# ON THE PROSPECT OF "DAUBERTIZING" JUDICIAL REVIEW OF RISK ASSESSMENT

#### THOMAS O. MCGARITY\*

## I

#### INTRODUCTION

In *Daubert v. Merrell Dow Pharmaceuticals, Inc.*,<sup>1</sup> the Supreme Court assigned a gatekeeper role to federal district courts hearing cases involving expert testimony. *Daubert* requires trial judges to so acquaint themselves with the scientific underpinnings of expert testimony in common-law litigation that they can determine whether that testimony represents a sufficiently sound application of scientific principles to a sufficiently robust set of scientific data to justify the expert's scientific conclusions.<sup>2</sup> At the same time, the trial judge is obliged to determine whether the scientific testimony is relevant to factual determinations the jury must make—that is, whether the information fits the issues raised by the case. <sup>3</sup> In the decade following the Supreme Court's decision, it has become quite clear that *Daubert* has had a profoundly negative impact on plaintiffs' attorneys' use of common-law torts to hold companies accountable for the adverse effects of their products and byproducts on human health and the environment.<sup>4</sup>

Attorneys for companies successfully using *Daubert* to avoid such accountability now urge the federal courts to assume a similar gatekeeper role in reviewing risk assessments undertaken by federal regulatory agencies. Lawyers for companies subject to federal health, safety, and environmental regulation hope that stringent substantive judicial review will relieve their clients of the burdens of much substantive regulation without the need for troublesome legislative battles they seem unable to win. Such stringent review would require the courts to evaluate the scientific conclusions underlying agency risk assessments with a *Daubert*-inspired "corpuscular" approach.<sup>5</sup>

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<sup>\*</sup> W. James Kronzer Chair in Trial and Appellate Advocacy, University of Texas School of Law.

<sup>1. 509</sup> U.S. 579 (1993).

<sup>2.</sup> *Id.* at 590 & n.9, 593-95. The Court later held that the trial court must play the same gate-keeper role with respect to expert testimony involving engineering conclusions. Kumho Tire v. Carmichael, 526 U.S. 137 (1999).

<sup>3. 509</sup> U.S. at 591, 593-95.

<sup>4.</sup> See infra text accompanying notes 106-09.

<sup>5.</sup> See infra text accompanying note 116.

Assigning a Daubert-like gatekeeper role to courts engaged in judicial review of agency risk assessments is a profoundly bad idea. First, federal judges are not qualified to undertake strict scrutiny of the scientific bases for riskassessment data and analysis. Judges' limited competence in areas involving scientific data and analysis, complex modeling exercises, and large uncertainties is well recognized in administrative law and has been effectively demonstrated by the courts themselves in post-Daubert toxic torts opinions. Second, because risk assessment is not a purely scientific enterprise, "Daubertizing" judicial review of agency risk assessments will bestow upon the courts a policymaking role that is entirely inappropriate for a politically unaccountable institution. Third, the agencies preparing these risk assessments are, as part of the executive branch, performing a function assigned to them by the legislative branch. So the potential for aggrandizing judicial power through intensifying judicial scrutiny of the science-policy determinations underlying agency-prepared risk assessments raises serious institutional issues not present in *Daubert*-style review of expert testimony in private tort litigation. Finally, the "Daubertization" of agency risk assessments would have a predictable impact on regulatory policy running directly counter to the precautionary policies animating most health, safety, and environmental statutes. The proponents of strict judicial risk-assessment scrutiny have a clear, normative agenda in mind. Having failed during the 104th Congress to reign in federal regulatory agencies by enacting sometimes draconian regulatory-reform legislation,<sup>6</sup> regulatory reformers are now attempting to gain regulatory relief in the courts by subtly assigning a more activist role to judges, who are perceived to be more sympathetic to their goals than Congress.<sup>7</sup> The courts should forcefully reject this invitation to reform protective health, safety, and environmental regulation to fit these judges' concepts of the proper role for federal regulation in society.

<sup>6.</sup> See William W. Buzbee, Regulatory Reform or Statutory Muddle: The "Legislative Mirage" of Single Statute Regulatory Reform, 5 N.Y.U. ENVT'L. L.J. 298 (1996); Thomas O. McGarity, Deflecting the Assault: How EPA Survived a "Disorganized Revolution" by "Reinventing" Itself a Bit, 31 Envtl. L. Rep. 11,249 (2001).

<sup>7.</sup> Observing the post-*Daubert* tendency of federal judges to bring about normative changes in the substantive law of torts, Professor Findley argues quite persuasively that the judges "have been using their evidentiary gatekeeper power to squarely align tort law with the conservative causal normative principles of epidemiology, thus moving the law sharply away from the more consumer protective social policies about risk embodied in the safety regulatory system." Lucinda M. Finley, *Guarding the Gate to the Courthouse: How Trial Judges Are Using Their Evidentiary Screening Role to Remake Tort Causation Rules*, 49 DEPAUL L. REV. 335, 336 (1999). The strict judicial scrutiny of scientific evidence implicit in the *Daubert* screening role is similarly an invitation to regulatory reformers on the bench to change substantive health and environmental protection law.

