The Potential Health Benefits of Controlling Hazardous Air Pollutants

Mary Jean Sawey
David R. Holtgrave
John D. Graham

Follow this and additional works at: http://digitalcommons.law.villanova.edu/elj

Part of the Environmental Law Commons

Recommended Citation
Available at: http://digitalcommons.law.villanova.edu/elj/vol1/iss2/3
THE POTENTIAL HEALTH BENEFITS OF CONTROLLING HAZARDOUS AIR POLLUTANTS*

MARY JEAN SAWEY†
DAVID R. HOLTGRAVE††
JOHN D. GRAHAM‡

TABLE OF CONTENTS

I. Legislative Background ........................................... 474
II. Existing Programs to Control Hazardous Air Pollutants ........................................................................... 476
III. The Role of Risk Assessment in Health Benefit Estimation ........................................................................ 478
   A. EPA’s Method of Risk Assessment ........................................ 478
   B. Risk Assessment and Benefit Estimation ........................................ 481
   C. General Shortcomings of EPA’s Method ........................................ 481
IV. The Health Risks of Air Toxics: Aggregate and Comparative Perspectives ........................................... 483
V. Assessment of Individual Pollutants ........................................ 484
   A. Qualitative Indications of Toxicity ........................................ 484
   B. Cancer Risk Estimates for Individual Pollutants ............ 485
   C. New Scientific Information ........................................ 485
   D. Legislative and Regulatory Implications ........................................ 486

* This article grew out of a request by the 100th Congress to the Congressional Research Service (CRS) for an analysis of the benefits of the proposed amendments (Senate Bill 1894) to the Clean Air Act (CAA). The original paper was one of six CRS contracted assessments of the current knowledge of and methods for estimating health benefits. Because our original analysis dealt specifically with hazardous air pollutants, the questions we raise and the conclusions we reach in this article remain relevant under current legislation. Finally, the authors wish to acknowledge John Blodgett for making this article possible.

† Assistant Professor of Radiation Oncology, University of Pennsylvania School of Medicine; Lecturer, Department of Health Policy and Management, Harvard School of Public Health. A.B. Rutgers College, 1977; M.S. S.U.N.Y. Stony Brook, 1983; Ph.D. New York University, 1987.

†† Assistant Professor of Family Medicine, Director of Clinical Decision Making, Oklahoma University Health Sciences Center. B.A. University of Missouri St. Louis, 1983; Ph.D. University of Illinois, 1988.

‡ Associate Professor of Policy and Decision Sciences, Department of Health Policy and Management, Director, Institute of Risk Analysis, Harvard School of Public Health. B.A. Wake Forest University, 1978; M.A. Duke University, 1980; Ph.D. Carnegie-Mellon University, 1983.

(473)
VI. Summary and Conclusions ........................................... 487

I. LEGISLATIVE BACKGROUND

The cornerstone of the Clean Air Act Amendments of 1970 was the complex relationship between federal and state government established for the control of the so-called "criteria" pollutants under sections 108 through 110. The criteria pollutants include ozone, lead, carbon monoxide, particulates, sulphur dioxide, and nitrogen dioxide. EPA was directed to establish ambient standards for these pollutants while the states were directed to implement emission controls that were necessary and sufficient to achieve the federal ambient standards.

The 1970 Amendments also included a distinct provision calling for federal regulation of any so-called "hazardous" air pollutant, defined as "an air pollutant to which no ambient air quality standard is applicable and which in the judgment of the Administrator causes, or contributes to, an increase in mortality, or an increase in serious irreversible, or incapacitating reversible illness." This definition was liberalized slightly in the Clean Air Act Amendments of 1977 to refer to any noncriteria air pollutant which "may reasonably be anticipated to result in an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness." The 1977 revision was intended "to emphasize the precautionary or preventive purpose of the Act."

