing practice, in complete defiance of the plain terms of the statut[e]," but stopped short of rejecting EPA's authority to apply RfCs in risk assessment.¹⁹² Having granted the relief sought by Ethyl on other grounds,¹⁹³ the court merely noted that EPA had provided "no explanation whatsoever for the application of a different [public health] standard," leaving unresolved whether use of RfCs in risk assessments as a mechanism for requiring regulated entities to prove the absence of risk is per se impermissible.¹⁹⁴

192. *Ethyl Corp. v. U.S. Environmental Protection Agency*, 51 F.3d 1053, 1063, 25 ELR 20817, 20823 (D.C. Cir. 1995).

193. The court held that EPA has no authority to consider the issue of public health under §211(f)(4) of the Clean Air Act. *Id.* at 1063-64. The court acknowledged, however, that EPA retained its authority to regulate where public health is a concern under §211(c). *Id.* at 1065.

194. *Id.* at 1063. See *Natural Resources Defense Council v. U.S. Environmental Protection Agency*, 824 F.2d 1146, 1153, 17 ELR 21032, 21036 (D.C. Cir. 1987) ("[W]e think it unlikely that science will ever yield *absolute* certainty of safety in an area so complicated and rife with problems of measurement, modeling, long latency, and the like.").

Another D.C. Circuit case, *Chemical Manufacturers Ass'n v. U.S. Environmental Protection Agency*, appears to sanction use of RfCs in risk assessment generally, but ultimately rejects the conclusion of the RfC-based risk assessment at issue.¹⁹⁵ The *Chemical Manufacturers Ass'n* case involved a challenge to the validity of EPA's determination that methylene diphenyl diisocyanate (MDI) was a "high risk" pollutant under §112 of the Clean Air Act. In presenting its challenge, the Chemical Manufacturers Association (CMA) did not attack use of the RfC per se in risk assessment. Rather, the CMA argued, among other things, that the health effect (nasal irritation) on which the RfC was based was "too minor a health effect to be classified as 'high risk'" under the terms of the statute.¹⁹⁶ The court agreed with the CMA, noting that "the agency's approach had the effect of treating as a 'high risk' a pollutant of which the only known health effect is non-serious . . . inconsistent with agency's own stated intentions."¹⁹⁷ On this basis, the court vacated EPA's determination on MDI.

Notably, EPA's use of the manganese RfC was far different and far more problematic than its use of the RfC in the MDI example. As the court explained, "EPA designated as high risk every hazardous air pollutant, including MDI, for which [EPA's predicted level of exposures] exceeded by at least one order of magnitude (i.e., was at least 10 times) the RfC for that pollutant."¹⁹⁸ In the case of manganese, by contrast, EPA compared estimated manganese exposures directly to the RfC.

Had EPA employed the manganese risk assessment approach in the case of assessing MDI, dicta in the court's decision suggest that the court would have rejected such an approach. As explained by the court, "[a]n ordinary reading of the Congress's mandate to the EPA would suggest that the agency must first identify whether exposure to a small amount of a hazardous air pollutant can ever cause an 'adverse public health effect,' and then determine the likelihood of that effect occurring."¹⁹⁹ On its face, exposures at a level deemed to be "safe" even for sensitive populations would hardly seem to qualify as the identification of an "exposure to a small amount of a hazardous air pollutant . . . 'adverse [to] public health'" thought necessary by the court. Of course, in the case of MDI, EPA did not rely on the RfC directly for its risk assessment, but rather, identified a level 10-fold higher than the RfC as a level potentially entailing a significant risk. The court therefore did not confront the more problematic case where a safe exposure level was being used as a risk/no risk demarcation, as occurred in the case of manganese.

Recommendations

Yet to be resolved in any direct way is whether this reliance on RfCs is consistent with EPA's regulatory authority under the environmental statutes that it implements, or more generally, whether such reliance serves the broader interests of society at large. The principal purpose of the Clean Air

195. 28 F.3d 1259, 1266-68, 24 ELR 21210, 21213-15 (D.C. Cir. 1994).

196. *Id.* at 1266, 24 ELR at 21214.

197. *Id.* at 1267, 24 ELR at 21214.

198. *Id.* at 1262, 24 ELR at 21211.

199. *Id.* at 1266, 24 ELR at 21214.

Act, for example, is "to protect and enhance the quality of the Nation's air resources so as to promote the public health and welfare *and the productive capacity of its population.*"²⁰⁰ Although application of RfCs in risk assessment clearly has the potential "to protect and enhance the quality of the Nation's air resources" by foreclosing the emission of substances above very conservative, "safe" screening levels, it also has the clear potential to stymie innovation and the enhancement of "the productive capacity" of the nation by establishing an overly protective public health standard.

To sanction regulatory action based on EPA's RfC-based approach is to sanction the adoption of a "zero-risk" public health standard, and to accord EPA the regulatory discretion to decide what is "safe" and what is not without effective evidentiary constraints. Only in the rarest of instances has Congress signaled a desire to protect the public from *all* risks by enacting a "zero-risk" public health standard.²⁰¹ To the contrary, the common thread among most environmental statutes is a desire by Congress to protect the public against "significant risks," recognizing "the pervasive nature of scientific uncertainty and the inherent limitations of scientific knowledge" as it pertains to day-to-day activities.²⁰²

As more RfCs and RfDs have been developed by EPA, their use in risk assessment has increased, as is illustrated by several recent high profile risk assessments. These risk assessments have addressed a variety of substances, including methyl tertiary butyl ether (MTBE),²⁰³ mercury,²⁰⁴ diesel particulate emissions,²⁰⁵ and of course, manganese. Among other things, EPA has indicated that it intends to rely on RfCs for the purpose of implementing its regulatory authority under §211 of the Clean Air Act,²⁰⁶ and for continued implementation of its authorities under Superfund.²⁰⁷

Against this background, the regulated community can take a number of steps to protect against the inappropriate application of RfCs in risk assessment. First, the regulated community must remain vigilant concerning ongoing RfC and RfD development proceedings within EPA. Although

200. 42 U.S.C. §7401(b)(1), ELR STAT. CAA §101(b)(1) (emphasis added).