#### DAUBERT AND THE CORPUSCULAR APPROACH TO SCIENTIFIC EVIDENCE

#### A. The Nature of Risk Assessment

Risk assessment is an analytical process that "uses available scientific information on the properties of an agent and its effects in biological systems to provide an evaluation of the potential for harm as a consequence of environmental exposure to the agent."<sup>8</sup> Risk assessment must be distinguished from risk management, which consists of the actions individuals and regulatory agencies take to reduce or eliminate the risks human beings encounter.<sup>9</sup> As currently conceived, risk assessment itself consists of four discrete analytical exercises: hazard assessment, dose-response assessment, exposure assessment, and risk characterization.<sup>10</sup> The enhanced judicial review envisioned by Alan Raul's proposal to Daubertize judicial review of risk assessment is probably most relevant to the hazard assessment function.<sup>11</sup>

#### 1. Hazard Assessment

Hazard assessment is "the process of determining whether exposure to an agent can cause an increase in the incidence of a health condition."<sup>12</sup> This function parallels the general causation determination in toxic tort litigation.<sup>13</sup> Typically, hazard assessment relies upon one or more of four different kinds of information: (1) controlled human experiments; (2) epidemiological studies; (3) animal testing studies; and (4) various studies of chemical structure, reactivity,

<sup>8.</sup> U.S. Envtl. Protection Agency, Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17,960, 17,963 (proposed April 23, 1996) [hereinafter 1996 Proposed Guidelines].

<sup>9.</sup> See COMM. ON THE INSTITUTIONAL MEANS FOR ASSESSMENT OF RISKS TO PUB. HEALTH, NAT'L ACAD. OF SCI., RISK ASSESSMENT IN THE FEDERAL GOVERNMENT: MANAGING THE PROCESS 18-19 (1983) [hereinafter NAS RED BOOK]. As described by the National Academy of Sciences:

Risk management, which is carried out by regulatory agencies under various legislative mandates, is an agency decision-making process that entails consideration of political, social, economic, and engineering information with risk-related information to develop, analyze, and compare regulatory options and to select the appropriate regulatory response to a potential chronic health hazard.

*Id.; see also* U.S. Envtl. Protection Protection Agency, Guidelines for Carcinogen Risk Assessment, 51 Fed. Reg. 33,992, 33,993 (published Sept. 24, 1986) [hereinafter 1986 Final Guidelines] ("Risk management combines the risk assessment with the directives of regulatory legislation, together with socioeconomic, technical, political, and other considerations, to reach a decision as to whether or how much to control future exposure to the suspected toxic agents.").

<sup>10.</sup> NAS RED BOOK, supra note 9, at 3.

<sup>11.</sup> See infra text accompanying note 110; Alan Charles Raul & Julie Zampa Dwyer, "Regulatory Daubert": A Proposal to Enhance Judicial Review of Agency Science by Incorporating Daubert Principles into Administrative Law, 66 LAW & CONTEMP. PROBS. 7 (Autumn 2003).

<sup>12.</sup> NAS RED BOOK, supra note 9, at 19.

<sup>13.</sup> To establish general causation, a plaintiff in a toxic tort action must prove that the substance to which he or she was exposed (and for which exposure the defendant was responsible) is capable of causing the injury for which the plaintiff is seeking compensation. *See In re* Joint E. & S. Dists. Asbestos Litig., 52 F.3d 1124, 1131 (2d Cir. 1995); Margaret A. Berger, *Eliminating General Causation: Notes Towards a New Theory of Justice and Toxic Torts*, 97 COLUM. L. REV. 2117, 2122 (1997).

mutagenicity, and DNA damage and repair.<sup>14</sup>

*a. Human tests.* Ordinarily, controlled human tests are the best evidence of toxicity to humans.<sup>15</sup> But, as a practical matter, cost and ethical considerations severely limit the extent to which controlled human studies are available for determining the toxicity of potentially toxic agents.<sup>16</sup> This is so in the case of carcinogenicity, for example, because it can take decades to manifest itself and is often fatal. The same is true for reproductive and developmental toxicity, the assessment of which raises serious ethical questions concerning the ability of the subject to consent to testing.<sup>17</sup>

*b. Epidemiological studies.* The next-best evidence for use in hazard assessment comes from epidemiological studies of human exposures occurring outside the context of a controlled experiment.<sup>18</sup> Epidemiology is a relatively new science, and epidemiologists have only in the last quarter century begun to develop consistent definitions and prescribe evaluative criteria for epidemiological studies.<sup>19</sup> An epidemiological study consists of a statistical comparison of human beings who have received a higher-than-normal exposure to a particular agent with others who have received little or none.<sup>20</sup> The two broad types of epidemiological studies are cohort studies and case-control studies. In cohort studies, groups of individuals who have received high exposures to the substance being studied are identified and compared to groups of similarly situated individuals who have received low exposures to determine possible differences in the occurrence of particular diseases.<sup>21</sup> Cohort studies can be conducted prospectively—by identifying the two cohorts in advance of the exposure and fol-

21. 1996 Proposed Guidelines, *supra* note 8, at 17,973; ROTHMAN & GREENLAND, *supra* note 16, at 79-91 (discussing generally cohort studies, data analysis study design, and related issues of classification and measurement).