Section 112 calls on EPA to (1) prepare a list of such hazardous air pollutants and (2) promulgate uniform National Emissions Standards for Hazardous Air Pollutants (NESHAPs) covering the relevant source categories for each listed pollutant. NESHAPs are to be set at the level which in the EPA Administrator's judgment "provides an ample margin of safety to protect the public health." The section sets out strict deadlines for regulating listed pollutants; emission standards for each source category are to be proposed within six months, and finalized within a year of the listing action. The law places no specific deadlines on when a pollutant must be listed and EPA is not currently required

by statute to list any specific substances as "hazardous" under section 112.

During the 1980's numerous legislative proposals were made to amend or replace section 112. Although none of these proposals were enacted, some of them received more serious consideration than others. In the 100th Congress, Title V of Senate Bill 1894 (the "Mitchell Bill") was the proposal that received the largest amount of political attention and debate. We focused our analysis in this report on some of the approaches contained in Title V. A bill with provisions similar to Title V, now referred to as Title III (section 112) of the Clean Air Act Amendments of 1990, was reintroduced in the 101st Congress.

The key provisions of Title V establish a list of 224 "hazardous" air pollutants and a mandatory schedule for issuing emission standards for the "major sources" — often large industrial facilities — of these pollutants. The standards are to be "technology-based," compelling the maximum reduction in emissions which can be achieved by application of the best available control technology. The "technology-based" test is considered more practical than the strict "ample margin of safety" language in section 112, although even the technological test does not permit explicit balancing of costs and benefits. The bill requires that standards for major sources of eleven specific pollutants be promulgated within 18 months of enactment. The remaining 213 covered pollutants are to be covered by technology-based standards over a ten-year period.

A second important provision of Title V authorizes EPA for the first time to list and regulate smaller "area" sources of hazardous air pollutants. Emissions from area sources are to be reduced by 25% from 1988 levels within four years. Additional reductions are expected in later years. The bill mandates listing of the following area source categories: degreasing and solvent cleaning.

   For a description of Title V, see McCarthy, CRS REPORT TO CONGRESS, HAZARDOUS AIR POLLUTANTS: AN ANALYSIS OF TITLE V OF S. 1894 (March 30, 1988). An alternative draft bill circulated by EPA Administrator Lee Thomas also received some consideration toward the end of the 100th Congress.

7. For a cogent statement of the rationale for Title V, see COMMITTEE ON ENV'T AND PUB. WORKS, CLEAN AIR STANDARDS ATTAINMENT ACT OF 1987, S. REP. NO. 231, 100th Cong., 1st Sess. 184-298 (1987) [hereinafter ATTAINMENT ACT REPORT].

operations, dry cleaners, pesticide applications, wood combustion units, gasoline marketing, mobile sources and materials transportation. Unlike the provision on major sources which compels technology-based standards, the provision on area sources gives the EPA Administrator wide latitude in deciding what steps are necessary to achieve these emission reductions. There are no enforcement or penalty provisions for noncompliance with this provision.

A third important provision of Title V amends section 112 to require that health-based standards for seven pollutants per year be initiated and that all source categories of listed pollutants be regulated eventually. Interim health-based standards that reduce lifetime cancer risks below one in a million are authorized for “possible” and “probable” carcinogens. Section 112 deadlines are relaxed to permit up to ten years for development of health standards.

Title V also contains several other provisions, such as new controls on catastrophic releases and new studies of aromatic hydrocarbons and hydrogen sulfides. However, we do not address these provisions in this report. 9

II. EXISTING PROGRAMS TO CONTROL HAZARDOUS AIR POLLUTANTS

In order to perform an objective assessment of the benefits of Title V, it would be necessary to project emission rates for the 224 listed air pollutants both with and without the regulations contemplated in Title V. The projected emission rates should take into account, for example, economic growth and the emission control programs that are likely to be implemented even if Title V is not enacted. In this section we discuss some existing control efforts that would need to be accounted for in a more detailed analysis of the incremental benefits of Title V.