201. *See, e.g.*, 21 U.S.C. §348(c)(3) (1988) (the Delaney Clause). Even where Congress has taken the extraordinary step of directing an agency to prohibit use of a substance that presents any level of risk to public health, the necessary precondition for regulatory prohibition is, of course, a specific showing of at least some risk.

202. *See* *Natural Resources Defense Council v. U.S. Environmental Protection Agency*, 824 F.2d 1146, 1153, 17 ELR 21032, 21036 (D.C. Cir. 1987); *Industrial Union Dep't v. American Petroleum Inst.*, 448 U.S. 607, 645, 10 ELR 20489, 20498 (1979) ("In the absence of a clear mandate . . . it is unreasonable to assume that Congress intended to give the Secretary [such] . . . unprecedented power over American industry.").

203. EPA, Assessment of Potential Health Risks of Gasoline Oxygenated with Methyl Tertiary Butyl Ether (MTBE), EPA/600/R-93/206 (Nov. 1993).

204. EPA, Mercury Study Report to Congress (Draft) (Aug. 1995).

205. *See* EPA, Health Assessment Document for Diesel Emissions (Review Draft), EPA/600/8-90/057Bb (Dec. 1994).

206. *See* EPA, Fuels and Fuel Additives Registration Regulations, Notice of Proposed Rulemaking, 57 Fed. Reg. 13168, 13210-11 (Apr. 15, 1992).

207. EPA, Regarding Use of IRIS Values in Superfund Risk Assessment, OSWER Directive #9285.7-16 (Dec. 21, 1993).

the mechanisms for public involvement in those proceedings are minimal, involvement to the full extent permitted by EPA's current policies may help to curtail unnecessarily conservative RfCs and RfDs. At a minimum, such involvement will help to create a more favorable administrative record for purposes of any subsequent administrative or legal proceedings where EPA or other federal agencies attempt to apply RfCs or RfDs in risk assessments.

Second, wherever federal agencies like EPA rely on RfCs or RfDs in risk assessment, the regulated community should consider carefully whether such reliance is consistent with the underlying public health standard the federal agency is seeking to implement through its risk assessment. As the U.S. Supreme Court has recognized, the burden is on federal agencies to justify regulatory action "on the basis of substantial evidence,"²⁰⁸ and regulatory action is typically appropriate only where it is necessary to protect against a significant risk to public health.

Whenever EPA or other federal agencies apply RfCs or RfDs in risk assessments to shift to the regulated community the burden of proving the absence of risk to avoid regulation (in lieu of "substantial evidence" supporting the need for such regulation), such efforts should be opposed in clear and unequivocal terms. This will create the strongest possible record for subsequent legal challenge, should such challenges become necessary.

Third, where these first two steps fail to convince EPA or other federal agencies that application of the RfC or RfD in risk assessment is inappropriate, careful consideration should be given to possible legal action directly challenging the nature of the risk assessment. As this Article has explained, a clear tension exists between the typical legal standard under which EPA and other federal agencies have authority to act to protect public health, i.e., the existence of a significant risk to public health, and the conclusions of risk assessments that rely on indisputably safe screening level exposures as a basis for regulatory action. This tension can provide the basis for legal action challenging inappropriate use of RfCs and RfDs in risk assessment.

208. *Industrial Union Dep't v. American Petroleum Inst.*, 448 U.S. at 653, 10 ELR at 20501.

Finally, as risk assessment becomes more prevalent as a decisionmaking tool, the absence of clearly defined principles for conducting risk assessments becomes ever more apparent. EPA and others²⁰⁹ have sought to fill this gap with their own definitions and approaches to risk assessment. But given the central nature of risk assessment as it relates to the day-to-day operation of the nation's business, a more legitimate forum for establishing consistent principles of risk assessment is Congress. Recent efforts to adopt risk-assessment legislation in Congress have failed largely because such legislation has been incorrectly perceived as a "retreat" from the environmental gains that have been made in this country over the past 20 years. The better view is that such legislation is necessary precisely because of the environmental successes of the past 20 years. The environmental consciousness of the nation is now so pervasive that very little economic activity occurs without some level of environmental oversight, including risk assessments. The regulated community should give thought to maintaining its efforts to promote the adoption of reasonable reform of the risk assessment process, including restrictions on the use of RfCs and RfDs in risk assessment.

Conclusion

To date, EPA and other public health authorities have been given wide latitude to decide how risk assessments should be conducted on a statute-by-statute basis. The result has been an inexorable movement toward the zero-risk paradigm reflected in RfC-based risk assessments, including the manganese assessment described in this Article. EPA has questioned the wisdom of applying the "innocent until proven guilty" standard of the American judicial system in matters of environmental policy, and its increased reliance on RfC-based risk assessments is a practical manifestation of a movement away from that standard. The question yet to be resolved is whether the "guilty until proven innocent" standard reflected in RfC-based risk assessments is a preferable alternative.

209. *See, e.g., NATIONAL RESEARCH COUNCIL, SCIENCE AND JUDGMENT IN RISK ASSESSMENT* (1994).