<sup>14.</sup> See NAS RED BOOK, supra note 9, at 20 ("Four general classes of information may be used in this step: epidemiologic data, animal-bioassay data, data on in-vitro effects, and comparisons of molecular structure."); WORK GROUP ON RISK ASSESSMENT, INTERAGENCY REGULATORY LIAISON GROUP, (IRLG), SCIENTIFIC BASES FOR IDENTIFICATION OF POTENTIAL CARCINOGENS AND ESTIMATION OF RISKS, 44 Fed. Reg. 39,858 (1979).

<sup>15.</sup> U.S. Envtl. Protection Agency, Guidelines for Reproductive Toxicity Risk Assessment, 61 Fed. Reg. 56,274, 56,278-79, 56,309 (1996) [hereinafter Reproductive Toxicity Guidelines]; U.S. Envtl. Protection Agency, Guidelines for Developmental Toxicity Risk Assessment, 56 Fed. Reg. 63,798, 63,809 (1991) [hereinafter Developmental Toxicity Guidelines].

<sup>16.</sup> KENNETH J. ROTHMAN & SANDER GREENLAND, MODERN EPIDEMIOLOGY 72 (2d ed. 1998); *see also* NAS RED BOOK, *supra* note 9, at 12 ("Ethical considerations prevent deliberate human experimentation with potentially dangerous chemicals.").

<sup>17.</sup> See Lainie Friedman Ross, Children as Research Subjects: A Proposal to Revise the Current Federal Regulations Using a Moral Framework, 8 STAN. L. & POL'Y REV. 159 (Winter 1997); Michael J. Loscialpo, Note, Nontherapeutic Human Research Experiments on Institutionalized Mentally Retarded Children: Civil Rights and Remedies, 23 NEW ENG. J. ON CRIM. & CIV. CONFINEMENT 139 (1997).

<sup>18.</sup> Reproductive Toxicity Guidelines, *supra* note 15, at 56,297.

<sup>19.</sup> ROTHMAN & GREENLAND, supra note 16, at 4.

<sup>20. 1996</sup> Proposed Guidelines, *supra* note 8, at 17,972 ("The goals of cancer epidemiology are to identify differences in cancer risk between different groups in a population or between different populations, and then to determine the extent to which these differences in risk can be attributed causally to specific exposures to exogenous or endogenous factors.").

lowing the groups through time—or retrospectively—through the use of historical records.<sup>22</sup> In case-control studies, groups of similarly situated individuals with and without a particular disease are compared to determine the extent of any differences in exposure to the substance being studied.<sup>23</sup> If the group with the disease has a higher degree of exposure, a statistical association between the exposure and the disease might exist.

The determinant of cause and effect in epidemiology is the concept of relative risk. The epidemiologist compares the frequency of the disease in the exposed population with the frequency in the unexposed population to determine whether relative frequency is increased by exposure.<sup>24</sup> If the data can be arranged so that it is possible to observe groups with varying degrees of exposure, then the existence of a dose-response relationship increases the epidemiologist's confidence in the results.<sup>25</sup>

Epidemiologists and other scientists frequently invoke tests of statistical significance to assess chance as a possible explanation for the observed data.<sup>26</sup> If the relationship between the exposure and a particular result is not statistically significant, then chance may explain the result. The statistical significance threshold level is determined by the scientist; it is a policy choice that depends upon the scientist's willingness to accept that what appears to be a cause-effect relationship may in fact be the product of chance. Thus, the fact that an association is not statistically significant at a given level does not negate a causeeffect relationship; it merely suggests that the probability of a chance result is sufficiently high in the scientist's opinion to preclude a cause-effect conclusion.<sup>27</sup>

The statistical power of an epidemiological study to support conclusions about cause-effect relationships depends upon four factors: (1) the size of the study group; (2) the level and duration of exposure; (3) the frequency of the relevant disease outcome in the general population; and (4) the level of excess risk to be identified.<sup>28</sup> The confidence with which one can draw conclusions from a study in which no adverse effects are detected depends entirely on its power to detect such effects.<sup>29</sup> Unfortunately, cost considerations generally lead to epidemiological studies that are not especially powerful. Therefore, a negative epidemiological study rarely warrants a strong conclusion that human exposure to a substance and disease share no cause-effect relationship.<sup>30</sup>

<sup>22. 1996</sup> Proposed Guidelines, supra note 8, at 17,973.

<sup>23.</sup> *Id.*; ROTHMAN & GREENLAND, *supra* note 16, at 93-114 (discussing study design and variants of case-control study design, and comparison to cohort studies).