First, EPA already has a significant program to address the problem of public exposure to hazardous air pollutants based on the authority given it in section 112. The progress of this program can be briefly summarized as follows: NESHAP standards have been proposed or promulgated for eight pollutants; decisions have been made not to regulate fifteen pollutants due to insufficient evidence or insignificant health risks; the agency's in-

9. For a discussion of these provisions, see ATTAINMENT ACT REPORT, supra note 7, at 296-98.
attention to list ten pollutants has been announced in order to allow adequate time to prepare emission standards; detailed health assessments of another twelve pollutants are in progress; and preliminary health and source screening is in progress for an additional twenty pollutants. EPA is likely to make limited and slow progress toward promulgating more standards under section 112 in the absence of Title V.

Hazardous air pollutants are emitted into the atmosphere from many of the same sources that are controlled for purposes of reducing ambient concentrations of criteria pollutants (e.g., ozone). As a result, regulations designed to control criteria pollutants often have the indirect effect of controlling emissions of air toxics. For example, metals and polynuclear compounds usually are emitted as particulate matter and their emission rates are reduced by particulate controls. Mobile and stationary source emissions of volatile organic compounds are controlled directly as ozone precursors. EPA studies show that indirect control of air toxics arising from control of criteria pollutants far exceeds the impact of the NESHAPs under section 112 regulations. Hence, an assessment of the incremental benefits of Title V should take into account the new controls of air toxics that are likely to result from additional programs to control criteria pollutants. Other titles of the Mitchell Bill aimed at criteria pollutants may also result in significant reductions in air toxics, even if Title V is not enacted.

Despite current EPA efforts to promulgate emissions standards for hazardous air pollutants, Title V, if enacted, would be likely to accelerate federal regulatory activity in this area. The new authority to enact standards under Title V is likely to reduce EPA's reluctance to promulgate emission standards because technology-based standards are more workable than the strict health-based statutory tests in section 112. (Even technology-based standards, however, may be less workable than those based on a case-by-case balancing of costs and benefits.) Moreover, confusion at EPA about the legal constraints imposed by the recent Vinyl Chloride decision on section 112 will also be removed. Title V may


also draw more political attention and agency resources to hazardous air pollutants, thereby causing more expeditious agency decision making. Although Title V's mandated listing process may relieve EPA of the legal burden to defend in detail the scientific rationale for each listing decision, the agency is likely to be burdened with the need to respond to dozens of new petitions to remove marginally hazardous pollutants from the list of 224 included in the bill.

III. THE ROLE OF RISK ASSESSMENT IN HEALTH BENEFIT ESTIMATION

Risk assessments for each of the 224 pollutants in Title V would be necessary to produce a rigorous estimate of health benefits of Title V. Synergistic effects (i.e. the combination of pollutants causing toxicity greater than the sum of their individual effects) that might occur among the 224 pollutants would need to be taken into account as well. The problem with this ideal analytical strategy is that it cannot be implemented today with available exposure and toxicity data. Consequently, it is not possible to produce a scientifically reliable estimate of the health benefits of Title V.

In order to provide a crude indication of the potential health benefits of Title V, section IV of this report summarizes what is known about the overall size of the air toxics problem. The primary focus is on cancer risk because that is the health endpoint which has dominated EPA's risk assessment activities. Before evaluating in section IV the recent estimates of the health risks of air toxics, this section reviews and critiques the risk assessment method employed by EPA. It is the method that was used in the studies to be reviewed in section IV, and is the only method for benefit estimation that is in widespread use at this time.

A. EPA's Method of Risk Assessment

Following the recommendations of a 1983 report of the National Research Council, EPA divides risk assessment into four stages:

(1) hazard identification: a study of the weight of the scientific evidence to determine whether or not a chemical or mixture poses a risk of adverse health effects to humans;

(2) dose-response assessment: a study of the quantitative rela-
tionship between the amount of exposure to the chemical and the incidence (or severity) of resulting illness;

(3) exposure assessment: a study of the number of people exposed to the chemical and their exposure profiles (concentration, frequency, and duration); and

(4) risk characterization: a summary of the overall magnitude of health risk attributable to exposure to the chemical, including some discussion of the degree of scientific uncertainty about the risk.12

Although risk assessment of toxic chemicals is still in its intellectual infancy, an entire consulting industry has arisen to fill the need for such studies.13

In the case of carcinogens, which have been the type of toxic air pollutant of greatest public concern, EPA has performed risk assessments since 1984 based on a specific procedure developed by the agency’s Carcinogen Assessment Group.14 A modified form of this procedure, described below, was incorporated into final EPA guidelines in 1986.15 These guidelines are now under reconsideration by EPA.

