Implementing Section 112 of the Clean Air Act - Sound Cancer Risk Reduction Policy or Chasing a Rat's Tale?

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According to data compiled by the Department of Health and Human Services, there were approximately one and a half times as many cancer deaths in the United States per 100,000 persons in 1992 as there were in 1950 and nearly three times as many as in 1900.¹ In absolute terms, cancer has risen from the tenth leading cause of death, resulting in four percent of all deaths in 1900, to the third leading cause of death, resulting in twenty-four percent of all deaths in 1992.² Yet, over the same period, human life expectancy increased by nearly thirty-two years.³ This increase in life expectancy may provide a plausible explanation for the corresponding increase in cancer rates.⁴ That is, because of the long latency period of most cancers, Americans in past years often died of other causes before the disease had a chance to fully develop.⁵ In fact, recent studies conclude that patients age sixty-five and older account for roughly two-thirds of all cancer deaths.⁶

1. THE INFORMATION PLEASE ALMANAC, ATLAS AND YEARBOOK 847 (47th ed. 1994) (203.1 cancer deaths per 100,000 persons occurred in the United States in 1992) [hereinafter INFORMATION].
2. INFORMATION, cited at note 1, at 847 (citing the Department of Health and Human Services, National Center for Health Statistics).
3. Id. at 850. Note that the increase of 32 years in life expectancy is based on data for those born in 1900 versus those born in 1992. If life expectancy is viewed as an aggregate measure of background risk, then it is has decreased, perhaps paradoxically, despite the dramatic industrial technological growth over the same period. See Peter Huber, Safety and The Second Best: Hazards of Public Risk Management In The Courts, 85 COLUM. L. REV. 277, 295 (1985).
5. CROSS, cited at note 4, at 17-18.
Nonetheless, data generated under the Emergency Planning and Community Right-to-Know Act\(^7\) indicated that 2.4 and 2.7 billion pounds of "toxic chemicals"\(^8\) had been released to the environment in 1988 and 1987 respectively.\(^9\) Coupled with the United States Environmental Protection Agency's (the "EPA") risk assessment that as many as 2700 excess cancer deaths a year may have resulted because of these emissions, Congress became convinced that the emission of hazardous air pollutants was a problem of grave importance and converted section 112 of the Clean Air Act\(^10\) ("Section 112") into an "aggressive new program" to regulate hazardous air pollutants.\(^11\)

Though attempts to apportion cancer mortality amongst potential causes cannot be made with certainty, studies indicate that air pollution might only account for one to five percent of the total number of estimated cancer deaths per year.\(^12\) Tobacco, diet, reproductive and sexual behavior, indoor air pollution,
natural radiation, and occupation have all been estimated to cause more cancer deaths than air pollution. The EPA estimate of 2700 deaths per year due to the emission of hazardous air pollutants from industrial sources accounts for only 0.6 percent of all cancer deaths and is less than its prediction of cancer mortality due to exposure to second hand tobacco smoke.

The law written by Congress to reduce the risk of cancer from air pollution, Section 112, is inextricably based and helplessly insistent upon the continued use of the science of risk assessment. Because of this symbiosis, the science of risk assessment, Section 112 and regulations recently promulgated thereunder, are evaluated in this comment. This evaluation starts with the premise that a sound cancer prevention policy should seek to reduce the incremental risk of cancer death as much as practicable; but to meet such a goal, costs associated with the marginal reduction of cancer risk must be kept at a rational level. Finally, in light of the limitations of the science of risk assessment, recommendations to improve Section 112 and EPA implementation are presented.

BACKGROUND

Regulation of Hazardous Air Pollutants Under The Clean Air Act

Section 112 was created to regulate and reduce exposure to substances that science has concluded to be hazardous, including those suspected of causing cancer. The law, as originally written, did not include a list of substances thought to be hazardous; it instead delegated that authority to the EPA by requiring it to decide, on a case-by-case basis, what substances should be regulated. Over the course of twenty years, the EPA succeeded in regulating only seven substances. To many, this performance

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13. Id. at 35. Estimates indicate the following contributions, in terms of percentage of total cancer deaths, per cause: Tobacco (24-40), diet (25-60), reproductive and sexual behavior (7-13), indoor air pollution (2-6), natural radiation (2-4), and occupation (1-20). Id.
14. Andrew J. Harrison, Jr., An Analysis of The Health Effects, Economic Consequences and Legal Implications of Human Exposure to Indoor Air Pollutants, 37 S.D. L. REV. 289, 300-01 (1992) (citing an EPA report that estimated 3800 cancer deaths per year result from second hand tobacco smoke). The estimate of 0.6 percent of cancer deaths is based on a cancer mortality rate in 1992 of 203.1 per 100,000 and a total population in the United States of roughly 250 million persons. See INFORMATION, cited at note 1, at 847.
15. Robertson, cited at note 9, at 200.
16. Id. at 200.
17. Id. at 201. Prior to 1990, the EPA established emission standards for the compounds arsenic, asbestos, benzene, beryllium, mercury, radionuclides, and vinyl
signified that Section 112 had been a failure. Accordingly, Section 112 was dramatically rewritten by Congress. The heart of Congress’ “aggressive new program” is a list of 189 pollutants defined as hazardous air pollutants. Of the 189 compounds, only eighty-eight were considered by the EPA to be carcinogenic. Furthermore, the conclusion of carcinogenicity for thirty-eight of these substances was based on data that the EPA considered limited or inadequate. More importantly, the data needed to assess the potential risk to people, the inhalation unit risk for carcinogens and inhalation reference concentration for threshold toxic substances, have not yet been developed.