<sup>24.</sup> See James J. Schlesselman, Proof of Cause and Effect in Epidemiologic Studies: Criteria for Judgment, 16 PREVENTIVE MED. 195, 197 (1987).

<sup>25.</sup> Id. at 201.

<sup>26.</sup> See id. at 196-97.

<sup>27.</sup> See id. at 197.

<sup>28.</sup> Reproductive Toxicity Guidelines, *supra* note 15, at 56,299; Developmental Toxicity Guidelines, *supra* note 15, at 63,809.

<sup>29.</sup> Reproductive Toxicity Guidelines, supra note 15, at 56,299.

<sup>30. 1996</sup> Proposed Guidelines, *supra* note 8, at 17,967 ("[W]hen cancer effects are not found in an exposed human population, this information by itself is not generally sufficient to conclude that the

The consistency of a result across different places, circumstances, and times enhances the confidence in a positive cause-effect conclusion.<sup>31</sup> The strength of the association, or the magnitude of the relative risk, is also an important factor.<sup>32</sup> Although the demand for strength varies, many epidemiologists insist upon a relative risk of at least 2.0 before they are willing to consider the possibility of a cause-effect relationship.<sup>33</sup> The specificity of the end-point is likewise relevant. If, for example, smoking is associated with squamous-cell carcinoma, cohort studies focusing upon both squamous-cell carcinomas and adenocarcinomas are not as specific as those focusing exclusively on the former.

Other important criteria in evaluating an epidemiological study's conclusions are coherence and biological plausibility. A cause-effect conclusion is not well-founded if it is not "consistent with the known facts of the natural history and biology of the disease."<sup>34</sup> On the other hand, if the conclusion is plausible, given the existing knowledge about how chemical agents interact with human cells, the cause-effect conclusion might be warranted. Animal studies and human experiments can lend great support to any conclusion that might be drawn from epidemiological analysis.<sup>35</sup> It is even appropriate for an epidemiologist to draw support from analogous systems.<sup>36</sup> For example, epidemiologists conducting studies on the health risks from passive exposure to environmental tobacco smoke might draw on the vast body of epidemiology on the health effects of active smoking. Reliance upon analogy is most appropriate when the two exposures being compared are sufficiently similar with respect to disease producing factors, a matter that is frequently highly debatable.

In recent years, scientists have learned how to enhance the power of individual epidemiological studies through meta-analysis of the data from several such studies. Meta-analysis is the process of statistically combining the results of many studies dealing with similar diseases and risk factors to yield additional information enhancing the epidemiologist's understanding of the associations between potentially toxic agents and their effects.<sup>37</sup> Many epidemiological studies (especially case-control studies) observe only a very small number of

36. Id.

agent poses no carcinogenic hazard to this or other populations of potentially exposed humans... because epidemiologic studies usually have low power to detect and attribute responses.").

<sup>31.</sup> Schlesselman, *supra* note 24, at 200.

<sup>32.</sup> Id.

<sup>33.</sup> The relative risk detected in an epidemiological study is the ratio of the incidence of the disease of interest in the exposed population to the incidence in the unexposed population. *See* MICHAEL D. GREEN, BENDECTIN AND BIRTH DEFECTS 30-34 (1996).

<sup>34.</sup> Schlesselman, *supra* note 24, at 200.

<sup>35.</sup> Id. at 201.

<sup>37. 1996</sup> Proposed Guidelines, *supra* note 8, at 17,974 ("When utilized appropriately, meta-analysis can enhance understanding of associations between sources and their effects that may not be apparent from examination of epidemiologic studies individually."); ROTHMAN & GREENLAND, *supra* note 16, at 643-73 (discussing generally meta-analysis techniques and data interpretation, quantitative methods for reviewing other epidemiologic studies and results reported in literature); Erica Beecher-Monas, *A Ray of Light for Judges Blinded by Science: Triers of Science and Intellectual Due Process*, 33 GA. L. REV. 1047, 1071-72 (1999) (observing that "[m]eta-analysis, which averages the results of many individual studies, is a way of reducing sampling error by increasing the size of the sample").

individuals. These studies therefore lack sufficient power to detect modest increases in relative risk that could be significant for an agent to which there is widespread human exposure. Meta-analysis increases the observational power of these studies by combining their results.<sup>38</sup>

In obtaining and analyzing epidemiological data, epidemiologists must carefully avoid "bias"<sup>39</sup>— in this context, "a systematic departure of results from the correct values as a consequence of errors in design or investigational technique."<sup>40</sup> Unfortunately, limitations in the data sources ordinarily available to epidemiologists can introduce unintended bias,<sup>41</sup> as can a variety of other influences:

Bias can arise from several sources, including noncomparability between populations of factors such as general health, diet, lifestyle, or geographic location; differences in the way case and control individuals recall past events; differences in data collection that result in unequal ascertainment of health effects in the populations; and unequal follow-up of individuals.<sup>42</sup>

The effect of bias on the validity of conclusions that the scientist may draw from epidemiological studies can range from negligible to devastating.<sup>43</sup>

One especially prevalent form of bias in epidemiology is the confounding factor, a risk factor for the disease at issue that is associated with the exposure under study in the source population and that is not otherwise affected by the exposure or the disease.<sup>44</sup> If confounding factors unrelated to the exposure of the substance at issue can account for the observed differences in disease rates, it might be inappropriate to conclude that the exposure to the substance is responsible for those differences.<sup>45</sup> Under such circumstances, epidemiologists must adjust their study designs or statistical analyses to reduce the effect of con-

- 39. See generally GREEN, supra note 33.
- 40. Schlesselman, supra note 24.