For purposes of identifying potential human carcinogens, EPA developed an adaptation of the classification scheme for weight of evidence used by the International Agency for Research on Cancer. This classification scheme, which produces a qualitative designation from the letter A through the letter E for the overall weight of evidence for carcinogenicity, is based on an amalgam of human and animal data for each suspect carcinogen.

Dose-response assessments are based on data obtained from epidemiological studies, animal experiments, or both. EPA prefers dose-response assessments based on adequate epidemiologic data over those based on animal studies, but because adequate epidemiologic data are usually not available, animal data are often used. The data set from animals which shows the greatest sensitivity is supposed to be given the greater emphasis in statistical modeling because it is possible that human sensitivity is as high or higher than the most sensitive responding animal species. When


counting tumors in animal experiments, EPA risk assessors are supposed to combine benign and malignant tumors, unless the benign tumors are not considered to have the potential to progress to malignancies of the same histogenic origin.

Because animal experiments are usually conducted at higher exposure levels than humans experience, the responses at high doses must be extrapolated to low doses. Although many mathematical models for extrapolation can be used to "fit" data from animal experiments, EPA guidelines state that "in the absence of adequate information to the contrary, the linearized multistage model will be employed." The "linearized" version of the multistage model is conservative because at low doses it compels the response rate to be essentially linear with respect to dose. In the vast majority of cases to date, EPA has used the linearized multistage model to perform low-dose extrapolation. EPA's overall dose-response assessment procedure is designed to produce a plausible upper bound on human risk at low doses.

Exposure assessment is concerned with the contact between a pollutant and a human population, and is measured by the number of people exposed to specific concentrations of a pollutant for a given period of time. For exposure to airborne carcinogens, continuous exposure outdoors over a 70-year lifetime is usually assumed. EPA uses the Human Exposure Model (HEM) to quantify the number of people exposed to air pollutants emitted by stationary sources. The HEM consists of an atmospheric dispersion model covering 160 receptor sites (up to 50 kilometers from the source), and includes meteorological data and census information on population distributions.

Results from HEM are combined with EPA's dose-response assessments to estimate both cancer risks to maximum exposed individuals (MEIs) and annual cancer incidence (often called population risk). These results are combined with the "weight of evidence" classification (A to E) and a qualitative discussion of uncertainties to form the so-called "risk characterization."


B. Risk Assessment and Benefit Estimation

Once the risk assessment is made, it can be used to support two approaches to making benefit estimates: the public health approach and the individual equity approach. The public health approach entails estimating the number of pollution-related cases of disease with and without the regulatory standard in question, with the difference between the two "population risk" estimates being considered the public health benefit of the standard. The individual equity approach entails identifying the citizen with the highest risk of pollution-related disease with and without the standard in question, with the difference in the two "individual risk" estimates being considered the equity benefit of the standard.

Although in practice the public health approach is more widely used, some environmentalists are primarily concerned about the significant risks faced by heavily exposed citizens. EPA uses the concept of the MEI to operationalize the individual risk approach. The MEI is often assumed to live at a factory's fence line for 70 years while breathing outdoor concentrations of pollutants for 24 hours a day. The MEI represents the highest theoretical exposure; it is thus hypothetical — i.e., no person is actually exposed to pollution under the described circumstances.

In our review of the health risks of air toxics, we will give some attention to both measures of health risk. We shall argue below that the motivation for bills such as Title V can be better understood if the individual equity perspective is considered.