18. Id. Some commentators have suggested that in the case of carcinogenic substances, the EPA was forced to apply a standard, created by Congress, that literally would have shut down entire industries. See Robertson, cited at note 9, at 200. At the time, Section 112 required that the administrator of the EPA set emission standards “at the level which in his judgment provides an ample margin of safety to protect the public health.” Clean Air Act § 112(b), 42 U.S.C. § 7412(b) (1982) (amended 1990). This created difficulties because under the “one hit” theories of cancer no such level would exist. CROSS, cited at note 4, at 5. See note 57 for a discussion of the “one hit” theories of cancer. Fearing the impact a “zero risk” standard would have on industry, the EPA decided to consider costs in the process of establishing standards under Section 112. Id. at 105. Consequently, much litigation and delay over the proper interpretation of Section 112 ensued. Id. Some sense was restored in 1987 when Judge Bork of the Circuit Court for District of Columbia decided that Congress intended the EPA to establish standards that would result in ambient levels that would pose an “acceptable risk to health” without considering cost. Natural Resource Defense Council v. EPA, 824 F.2d 1146, 1166 (D.C. Cir. 1987). The court, however, went on to hold that the EPA could consider costs in the determination of the “ample margin of safety” that was required above and beyond the safe level. NRDC, 824 F.2d at 1165-66. Following the decision, the EPA defined a safe ambient level for suspected substances as that which would result in one excess cancer death per 10,000 people. 54 Fed. Reg. 38,044, 38,045 (1989) (codified at 40 C.F.R. Pt. 61).

19. See Waxman, cited at note 9, at 1773-74.
20. 42 U.S.C. § 7412(b)(1) (Supp. 1993). The term hazardous air pollutant is defined under the Act as “any pollutant listed pursuant to subsection (b).” 42 U.S.C. § 7412(a)(6) (Supp. 1993). The list was for the most part borrowed from regulations passed pursuant to the Emergency Planning and Community Right-to-Know Act. Robertson, cited at note 9, at 202. The current list is provided in 40 C.F.R. § 372.65.

21. COMMITTEE ON RISK ASSESSMENT OF HAZARDOUS AIR POLLUTANTS, SCIENCE AND JUDGMENT IN RISK ASSESSMENT A50-61 (Prepublication Copy, 1994) [hereinafter COMMITTEE].
22. COMMITTEE, cited at note 21, at A50-61. The EPA ranks substances believed to be carcinogens by the weight of the evidence according to category. Id. at 7-1. The categories, in descending order of strength of evidence are as follows: “Group A — Known human carcinogen; Group B1 — [Probable human carcinogen] — limited human data; Group B2 — [Probable human carcinogen] — inadequate human data, sufficient animal data; Group C — Possible human carcinogen — no human data, limited animal data; Group D — Not classifiable as to human carcinogenicity — Inadequate or no human or animal data.” Id. at A53.
23. Id. at A38. The reference concentration is the maximum level, typically
oped for 125 of the 189 listed substances. It has been acknowledged that the EPA does not have the necessary resources to conduct a thorough risk analysis of all 189 listed substances within the proscribed time period. Notwithstanding these practical limitations, when deemed appropriate, the list may be modified by any person or by the EPA.

Facilities that emit one or more of the listed substances may be required to reduce such emissions. The emission standards establishing the manner in which such reductions will be achieved differ depending upon whether a given source has been classified as a “major” or “area” source. Emission standards of both the area and major source groups have been further organized in terms of categories and subcategories. Congress has designated a major source as any stationary source “that emits or has the potential to emit considering controls, in the aggregate, ten tons per year or more of any hazardous air pollutant or twenty-five tons per year or more of any combination of hazardous air pollutants.” Sources not meeting this definition are designated as area sources. Source categories may be deleted from the list. However, if they were listed because they emit substances suspected of causing cancer, the Clean Air Act now requires “that no source in the category... emits such hazardous air pollutants in quantities that may cause a lifetime risk greater than one in one million” to the maximally exposed individual before the source category may be deleted.

Emission standards promulgated by the EPA are required to

indicated in units of micrograms per cubic meter, at which a given substance will not subject a person to a significant risk of adverse affects. Id. The hypothetical person is assumed to be the most sensitive. Id. Exposure is assumed to be continuous, for a lifetime, and by inhalation. Id.