<sup>38.</sup> In the words of the National Academy of Sciences' Committee on the Epidemiology of Air Pollution, "[i]n essence, meta-analysis assumes that the results of studies can themselves be treated as random variables with predictable distributions." COMM. ON THE EPIDEMIOLOGY OF AIR POLLUTANTS, NAT'L RES. COUNCIL, EPIDEMIOLOGY AND AIR POLLUTION 218 (1985). Meta-analysis is not appropriate, however, when the studies being combined are not comparable or contain substantial confounding or other biases for which the statistical analysis does not adjust. 1996 Proposed Guidelines, *supra* note 8, at 17,974 (noting that meta-analysis may not be useful "when there are substantial confounding or other biases that cannot be adjusted for in the analysis").

<sup>41.</sup> See ROTHMAN & GREENLAND, supra note 16, at 119 (citing one study that "listed dozens of possible biases that can distort the estimation of an epidemiologic measure"); Schlesselman, supra note 24, at 195 (noting that the "[c]auses of bias are legion"); see also Joseph Sanders, Scientific Validity, Admissibility, and Mass Torts After Daubert, 78 MINN. L. REV. 1387, 1420-21 (1994) (explaining recall bias in cases involving pharmaceuticals).

<sup>42. 1996</sup> Proposed Guidelines, *supra* note 8, at 17,973; see also ROTHMAN & GREENLAND, *supra* note 16, at 119-33 (describing three general types of bias: selection bias, confounding, and information bias).

<sup>43.</sup> Schlesselman, supra note 24, at 195.

<sup>44.</sup> ROTHMAN & GREENLAND, *supra* note 16, at 123-25; *see also* 1996 Proposed Guidelines, *supra* note 8, at 17,974; Schlesselman, *supra* note 24, at 196 (observing that "confounding variables are those that satisfy three conditions: (a) they are determinants or correlates of the disease under study; (b) they are unequally distributed among exposed and unexposed individuals; (c) they are not consequences of the study exposure").

<sup>45.</sup> ROTHMAN & GREENLAND, *supra* note 16, at 59-62, 120-25.

founding factors.<sup>46</sup> When complete adjustment is not possible, however, it might still be able to draw conclusions about an agent's ability to cause disease if multiple studies with different potential confounders yield similar results.<sup>47</sup>

*c.* Animal studies. Because human testing is usually impossible, and because epidemiological studies are very difficult to undertake and reveal important information only after harm to human beings has already occurred, regulatory agencies rely heavily upon laboratory animal tests to evaluate the toxicity of chemicals.<sup>48</sup> For example, under the Federal Insecticide, Fungicide, and Rodenticide Act ("FIFRA"),<sup>49</sup> the Environmental Protection Agency ("EPA") requires pesticide registrants to produce a basic set of core animal testing studies prior to marketing their products.<sup>50</sup> Most agencies rely upon the default assumption that "positive effects in animal cancer studies indicate that the agent under study can have carcinogenic potential in humans."<sup>51</sup> Positive results in two or more animal species is strong evidence of human carcinogenicity.<sup>52</sup> Likewise, two or more negative animal tests support the conclusion that an agent does not cause cancer in humans—in the absence of human data to the contrary.<sup>53</sup>

However, despite the obvious importance of animal studies to toxicological evaluation, parties opposed to agency reliance on animal studies always raise the issue of inter-species extrapolation. Human beings do not always metabo-

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Id.

<sup>46. 1996</sup> Proposed Guidelines, supra note 8, at 17,974.

<sup>47.</sup> *Id.* ("If consistent increases in cancer risk are observed across a collection of studies with different confounding factors, the inference that the agent under investigation was the etiologic factor is strengthened, even though complete adjustment for confounding factors cannot be made and no single study supports a strong inference.").

<sup>48.</sup> NAS RED BOOK, *supra* note 9, at 22 ("Limitations [on epidemology] require reliance on less direct evidence that a health hazard exists.").

<sup>49. 7</sup> U.S.C. §§ 136-136y (2000).

<sup>50.</sup> Purposes of the Registration Data Requirements, 40 C.F.R. § 158.202(e) (2003). EPA explains the utility of one type of animal study in this way:

<sup>(</sup>e) Hazard to humans and domestic animals. Data required to assess hazards to humans and domestic animals are derived from a variety of acute, subchronic and chronic toxicity tests, and tests to assess mutagenicity and pesticide metabolism.