C. General Shortcomings of EPA Risk Assessments

In the process of reviewing numerous EPA risk assessments of air toxics and the critiques of these assessments, we have identified some systematic weaknesses in the EPA method that should be recognized. Some of the weaknesses are inherent to the inadequate state of environmental science while others are correctable with available data or alternative modeling procedures. We have identified six systematic weaknesses in the EPA risk assessment method.

1. Omission of Noncancer Endpoints:

Acute illnesses, reproductive effects, immune suppression, liver and kidney damage, hypertension, respiratory illnesses, and

neurological effects are among the noncancer endpoints omitted from EPA's risk assessment.

2. Point Estimates versus Upper Bounds:
Conservative assumptions are intended only to provide an upper bound on the actual cancer risk, which scientists may or may not consider plausible in specific cases.

3. Insufficient Use of Delivered-Dose Information:
Pharmacokinetic data are available that contradict the assumption implicit in the EPA method that the amount of a toxic pollutant that reaches target cells in the body is proportional to the amount of the pollutant inhaled. Such data, if utilized, might result in more accurate risk estimates.20

4. Insufficient Use of Mechanistic Information:
New information regarding differences in biological mechanisms between species could lead to either higher or lower estimates of cancer risk in humans than does EPA's normal procedure.

5. Unrealistic Exposure Assumptions:
EPA's exposure assessment procedures are likely to produce misleading estimates of the exposure reductions that can be accomplished through the air toxics standards envisioned in Title V. Baseline exposure assessments typically assume that people breathe pollutants outdoors for 24 hours per day over the life of a facility, even though people spend the vast majority of their time indoors.

On the other hand, EPA may underestimate the number of people exposed to some pollutants, because only residents within 50 kilometers of the source are considered in exposure modeling.

6. Neglect of Synergisms and Susceptible Groups:
Current EPA methods do not explicitly account for the possibility of synergistic and antagonistic effects of various pollutants (i.e., two carcinogenic pollutants — say, asbestos and tobacco smoke — produce more cancer together than the sum of their individual effects) nor do they account for the possibility that there are human subpopulations that are more sensitive to carcinogenic exposures than the most sensitive animal species that has been tested.21 EPA is currently addressing these issues.


21. A. Finkel, Heterogeneity in Human Susceptibility to Environmental
IV. THE HEALTH RISKS OF AIR TOXICS: AGGREGATE AND COMPARATIVE PERSPECTIVES

In this section we review and evaluate what is known about the magnitude of the health risks due to hazardous air pollutants. We consider both the risks on a population basis (a public health perspective) and the excess lifetime risks faced by heavily exposed individuals (an equity perspective). In performing this review, we provide some perspective on how big various source contributors are; indoor versus outdoor pollution, large stationary versus small area sources, and stationary versus mobile sources. We also examine whether the overall problem is getting larger or smaller. By taking this synoptic view of the problem, we seek to offer a rough indicator of the magnitude of potential health gains to be derived from aggressive implementation of a bill like Title V. We do not, however, address how much of this complex problem would actually be curtailed by legislation because we could find no scientific basis for making such estimates.

Relatively few attempts have been made to assess the overall magnitude of the air toxics problem. Our view is based on three EPA reports that we found to be useful: the “1985 Air Toxics Report,” 22 the “1987 Unfinished Business Report,” 23 and the “1988 Urban Air Mixtures Reports.” 24

Although it is difficult to generalize about trends in human exposure to numerous individual pollutants, the best available evidence is that the overall size of the air toxics problem is getting better rather than worse. The 1985 Air Toxics Report discusses an EPA analysis of monitoring and emissions data that was made in order to evaluate progress between 1970 and 1980 for sixteen pollutants. The estimated cancer incidence rate for these air pollutants in 1980 was less than half the rate for 1970, that is 6.8 per


22. UNITED STATES ENVTL. PROTECTION AGENCY, THE AIR TOXICS PROBLEM IN THE UNITED STATES: AN ANALYSIS OF CANCER RISKS FOR SELECTED POLLUTANTS (May 1985).