24. Id. at A50-61.
25. Id. at 12-2.
26. 42 U.S.C. § 7412(b)(3). In the case of a substance that was originally listed because it was a suspected carcinogen, the petitioner would have to show with adequate data that the substance was no longer reasonably anticipated to threaten the adverse health effect of carcinogenicity. 42 U.S.C. §§ 7412(b)(2)-(3).
27. 42 U.S.C. § 7412(d).
29. Id. at § 7412(d)(1). A source category is simply a grouping of industries that have “common features” such that regulation should be done “in the same way on the same schedule.” 57 Fed. Reg. 31,576, 31,578 (1992). An initial list of categories and subcategories of sources has been promulgated by the EPA. See 57 Fed. Reg. 31,576 (1992).
31. Id. at § 7412(a)(2).
32. See notes 89-91 and accompanying text for an explanation of the concept of the maximally exposed individual.
reduce emissions from major sources to the maximum degree possible by employing the maximum available control technology ("MACT") and require, at the EPA administrator's discretion, that area sources be controlled with generally available control technologies or management practices ("GACT"). Methods for reducing emissions under these standards include installing equipment that will eliminate or reduce emissions by collection and treatment, changing work practices, improving process containment, substituting materials, or by making other process changes. MACT for new sources may be more restrictive than for existing sources. In addition, revised Section 112 protects an existing operator from having to install the MACT if a process change would result in a mere de minimis increase in actual emissions or if a process change resulting in emissions greater than de minimis are offset by reductions elsewhere. If emissions are greater than de minimis and the operator is unable to offset the emissions, then the process change would be considered a modification requiring the MACT, as specified

34. 42 U.S.C. §§ 7412(d)(2),(5). Section 112(d)(2) provides that "[e]mission standards promulgated under this subsection and applicable to new or existing sources of hazardous air pollutants shall require the maximum degree of reduction in emissions of the hazardous air pollutants." Id. at § 7412(d)(2). Section 112(d)(5) provides that:

With respect only to categories and subcategories listed pursuant to subsection (c), the Administrator may . . . elect to promulgate standards or requirements applicable to sources in such categories or subcategories which provide for the use of generally available control technologies or management practices by such sources to reduce emissions of hazardous air pollutants.

Id. at § 7412(d)(5).

35. Such methods include incineration or acid gas scrubbing.


37. A source is defined as new if it has been constructed or reconstructed after an emission standard has been promulgated. 42 U.S.C. § 7412(a)(4).

38. 42 U.S.C. § 7412(d)(3). New sources "in a category" must be regulated by a standard that is "not less stringent than the emission control that is achieved in practice by the best controlled similar source" while existing sources "in a category" must be regulated by a standard that is "not less stringent . . . than the average emission limitation achieved by the best performing 12 percent of existing sources."

Id.

39. 42 U.S.C. § 7412(g)(1)(A). Section 112(g)(1)(A) allows changes resulting in increases above de minimis without requiring the installation of MACT (that would ordinarily be required with a modification) provided "if such an increase in the quantity of actual emissions of any hazardous air pollutant from such source will be offset by an equal or greater decrease in the quantity of emissions of another hazardous air pollutant (or pollutants) from such source that is deemed more hazardous."

Id.

40. Modification is defined as:

Any physical change in, or change in the method of operation of, a major source that increases the actual emissions of any hazardous air pollutant emitted by such source by more than a de minimis amount or that results in the emission of any hazardous pollutant not previously emitted by more than a de minimis amount.
for existing sources.  

Because de minimis is not defined, and a means to rank hazardous air pollutants is necessary to implement offset provisions, Congress specifically required the EPA to publish guidance on the matter.  

In response, the EPA issued proposed regulations. The proposed regulations treat a new facility, or "green field facility," as a "constructed major source" and require control of emissions with MACT as specified for new sources. Moreover, where an existing source would be changed such that an additional ten tons of a single hazardous air pollutant or twenty-five tons of hazardous air pollutants in the aggregate, would be emitted, the EPA proposed that such a change be treated as the construction of a new source or in the alternative as a modification. If such a change is treated as a modification, an operator of a facility would be permitted to take credit for offsets per Section 112(g) or would be required to install the MACT as specified for existing sources.

The EPA's proposed regulation also lists de minimis emission levels of the listed hazardous air pollutants. Emission values associated with carcinogens were calculated by conducting ambient dispersion modeling based on a "model plant," in conjunction with "upper bound" inhalation unit risk potency factors corresponding to a lifetime risk of contracting cancer of one in one million.