<sup>(6)</sup> Metabolism studies. Data from studies on the absorption, distribution, excretion, and metabolism of a pesticide aid in the valuation of test results from other toxicity studies and in the extrapolation of data from animals to man. The main purpose of metabolism studies is to produce data which increase the Agency's understanding of the behavior of the chemical in its consideration of the human exposure anticipated from intended uses of the pesticide.

<sup>51. 1996</sup> Proposed Guidelines, *supra* note 8, at 17,967.

<sup>52.</sup> Id. ("[I]f no adequate human data are present, positive effects in animal cancer studies are a basis for assessing the carcinogenic hazard to humans.").

<sup>53.</sup> Id. ("When cancer effects are not found in well-conducted animal cancer studies in two or more appropriate species and other information does not support the carcinogenic potential of the agent, these data provide a basis for concluding that the agent is not likely to possess human carcinogenic potential, in the absence of human data to the contrary.").

lize toxic agents in the same way laboratory animals do, and human organs and reproductive systems are by no means identical to those of mice and rats.<sup>54</sup> The dose an animal receives in a standard feeding experiment can be difficult to compare to real-world human exposure, though pharmacokinetic studies on the animals and human beings can facilitate such comparisons.<sup>55</sup> The usefulness of laboratory animal data can therefore be enhanced considerably by pharmacokinetic studies on the animals and on human beings.<sup>56</sup> In short, "there are no doubt occasions in which observations in animals may be of highly uncertain relevance to humans."<sup>57</sup> The decision to rely upon animal studies, therefore, represents a policy judgment that the informational value of animal studies outweighs the attendant uncertainties of inter-species extrapolation.<sup>58</sup>

*d.* Other kinds of studies. In recent years, scientists have developed less expensive carcinogenicity tests that probe the mutagenic potential of an agent under in vitro conditions.<sup>59</sup> Although such tests can provide some evidence of carcinogenicity, they are not sufficiently accurate to support such a finding independently.<sup>60</sup> Risk assessors can also compare the chemical structure of an agent to the chemical structure of a substance with a known toxicity to assess the potential human hazard.<sup>61</sup> But since it is well known that two chemicals having almost identical chemical structure is even weaker support for a hazard assessment than in vitro tests.<sup>63</sup> Consequently, in-vitro tests and structure-activity

<sup>54.</sup> Developmental Toxicity Guidelines, supra note 15, at 63,798.

<sup>55.</sup> Reproductive Toxicity Guidelines, *supra* note 15, at 56,302; Developmental Toxicity Guidelines, *supra* note 15, at 63,813. Pharmacokinetic studies provide detailed information on exposures to pesticides and their metabolites in particular human and animal organs or tissues. *Id.* 

<sup>56.</sup> Reproductive Toxicity Guidelines, *supra* note 15, at 56,302; Developmental Toxicity Guidelines, *supra* note 15, at 63,813. In the context of reproductive and developmental toxicity, pharmacokinetic studies yield useful information on "absorption, half-life, steady-state and/or peak plasma concentrations, placental metabolism and transfer, excretion in breast milk, comparative metabolism, and concentrations of the parent compound and metabolites," all of which can be quite useful in risk assessment. Developmental Toxicity Guidelines, *supra* note 15, at 63,813; *see also* Reproductive Toxicity Guidelines, *supra* note 15, at 56,302. For example, a good pharmacokinetic study that yields data on the dose received by the particular organ or tissue of concern can aid in comparing the animal testing dose with human exposure. Developmental Toxicity Guidelines, *supra* note 15, at 63,813.

<sup>57.</sup> NAS RED BOOK, supra note 9, at 22.

<sup>58.</sup> See Thomas O. McGarity, Substantive and Procedural Discretion in Administrative Resolution of Science Policy Questions: Regulating Carcinogens in EPA and OSHA, 67 GEO. L.J. 729, 743-44 (1979).

<sup>59.</sup> See NAS RED BOOK, supra note 9, at 22 ("Considerable experimental evidence supports the proposition that most chemical carcinogens are mutagens and that many mutagens are carcinogens.").

<sup>60.</sup> *Id.* at 23 ("Such data, in the absence of a positive animal bioassay, are rarely, if ever, sufficient to support a conclusion that an agent is carcinogenic.").

<sup>61.</sup> Id. ("Comparison of an agent's chemical or physical properties with those of known carcinogens provides some evidence of potential carcinogenicity.").

<sup>62.</sup> U.S. ENVTL. PROTECTION AGENCY, GUIDANCE FOR IDENTIFYING PESTICIDE CHEMICALS AND OTHER SUBSTANCES THAT HAVE A COMMON MECHANISM OF TOXICITY 4, 8 (1999), *available at* http://www.epa.gov/fedrgstr/ERA-PEST/1999/February/Day-05/6055.pdf; *see* Notice of Availability, 64 Fed. Reg. 5796 (Feb. 5, 1999).