23. UNITED STATES ENVTL. PROTECTION AGENCY, UNFINISHED BUSINESS: A COMPARATIVE ASSESSMENT OF ENVIRONMENTAL PROBLEMS (Feb. 1987).

24. UNITED STATES ENVTL. PROTECTION AGENCY, SANTA CLARA VALLEY INTEGRATED ENVIRONMENTAL MANAGEMENT PROJECT STAGE TWO REPORT (Sept. 1987); UNITED STATES ENVTL. PROTECTION AGENCY, KANAWHA VALLEY TOXICS SCREENING STUDY FINAL REPORT (July 1987); UNITED STATES ENVTL. PROTECTION AGENCY, BALTIMORE INTEGRATED ENVIRONMENTAL MANAGEMENT PROJECT: PHASE I REPORT (May 1987); UNITED STATES ENVTL. PROTECTION AGENCY, FINAL REPORT OF THE PHILADELPHIA INTEGRATED ENVIRONMENTAL MANAGEMENT PROJECT (Dec. 1986).
million incidences per year in 1980, compared to 17.5 per million incidences in 1970. Likewise, EPA projects that the mobile source air toxics problem is declining and should be by 1996 40% smaller than it was in 1986.

For both mobile and stationary sources, EPA believes that criteria pollutant programs have done more in the past to reduce air toxics than have programs aimed directly at air toxics. These trends can be expected to continue in the future as new industrial plants (subject to stringent criteria pollutant control) replace older plants and new motor vehicles (also subject to stringent criteria pollutant control) replace old motor vehicles. The temporal picture for air toxics from nonmobile area sources is not clear: we could find no evidence regarding whether the problems from small area sources were getting better or worse.

V. ASSESSMENT OF INDIVIDUAL POLLUTANTS

In this section we summarize what is known about the toxicity of those pollutants which either (a) have already been listed under section 112, or (b) have been added to EPA's "intent-to-list" list. These are also the pollutants that would receive the highest regulatory priority under the provisions of Title V. We also report EPA's cancer risk estimates for several dozen individual pollutants, including both estimated risk to the MEI and estimated national population incidence. As we explain below, published EPA estimates for several of these pollutants are questionable because they do not incorporate the most recent scientific information.

A. Qualitative Indications of Toxicity

Qualitative indications of toxicity for the carcinogenicity (IARC scheme), genotoxicity, teratogenicity, neurotoxicity, and immunotoxicity endpoints have been determined for the hazardous air pollutants considered in section 112. Our analysis of these endpoints indicates that many of these pollutants have positive indications of toxicity for noncancer as well as cancer endpoints. This finding underscores the need to develop risk assessment methods for noncancer endpoints. However, it should be emphasized that such indications of toxicity in experimental animals and occupational exposure settings do not demonstrate human toxicity at normal concentrations of these pollutants found in the ambient air.
B. Cancer Risk Estimates for Individual Pollutants

We examined EPA cancer risk estimates for 23 selected hazardous air pollutants, including the weight-of-the-evidence classification for each pollutant. EPA considers these numbers to be upper bounds on the actual cancer risk to humans (i.e., true risk is unlikely to be larger than the upper bound, is probably smaller, and may be zero). It should be emphasized that EPA's risk assessment method has some serious shortcomings that need to be considered when evaluating this kind of information.

These cancer risk estimates suggest that all hazardous air pollutants do not pose equivalent carcinogenic risks. Differences in exposure, potency, and weight of the evidence combine to make some pollutants much more serious problems than others. It should also be noted that some pollutants with large risks to the MEI (e.g., coke oven emissions) do not pose such large risks to the population as a whole. Likewise, some pollutants pose less significant risks to the MEI (e.g., formaldehyde), yet are a relatively large problem on a population basis.

C. New Scientific Information

The EPA risk estimates reported above for several pollutants should be regarded as especially questionable because they do not take account of the most recent scientific knowledge about dose-response relationships. While we have not undertaken a comprehensive review of the available data on each pollutant, we are aware that the EPA risk assessments of benzene, formaldehyde, gasoline vapor, perchloroethylene and trichloroethylene do not incorporate recent advances in scientific knowledge.