42. 42 U.S.C. § 7412(g)(1)(B).
43. See 59 Fed. Reg. 15,504 (1994) (to be codified at 40 C.F.R. pts. 63 and 70) (proposed April 1, 1994). In general, rules promulgated by the EPA pursuant to Section 112 will not supersede more stringent state standards. See 58 Fed. Reg. 62,262, 62,263-64 (1993) (to be codified at 40 C.F.R. pts. 9 and 63) (final rule proposed November 26, 1993) (The purpose of the rule is to "allow the EPA and the States to work together to minimize potential program redundancies ... and to assure that all sources of hazardous air pollutants ... meet standards ... that are no less stringent than corresponding Federal requirements.").
44. 59 Fed. Reg. at 15,517.
45. Id. at 15,517-19.
46. Id. at 15,525.
47. Ambient dispersion modeling is a mathematical computer based method for calculating ground level concentrations of substances released to the atmosphere. See notes 85-86 and accompanying text for a detailed discussion of dispersion modeling.
48. 59 Fed. Reg. at 15,526. See notes 55-100 for a discussion of risk assessment methodology. Typically, scientists and engineers that conduct dispersion modeling input unity as an emission factor and later scale the predicted ground-level concentration based on the actual emission rate. Here the EPA has essentially done the opposite. That is, the de minimis level was estimated by scaling the input to the program (two tons per year) by multiplying it by the de minimis ambient level and dividing by the result predicted by the dispersion model. Id. The de minimis level is simply the level at which indicated exposure is associated with a risk of one in one.
In addition to the assumptions made to arrive at the “model plant” dispersion analysis, several additional critical assumptions were made. First, the EPA adjusted the inhalation unit potency factor upward by a factor of ten on the assumption that MACT would be installed within seven years, when standards are finally promulgated. In essence, the EPA has assumed that the entire lifetime de minimis dose would be absorbed in seven as opposed to the normally assumed lifetime of seventy years. If no inhalation unit potency factor existed for substances characterized as probable or known human carcinogens, the EPA assigned a default de minimis value of one ton per year. Finally, any de minimis emission rate estimated to exceed ten tons per year was assigned a default value of ten tons per year. The EPA made this assumption because it could not reconcile the possibility that an emission rate could be classified as “major” for the purpose of categorizing a particular source and yet be classified as insignificant when evaluating whether or not emission increases should be controlled.

Paragraph 63.40(e) of the proposed regulation anticipates that the promulgated MACT standards may have applicability cutoff emission levels that are higher than the de minimis rates under the proposed regulation. In such an event the applicability level will supersede the de minimis level. The applicability level for additional emission controls at process vents is determined by the calculation of total resource effectiveness (TRE). 57 Fed. Reg. 62,608, 62,636 (1992) (to be codified at 40 C.F.R. Pt. 53) (proposed December 31, 1992). The TRE is “a measure of the supplemental total resource requirement [additional fuel required for control by flare or incineration] per unit reduction of . . . [total organic carbon] associated with a vent stream.” 40 C.F.R. § 60.701. This method of estimating applicability is essentially a determination of economic feasibility. Id. In addition the HON has provisions for emission offsets and averaging. 57 Fed. Reg. at 62,613. Consequently the EPA statement that it believed “that the proposed rule [the HON]
cutoff levels will not necessarily be based upon health effects unlike the de minimis rate standards.

Because control of emitted substances is required under Section 112 before a determination of potency and without regard to source specific demonstrations of harm, Section 112 is unquestionably a radical departure from the approach used prior to 1990. Despite this departure, the use of risk assessment will continue to play an important role in the regulation of hazardous air pollutants.

**Risk Assessment**

Risk assessment is a procedure used to determine what effect substances that are released to the environment will have on humans.66 Cancer is a group of diseases whose etiology is studied by the method of risk assessment because many diseases in this category are believed to be caused or affected by exposure to substances in the environment.57 When risk assessment is employed quantitatively the effects are calculated in terms of the number of additional cancer deaths per the population that might occur as a result of long-term exposure to suspected substances.58 The risk number is obtained by multiplying two key parameters: the exposure level and the potency factor.59 Each of these are estimated based upon a complex array of data, predictive models, and assumptions; all of which involve varying degrees of uncertainty.60

The identification of substances as carcinogenic and the determination of the strength or potency to which these substances

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56. CROSS, cited at note 4, at 52.
57. Id. at 5. A living organism is maintained by the constant division and replication of millions of cells. Id. at 4. Uncontrolled abnormal cell growth, which ultimately may lead to the growth of tumors, occurs when this normal process of cell division is disrupted. Id. at 5. The disruption is believed to occur as a consequence of the inhalation, ingestion, or dermal contact with suspect substances. Id. Some theories (commonly known as "one-hit" theories) postulate that exposure to even a single molecule may trigger the disease. Id. The lag period between exposure to the suspect substance and to the manifestation of the disease as cancerous tumors (a span known as the latency period) could be as long as thirty years. Id. at 7.
58. Id. at 52.
59. Id. at 61-62.
60. Because of the uncertainty of risk assessment and because the use of risk assessment is necessary under Section 112, Congress included a provision in the Clean Air Act Amendments requiring the study and recommendation of improvement of risk assessment methodology. See 42 U.S.C. § 7412(o). The study has been issued and is entitled Science and Judgment in Risk Assessment.
are believed to cause cancer is established primarily by animal and epidemiological studies.\textsuperscript{61} While epidemiological studies of populations perhaps provide the best means of identifying substances as carcinogenic in humans, such studies alone are not sufficient.\textsuperscript{62} Frequently, it is impossible to find populations similar in all respects except their exposure to a suspected substance.\textsuperscript{63} In addition, because of the difficulty in quantifying the magnitude and duration of exposure, lack of control of exposure, and the relative insensitivity of the method,\textsuperscript{64} such studies are rarely practical for determining potency factors.\textsuperscript{65}