<sup>63.</sup> NAS RED BOOK, *supra* note 9, at 23 ("[S]uch studies are best used to identify potential carcinogens for further investigation and may be useful in priority-setting for carcinogenicity testing.").

analyses play very modest roles in most risk assessments, and agencies rarely rely heavily upon such studies to support regulatory actions.<sup>64</sup>

2. Dose-Response Assessment

Dose-response assessment is "the process of characterizing the relation between the dose of an agent administered or received and the incidence of an adverse health effect in exposed populations and estimating the incidence of the effect as a function of human exposure to the agent."<sup>65</sup> Dose-response assessment typically "requires extrapolation from high to low dose and extrapolation from animals to humans."<sup>66</sup> Such extrapolations typically involve complex mathematical models themselves based upon critical assumptions about the interactions between chemicals and the human body. As scientists have learned more about interactions between chemicals and DNA, carcinogenesis doseresponse models have been modified to reflect new scientific understandings.<sup>67</sup> Even so, the models are still highly dependent upon assumptions. A risk assessor's choice among available models is usually driven as much by regulatory policy as by some underlying understanding of human physiology and toxicology.<sup>68</sup>

#### 3. Exposure Assessment

The extent of human exposure to an environmental contaminant is the third critical piece of the risk assessment puzzle.<sup>69</sup> A properly conducted exposure assessment "identifies human populations exposed or potentially exposed to an agent, describes their composition and size, and presents the types, magnitudes, frequencies, and durations of exposure to the agent."<sup>70</sup> An accurate exposure assessment for most chemical substances must include an assessment of dietary, dermal, and inhalation exposures to the agent. Since "there is no single approach to exposure assessment that is appropriate for all cases,"<sup>71</sup> the exposure component of a risk assessment can come from "monitoring information,"

<sup>64. 1996</sup> Proposed Guidelines, *supra* note 8, at 17,979; 1986 Final Guidelines, *supra* note 9, at 33,993.

<sup>65.</sup> *Id.* at 19.

<sup>66.</sup> *Id.* at 20.

<sup>67.</sup> *See, e.g.*, National Primary Drinking Water Regulations; Arsenic and Clarifications to Compliance and New Source Contaminants Monitoring, 65 Fed. Reg. 38,888, 38,901 (proposed June 22, 2000) (to be codified at 40 C.F.R. pts. 141 and 142) (reviewing conclusions of a panel assembled by the National Research Council and an expert panel assembled by EPA on the shape of the dose-response curve for arsenic).

<sup>68.</sup> See National Primary Drinking Water Regulations; Arsenic and Clarifications to Compliance and New Source Contaminants Monitoring, 66 Fed. Reg. 6976, 7004 (Jan. 20, 2001) (to be codified at 40 C.F.R. pts. 9, 141, and 142) ("The use of a linear procedure to extrapolate from a higher, observed data range to a lower range beyond observation is a science-policy approach that has been in use by Federal agencies for four decades."); McGarity, *supra* note 58, at 732.

<sup>69.</sup> See NAS RED BOOK, supra note 9, at 20 (defining exposure assessment as "the process of measuring or estimating the intensity, frequency, and duration of human exposures to an agent currently present in the environment").

<sup>70.</sup> Developmental Toxicity Guidelines, *supra* note 15, at 63,799.

<sup>71. 1986</sup> Final Guidelines, supra note 9, at 33,998.

modeling results, and/or reasoned estimates."<sup>72</sup> Because of the dearth of empirical data in cases of occupational and environmental exposures, risk assessors frequently rely upon mathematical models to assess exposure.<sup>73</sup> Any "assumptions, approximations, and uncertainties" inherent in the application of the model to existing data should be clearly stated because they are likely to "have a major effect on the risk assessment."<sup>74</sup>

#### 4. Risk Characterization

An agency planning to employ risk assessments in its regulatory decisions must be prepared to explain those assessments to the regulated community and to the general public. Risk characterization is "the process of estimating the incidence of a health effect under the various conditions of human exposure described in exposure assessment."<sup>75</sup> In addition to combining hazard and exposure assessments into a single risk assessment, a risk characterization should evaluate the overall quality of the risk assessment, describe in understandable terms the nature and extent of the potential harm, and convey a sense of the uncertainties that surround any quantitative statements.<sup>76</sup> In particular, a risk characterization should identify any assumptions or inferences that the assessor employs and the policy or scientific basis for such inferences. When scientists have not reached a consensus about a particular issue—as is often the case—the agency should present both sides of the issue and explain the policy reasons for its conclusion.<sup>77</sup>

5. The "Weight-of-the-Evidence" Approach to Risk Assessment

In the final analysis, the health and the environmental risks posed by toxic agents cannot be proven with "the compelling certainty that is reached through mathematical reasoning."<sup>78</sup> Epidemiological proof is ephemeral in nature and therefore subject to much disagreement; there is no perfect epidemiological study.<sup>79</sup> In the real world of physical constraints and economic limitations, identifying confounding factors and other sources of bias is an easy task. It is possible that "the most one can ever claim for a study is that it provides evidence

<sup>72.</sup> Id.