In the case of benzene, new epidemiological information (from the Rinsky cohort) and a more biologically realistic model (the Clement model) have been used to estimate the leukemogenic risks of human exposure to ambient concentrations of benzene. The new data and the new method generate risk estimates that are a factor of ten smaller than those generated by the normal procedure used by EPA. Risk assessors at EPA are now considering this new information.

In the case of formaldehyde, scientists at the Chemical Industry Institute of Toxicology have demonstrated a nonlinearity between the amount of formaldehyde inhaled and the amount of

formaldehyde delivered to DNA in the nasal cavity of rats and monkeys.\textsuperscript{26} The results suggest that EPA risk assessors have overestimated substantially the amount of formaldehyde that is delivered to target cells at normal ambient concentrations in the environment. Again, risk assessors at EPA are now considering this new information.

Some chemicals, notably gasoline vapor and perchloroethylene, produce kidney tumors in male rats that arise from a biological mechanism that has been shown to be unique to the male rat.\textsuperscript{27} As a result, EPA risk estimates and weight-of-the-evidence classifications for these pollutants are now under reconsideration.

Many chemicals, including trichloroethylene, perchloroethylene, and gasoline vapor, appear to produce liver tumors in mice by a nongenotoxic mechanism called peroxisome proliferation. This mechanism is believed to be nonlinear at low concentrations and may involve a threshold.\textsuperscript{28} As a result, EPA risk assessments which assume low-dose linearity may be inappropriate. EPA risk assessors are also considering the evolution of science in regard to peroxisome proliferators.

Overall, policy makers should be aware that the scientific fields of “pharmacokinetics” (fate of chemicals in the body) and “pharmacodynamics” (mechanisms of toxic insult at the cellular level) are rapidly evolving. Scientists have gone beyond long-term animal bioassays and traditional epidemiology and are beginning to elucidate the uptake, distribution, fate and mechanistic activity of toxic chemicals. In light of such progress, legislative approaches that cannot accommodate advances in science should be avoided, and more flexible approaches to regulatory policy should be encouraged.

D. Legislative and Regulatory Implications

In a recent review of 132 federal regulatory decisions in the post-1980 period, Curtis Travis and colleagues uncovered a historical relationship between cancer risk estimates and regulatory

\textsuperscript{26} Starr & Buck, The Importance of Delivered Dose in Estimating Low-Dose Risk From Inhalation Exposure to Formaldehyde, 4 FUNDAMENTAL APPLIED TOXICOLOGY 740 (1984).

\textsuperscript{27} Telephone interview with Dr. James Swenberg, Director of the Department of Biochemical Toxicology and Pathology, Chemical Industry Institute of Toxicology, (Oct. 25, 1988).

\textsuperscript{28} Stott, Chemically Induced Proliferation of Peroxisomes: Implications for Risk Assessment, 8 REGULATORY TOXICOLOGY & PHARMACOLOGY 125 (1988).
decisions. Chemical exposures with lifetime individual risks of cancer in excess of 4 in 1,000 were always regulated. Risks less than 1 in a million were never regulated. Individual risks in between these extremes were regulated when population risk was large and not regulated when population risk was small. Cases with intermediate population risks were resolved based on cost-effectiveness considerations.

We applied the Travis framework to selected hazardous air pollutants to gain some insight into potential regulatory implications. Three of the twenty three pollutants had individual risks in the “must regulate” region. The other twenty pollutants were in the intermediate region to be resolved by consideration of population incidence and cost-effectiveness. None of the pollutants were in the “must not regulate” region. These conclusions are obviously not indisputable since one can question the value judgments embedded in historical regulatory decisions.