Potency determination is possible with laboratory animal studies ("bioassays") because such studies provide an opportunity for dose control and examination of tissue damage.\textsuperscript{66} The biggest disadvantage of this method of study is simply that human beings are not laboratory animals.\textsuperscript{67} Nonetheless, the use of carcinogenic potency information to predict human response requires the assumption that the test species will respond to an external stimulus in the same manner as humans would if similarly exposed and that the response in either case would be proportional to the stimulus received.\textsuperscript{68}

Animals used in the bioassays are typically subjected to doses dramatically higher than what humans would expect to receive in the environment.\textsuperscript{69} Additionally, the studies at a given dose are rarely repeated for few data points.\textsuperscript{70} To relate the results of the bioassays to possible potency in humans, models are employed that extrapolate incidents of malignant tumors in the bioassays at a high test dose to low environmental doses in humans and extrapolate the difference in results due to the size difference between the test animal and a typical human.\textsuperscript{71} The most frequently used model extrapolating the dose-response of the bioassays to humans uses a linear extrapolation at low dose

\textsuperscript{61.} COMMITTEE, cited at note 21, at 4-1. Epidemiology is the study of disease by comparison of similar populations in all respects except their exposure to a substance suspected of causing the disease. CROSS, cited at note 4, at 45-46.
\textsuperscript{62.} CROSS, cited at note 4, at 45-46.
\textsuperscript{63.} Id. at 46.
\textsuperscript{64.} Id. at 47. Increases in cancer rates less than an additional cancer death per thousand people can not be detected by epidemiological studies with reliable statistical significance. Id.
\textsuperscript{65.} Id.; see also COMMITTEE, cited at note 21, at 4-2.
\textsuperscript{66.} COMMITTEE; cited at note 21, at 4-3.
\textsuperscript{67.} Id.
\textsuperscript{68.} Id. at 7-14.
\textsuperscript{69.} Id. at 7-16.
\textsuperscript{70.} Id.
\textsuperscript{71.} COMMITTEE, cited at note 21, at 7-16, 7-17.
levels and is known as the linearized multistage model. The theory and assumptions underlying this model are somewhat simplistic and do not account for mechanisms that might more accurately suggest a nonlinear low-dose response. For example, substances that are not believed to act directly upon a cell's DNA, "nongenotoxins" or "promoters," are not contemplated by the theory underlying the linear multistage model.

Similarly, metabolic differences, due to the dramatic difference in species size between the tested animals and potentially exposed humans, are generally extrapolated based on body surface area. Such an extrapolation assumes that there is a proportional relationship between the administered dose and the delivered dose at the "target-site." Yet, the risk of cancer is often not derived directly from the administered substance, but rather from the delivered metabolites of the substance. Pharmacokinetic modeling replaces the assumption of proportionality between the delivered and administered dose by modeling the actual physiological processes upon which a more accurate prediction of metabolite level at a "target" organ can be made. Where such knowledge and data exists, the resultant

72. Id. The linear extrapolation of the low dose portion of the curve is made conservatively and represents the statistical upper bound (95th percentile slope) of a substance's potency. Id. The 95th percentile represents the point at which 95 observations out of 100 are lower than the remaining five. So for example the mean, the point at which there are as many observations above as there are below, is also referred to as the 50th percentile. Though the confidence level is greater with the conservative extrapolation, because there are fewer observations, uncertainty also increases. Bruce A. Egan, Science and Technology, in CLEAN AIR AND REGULATION 43, 49 (Timothy A. Vanderver, Jr. et. al. eds., 1992).

73. COMMITTEE, cited at note 21, at 7-17, 7-18. But note that a more sophisticated approach (incorporating mechanistic theories for example) would invariably require data on the interaction of substances with biological mechanisms that are often unavailable. Id. at 7-18.

74. Under some theories of the disease it is believed that the initially altered cell does not immediately lead to the growth of other abnormal cells. CROSS, cited at note 4, at 5-6. Rather, it is believed that such cells lie dormant until another substance, called a "promoter" converts the initiated cells into neoplastic ones. Id. at 6. Neoplastic cells are cells that exhibit abnormal growth. WEBSTER'S DICTIONARY MD-38 (Encyclopedic ed. 1987).

75. COMMITTEE, cited at note 21, at 7-18, 7-19.

76. Id. at 7-27.

77. Id. at 7-26. The administered dose is the dose inhaled, ingested or received dermally, whereas the delivered dose at the "target site" is the dose received at the affected organ. Id.

78. Id. at 6-9. The administered and biologically delivered dose differ because various biological responses occurring within the human body transform the hazardous substances. Id. at 7-26, 7-27. Metabolites are substances that are products of physical or chemical processes occurring within a living organism. WEBSTER'S DICTIONARY MD-35 (Encyclopedic ed. 1987).