VI. SUMMARY AND CONCLUSIONS

This report examines the scientific evidence relevant to assessing the potential health benefits of new legislation aimed at controlling “hazardous” air pollutants. Using a risk assessment framework, the report considers two kinds of benefits: reducing risk to heavily exposed citizens (an equity perspective) and reducing risk to the United States population as a whole (a public health perspective). The report is based on EPA risk assessments and selected information from the peer-reviewed scientific literature. For illustrative purposes, the report focuses on the potential benefits of several provisions in Title V of the Mitchell Bill.

In conclusion, this report makes nine major findings. First, significant control of human exposure to hazardous air pollutants is likely to occur in the future even without new legislation due to state air toxics programs, federal programs aimed at criteria pollutants, and semi-voluntary corporate policies. However, new technology-based legislation (including flexibility to consider costs and benefits) would foster more expeditious and practical federal rules than would result from aggressive implementation of the current health-based statutory authority in section 112 of the Clean Air Act Amendments of 1970.

Second, the health benefits of requiring federal regulation of

200 or so noncriteria pollutants cannot be estimated with any reasonable degree of precision due to insufficient scientific data on toxicity and human exposure. Some (but not all) of this information is now being collected.

Third, the cancer risk assessment methods used by EPA have important shortcomings that need to be understood by policy makers. When hard data are lacking, assumptions are made that are intended to produce an upper bound on actual cancer risk. The risk estimates for specific pollutants may be grossly inflated or, in some cases, too low (despite the conservative assumptions). EPA is now reconsidering its approach to cancer risk assessment in light of new scientific information and several years of difficult experience with such a simplistic approach.

Fourth, the results of EPA risk assessments suggest that hazardous air pollutants are not responsible for a significant proportion of the national cancer toll (e.g., less than 1% of annual cancer mortality is attributable to the forty-five pollutants studied by EPA). Although EPA has estimated that up to 2,000 cancers per year may be attributable to air toxics, the agency's recent urban air mixture studies suggest that this figure is probably too large. Some citizens, however, are exposed to large personal risks of cancer due to air toxics. Hence, the rationale for new legislation should be understood more as a "risk equity" measure than a "public health" measure.

Fifth, for the forty-five pollutants with enough data to permit a cancer risk assessment, it appears that most of the benefits of broad-based legislation would result from control of a handful of pollutants. Congress may want to consider a more targeted legislative approach that concentrates public and private sector resources on the most serious problems.

Sixth, human exposure to hazardous air pollutants is capable of producing various forms of illness besides cancer (e.g., reproductive effects, immune suppression, neurological effects, and kidney damage). Although EPA risk assessments of specific pollutants focus on cancer, the health benefits of emission controls resulting from Title V might include reductions in the incidence of noncancer as well as carcinogenic health effects. New data and methods for assessing noncancer effects are sorely needed.

Seventh, much congressional attention has been devoted to the need for control of air toxics at large industrial (so-called "point") sources. However, our review suggests that indoor sources of air pollution and smaller, more diffuse "area" sources
HAZARDOUS AIR POLLUTANTS

(e.g., wood stoves and dry cleaning operations) account for more emissions than do large industrial sources. New legislative proposals should account for this comparative perspective when considering allocation of scarce administrative resources and the addition of nondiscretionary administrative duties and deadlines.

Eighth, the emissions of hazardous air pollutants from mobile sources, although relatively quite significant, are expected to decline 40% by 1995 due to existing emission control programs. The EPA estimates of cancer risks from mobile sources are highly uncertain and the true risks could be anywhere from zero to several hundred cases per year. EPA is considering revisions to the risk estimates for several key mobile source pollutants in light of new scientific information. If Congress elects to promote methanol as an alternative fuel, emissions of some air toxics (e.g., formaldehyde) may increase in the 1990s.

Finally, the EPA cancer risk assessments for several important pollutants (benzene, formaldehyde, gasoline vapor, perchloroethylene, and trichloroethylene) do not account for recent scientific information about pharmacokinetics and pharmacodynamics. These pollutants account for a substantial fraction of the national cancer risk attributable to hazardous air pollutants. We are encouraged that EPA is considering revision of its risk assessments in response to this new information.