79. COMMITTEE, cited at note 21, at 7-26.
potency factor is predicted with greater certainty and is often dramatically lower. For example during the EPA's evaluation of the substance methylene chloride pharmacokinetic, models predicted that the specific metabolic pathway associated with the suspected substance was significantly less active in humans than in mice. Because there was consensus in the scientific community, the EPA lowered the potency factor.

The second variable in the risk assessment equation requires a prediction of exposure levels. An atmospheric dispersion model predicts annual average ground level concentrations of released substances based on a variety of input factors including: actual hourly weather data, terrain elevation, site elevation, emission rate and variability, and discharge velocity. Because dispersion models can only approximate the complexity of atmospheric fluid dynamics, their accuracy in most applications is generally accepted as only being within a factor of two.

At both sections (estimation of residual risk and delisting) in revised Section 112, where Congress requires the demonstration of excess risk, the demonstration of risk must be made for the "individual in the population who is most exposed

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80. Id. at 7-26.
81. Id. at 6-11.
82. The EPA sought review of its proposed revision of the methylene chloride potency factor on the basis of pharmacokinetic modeling by the Science Advisory Board. Id.
83. Id. The potency factor was lowered from 4.1 x 10^{-6} excess cancer deaths due to a 1 micro gram per cubic meter of exposure to 4.7 x 10^{-7}. Id. at 6-9 and 6-11.
84. Egan, cited at note 72, at 48.
85. Id. at 53-54. Therefore if the model predicts that a given concentration at a particular point will be ten micro grams per cubic meter the actual value could be as low as zero or as high as thirty.
86. 42 U.S.C. § 7412(f)(2). Section 7412(f)(2) provides in part: If standards promulgated pursuant to subsection (d) and applicable to a category or subcategory of sources emitting a pollutant (or pollutants) classified as a known, probable or possible human carcinogen do not reduce lifetime excess cancer risks to the individual most exposed to emissions from a source in the category or subcategory to less than one in one million, the Administrator shall promulgate standards under this subsection for such source category. Id.
87. 42 U.S.C. § 7412(c)(9)(B)(i). Section 7412(c)(9)(B)(i) provides: In the case of hazardous air pollutants emitted by sources in the category that may result in cancer in humans, a determination that no source in the category (or group of sources in the case of area sources) emits such hazardous air pollutants in quantities which may cause a lifetime risk of cancer greater than one in a million to the individual in the population who is most exposed to emissions of such pollutants from the source (or group of sources in the case of area sources). Id.
to emissions of such pollutants. To arrive at the exposure level that the maximally exposed individual is believed to receive, the EPA assumes such an individual will remain at the location of the highest predicted annual concentration for a lifetime of seventy years. While this approach greatly simplifies the estimation of excess risk, it is unrealistically conservative because people are generally mobile, do not live at the same location for the duration of their lives, and do not spend twenty-four hours a day outside. This approach also fails to account for variability in human response.

Congress included a provision in Section 112 that required the National Academy of Sciences (the "NAS") to study the EPA risk assessment methodology. Based on the results of the NAS study, the administrator of the EPA is required to revise its "Guidelines for Risk Assessment." To the extent that the EPA decides not to implement the report's recommendations, it must provide a detailed explanation.

The report recently issued by the NAS produced a laundry list of recommendations for improving EPA risk assessments. Of the changes recommended, perhaps the most fundamental are those concerning uncertainty analysis. Uncertainty, in the context of risk assessment, is the existence of doubt as to whether predicted effects of exposure will actually occur. As discussed above, estimates of risk are calculated based on the results of experiments that are converted into a risk number through models, each requiring further data and, where scientific gaps in knowledge exist, assumptions. Uncertainty exists for each model, theory, and assumption, and in the aggregate can be quite substantial. Estimates of potency could vary by a factor as high as $10^{10}$, according to an analysis conducted on the risk assessment studies of saccharin.

Current EPA practice often states risk as a single point and typically qualifies uncertainty by stating that the estimate of risk is within a "plausible upper bounds" and that the lower

88. Id.
89. COMMITTEE, cited at note 21, at 3-3, 3-4.
90. Id.
91. 42 U.S.C. § 7412(o).
92. Id. at § 7412(o)(7).
93. Id.
94. COMMITTEE, cited at note 21, at 12-1 to 12-24.
95. Id. at 9-2.
96. Id.
97. Id.
bound of confidence "could be as low as zero." The NAS report concluded that this approach to uncertainty creates a "false sense of security" and that the only way to combat this illusion is by quantifying the uncertainty inherent in risk assessment results. Only with a measure of the certainty of risk and the corresponding benefit associated with the reduction of that risk through the use of control measures (at cost), can risk managers, such as Congress and the EPA, be expected to make intelligent decisions.

An Optimal Approach — Incentive Based Regulation

Market-oriented and incentive-based regulation schemes are capable of delivering a greater level of cancer reduction than can be obtained by the traditional "command and control" method of regulation (as employed by Section 112) because these approaches avoid over-regulation. The command and control approach meets its goal of reduced pollution either by commanding that certain technology be installed to reduce emissions or by commanding that emissions not exceed a specified performance standard. The disadvantage of this approach is that it does not differentiate amongst sources in terms of marginal control cost. That is, all sources must reduce emissions to the same degree or install the same type of equipment even though to do so might cost some sources dramatically more than others. Variable marginal costs result

98. Id. at 9-23.
100. The measure of certainty is essentially a measure of the extent of conservatism of a given estimate of risk. Id. at 9-7. By providing more than a single-point estimate the decision maker will be able to evaluate how the level of risk varies with confidence levels (e.g. mean versus 95th percentile, etc.). Id. Moreover, the measure of uncertainty can provide a framework for choosing (or not choosing) alternate models or approaches to the many traditional conservative default assumptions currently used by the EPA in conducting risk assessment. Id. Without this information, the Committee concludes, risk managers are essentially "operating in the dark": when it comes to making choices regarding the appropriate level of conservatism. Id.
101. Id. at 9-7.
102. But see notes 116-18 and the accompanying text for a discussion refuting the contention that such approaches should never be used in the regulation of purely local harm.
104. Hahn, cited at note 103, at 5-6. Marginal cost in the context of emission reduction is the rate of cost of the next increment of emission reduction. Id. at 6 n.25.
105. Id. The EPA perpetuates the misconception that air pollution control is a function of the quantity of pollutant controlled or removed by publishing control cost
in over-regulation when the costs for controlling some of these sources become dramatic in comparison to the benefits obtained. Unfortunately, specifying a minimally acceptable marginal rate in the command and control process of standard setting has not been well received by the courts.\textsuperscript{106}

Conversely, incentive-based policies avoid the problem of over-regulation by equalizing the marginal cost of control.\textsuperscript{107} A pollution tax is one possible method for meeting this goal.\textsuperscript{108} Assuming the operator would always choose to pay a pollution tax as opposed to paying for the cost of controls that exceed the tax, the marginal control cost essentially becomes the same as the tax level. Consequently, over-regulation is avoided because the marginal cost of control (here, the tax rate) would never be less than the value of the benefits received.\textsuperscript{109}

**ANALYSIS AND CONCLUSION**

Revised Section 112 advances the goal of cancer risk reduction poorly because it requires control when: potential benefits are known to be unlikely (below de minimis); potential benefits are unknown; potential benefits are highly uncertain; and because it sets a risk-management level that is excessively low. Fortunately, because of recent EPA action some of these deficiencies may have been ameliorated.

If the methodology of the EPA's proposed regulations used to establish de minimis levels is also used to set minimum applicability levels, then over-regulation in the face of known trivial benefits could be largely avoided. Such an approach also represents sound risk reduction policy because the potency value used to estimate de minimis levels are unlikely to be higher.\textsuperscript{110}

\textsuperscript{106} See note 18 for a discussion of Judge Bork's famous opinion in *Natural Resource Defense Council v. EPA* that prohibited the EPA from considering cost when setting safe levels for exposure to hazardous air pollutants.

\textsuperscript{107} Hahn, cited at note 103, at 7-8.

\textsuperscript{108} Id.

\textsuperscript{109} Id. at 5-6.

\textsuperscript{110} In fact the EPA acknowledges that upper bound potency factors as used in the de minimis calculation "do not necessarily reflect the true risk, but often represent a conservative risk level which is an upper bound that is unlikely to be exceeded." 57 Fed. Reg. 31576, 31587 (1992).
What is troubling is that the EPA intends to set a maximum de minimis level at ten tons.

The emission level that triggers the major source standards was derived without regard to specific potency information. In fact, the purpose of the scheme was to regulate without complete knowledge concerning risk, not to regulate in the face of such information. The fact that the total annual emissions might exceed the arbitrary level of ten tons per year (and still be considered below de minimis per the EPA’s model plant calculation) does not alter the fact that such emissions are nonetheless trivial. Such a result was inevitable. If Congress had intended otherwise, it is doubtful that they would have included provisions to avoid control below de minimis level in the first place. The EPA needs to modify this deficiency by removing this arbitrary limit on de minimis emission rates before finalizing the proposed regulation. The present interpretation is an invitation for litigation.

Each performance standard will have an applicability level. For example, the HON sets an applicability level based on what is essentially economic feasibility. Such an approach is commendable because over-regulation is avoided by setting a marginal rate beyond which additional controls are not required. This, however, ignores the benefit side of the equation. In other words, it does not guarantee that there will be any benefit when the applicability equation indicates that controls must be installed. For this reason, the EPA should apply the methodology developed for de minimis determination as an additional applicability test for each performance standard.

Section 112 also over-regulates because it requires control of substances in the absence of potency factors and when the uncertainty in the benefits (cancer risk reduction) of control may

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111. EPA regulations or rules are promulgated in what is essentially a notice and comment procedure. 42 U.S.C. § 7607(d). The procedural requirements for challenging EPA regulations, delineated in section 307 of the Clean Air Act, first require a potential litigant to raise their objection to the regulation “with reasonable specificity for public comment (including any public hearing).” Id. at § 7607(d)(7)(B). If not so raised or if the litigant cannot show that such objection would have been impracticable then they may not raise such objections in a petition for judicial review. Id. If proper objection had been made during the comment period then a petition for review of the Administrator’s action must be filed “within sixty days from the date [of] notice of such promulgation.” Id. at § 7607(b)(1). This is essentially the only opportunity to adjudicate the validity of a given regulation as the Supreme Court has disallowed challenges to administrative rules made outside the rule-making process. See Federal Power Commission v. Texaco, Inc., 377 U.S. 33 (1964); see also American Airlines, Inc. v. Civil Aeronautics Board, 339 F.2d 624 (D.C. Cir. 1966).

112. See note 55 and accompanying text.
be excessive. The EPA approach in setting de minimis levels, when no potency factor exists, is to guess, while the EPA's approach in dealing with uncertainty has been to ignore.\textsuperscript{113} If the recommendations of the NAS report are followed, eventually useful estimates of uncertainty may accompany risk assessments. At a minimum, this information will better inform all concerned parties before further standards are set.

Quantifying uncertainty will pose a new question. What should be done when results of a particular risk assessment\textsuperscript{114} are known to be outlandishly uncertain? Instead of automatically requiring control when the risk-management level is exceeded, it would be better to set a level of uncertainty at which no control would be required. When the upper bound of the risk assessment predicts a risk greater than the management level, instead of allowing the externalization of the uncertain risk, the polluter should be forced to internalize the potential cost to the population by paying a pollution tax. Emission reduction would be encouraged while certain benefits, pollution reduction or revenue generated by the tax, would be assured. This is similar to the approach that Congress is currently pushing to address a far greater source of cancer, smoking.\textsuperscript{115}

Proponents of Section 112 believe that where reductions in ambient levels of hazardous air pollutants are desired, incentive-based approaches to regulation are not suitable.\textsuperscript{116} The rationale for such a conclusion is that incentive-based approaches are unfair if localized harm will result. What this argument presumes is that there will be harm. It fails to justify why incentive-based approaches should not be used where the harm is highly uncertain.

Even with these changes, Section 112 will remain a poor attempt at risk reduction as long as the risk-management level for de minimis and applicability levels remain at the absurdly low level of one excess cancer death per one million persons. In a world with unlimited resources such a goal might make sense. Unfortunately, in the real world of limited resources the bottom line requires that risk managers make intelligent choices. Repro-

\textsuperscript{113} See notes 100-03 and accompanying text.

\textsuperscript{114} See note 97 and accompanying text for a discussion of the outlandishly uncertain risk assessment analysis of saccharin.

\textsuperscript{115} Ways-Means Democrats Begin Grappling With Scope of Health Care Reform Bill, [1994] Daily Rep. for Exec. (BNA) No. 71, at D-64 (April 14, 1994) (Representative Stark, (D-Calif) a member of the House Ways and Means Committee that is currently working on a draft bill, suggests that the final package will include a stiff tobacco tax, perhaps as high as $1.25 per pack).

\textsuperscript{116} Waxman, cited at note 9, at 1752.
sentative Rowland of Georgia asserted, just prior to the passage of the Clean Air Act Amendments that, "[c]olleagues in the medical profession consistently ask me why we are willing to spend millions on protecting a theoretical [one] in [one] million people when we could spend a few thousand dollars and save hundreds of thousands of children with simple immunization and/or rehydration therapies." As long as better choices remain, an arbitrary risk management level of one in one million will continue to be a poor risk management choice and in particular, poor cancer reduction policy.

Understanding, assessing, and regulating cancer risk due to hazardous air pollution in an intelligent well-reasoned manner is a complex and quite possibly impossible task. While humankind through science has succeeded in extending life, in doing so we may have discovered that death comes in new ways. Ironically, it is the same science that continues to struggle mightily to explain what triggers this new means of death (cancer) and the same science that suggests, with unmeasured uncertainty, that hazardous air pollution is a small contributor.

Yet, we cannot begin to sensibly meet the goal of reducing cancer risk, if the first step, assessing risk, is not made with some reasonable degree of certainty. Unless and until an attempt is made to ascertain the measure of uncertainty in risk assessments, we are only fooling ourselves if we choose to believe that Section 112 of the Clean Air Act reduces the risk of contracting cancer.

If the science of risk assessment improves the uncertainty, its results should decrease the risk accordingly. Because the uncertainty of current risk assessments err on the conservative side, such theoretical improvements should require less control and make risk management less costly. But even with such improvements if we continue to regulate blindly and if we continue to over-regulate by failing to make intelligent risk management decisions, we may merely be continuing a chase of the rat's tale.

Stephen Yula

117. 136 CONG. REC. E3710 (1990). In Congressman Rowland's view the intent behind Section 112(o) (the provision requiring the NAS study of risk assessment) was to also provide for a "thorough, no holds-barred reexamination" of the assumptions that served as the basis for Section 112. 136 CONG. REC. at E3710.