<sup>73.</sup> NAS RED BOOK, *supra* note 9, at 27 ("[T]ypically[,] exposure data are incomplete and must be estimated.").

<sup>74. 1986</sup> Final Guidelines, *supra* note 9, at 33,998. *See generally* Thomas O. McGarity, *Politics by Other Means: Law, Science, and Policy in EPA's Implementation of the Food Quality Protection Act*, 53 ADMIN. L. REV. 103, 130-33 (2001) (discussing the difficulties of conducting accurate exposure assessments for pesticide exposures).

<sup>75.</sup> NAS RED BOOK, *supra* note 9, at 20; *see also* 1996 Proposed Guidelines, *supra* note 8, at 17,971 ("The risk characterization process first summarizes findings on hazard, dose response, and exposure characterizations, then develops an integrative analysis of the whole risk case."); Developmental Toxicity Guidelines, *supra* note 15, at 63,819.

<sup>76.</sup> Reproductive Toxicity Guidelines, *supra* note 15, at 56,308; Developmental Toxicity Guidelines, *supra* note 15, at 63,819; 1986 Final Guidelines, *supra* note 9, at 33,993.

<sup>77.</sup> Developmental Toxicity Guidelines, supra note 15, at 63,819.

<sup>78.</sup> Schlesselman, supra note 24, at 198.

<sup>79.</sup> Id. at 199.

which may be construed as supporting or refuting certain explanations for the findings."<sup>80</sup> Thus, in evaluating epidemiological proof, "what we should seek is not a criterion of proof, but rather *aides de judgment*—guides to assist weighing study evidence and reaching informed opinion."<sup>81</sup>

Most agencies have adopted a weight-of-the-evidence approach to assessing human health risks.<sup>82</sup> Under this approach, the agency considers all proffered studies and determines the weight to be afforded each on the basis of identified strengths and weaknesses. Some studies are so poorly conducted that they are entitled to no weight at all. Many studies, otherwise flawed in one or more respects, may nonetheless be appropriately considered to the extent they add to or detract from conclusions based upon more reliable studies.<sup>83</sup> Animal studies are properly considered under the weight-of-the-evidence approach, as are meta-analyses of epidemiological studies that might be flawed to some extent.<sup>84</sup>

81. Id.

82. EPA explained the weight-of-the-evidence approach in the context of carcinogen risk assessment as follows:

Judgment about the weight of evidence involves considerations of the quality and adequacy of data and consistency of responses induced by the agent in question. The weight of evidence judgment requires combined input of relevant disciplines. Initial views of one kind of evidence may change significantly when other information is brought to the interpretation. For example, a positive animal carcinogenicity finding may be diminished by other key data; a weak association in epidemiologic studies may be bolstered by consideration of other key data and animal findings. Factors typically considered are illustrated in figures below. Generally, no single weighing factor on either side determines the overall weight. The factors are not scored mechanically by adding pluses and minuses; they are judged in combination.

1996 Proposed Guidelines, supra note 8, at 17,981.

83. See Sanders, supra note 41, at 1390 (noting that "scientific validity has multiple meanings and is always a matter of degree").

84. EPA's 1986 Carcinogen Risk Assessment Guidelines presented a system for stratifying the agency's weight-of-the-evidence judgment based upon the existing studies. Drawing heavily upon a classification scheme used by the International Agency for Research on Cancer, EPA divided chemical agents into the following categories:

- (1) Group A Carcinogenic to Humans;
- (2) Group B Probably Carcinogenic to Humans;
- (3) Group C Possibly Carcinogenic to Humans;
- (4) Group D Not Classifiable as to Human Carcinogenicity; and
- (5) Group E Evidence of Non-Carcinogenicity for Humans.

1986 Final Guidelines, supra note 9, at 33,996.

According to the classification scheme, the risk assessor puts an agent in Group A if there is "sufficient evidence from epidemiologic studies to support a causal association between exposure to the agents and cancer." *Id.* at 34,000. Epidemiological evidence is sufficient if it indicates a causal relationship between the agent and human cancer. *Id.* at 33,999.

Group B includes agents for which the weight of evidence of human carcinogenicity based on epidemiologic studies is limited, and agents for which the weight of evidence of carcinogenicity based on animal studies is sufficient. Epidemiological evidence is limited if it indicates "that a causal interpretation is credible, but that alternative explanations, such as chance, bias, or confounding, could not adequately be excluded." *Id.* Animal studies are sufficient if they indicate there is "an increased incidence of malignant tumors or combined malignant and benign tumors (a) in multiple species or strains; or (b) in multiple experiments (*e.g.*, with different routes of administration or using different dose levels); or (c) to an unusual degree in a single experiment with regard to high incidence, unusual site or type of tumor, or early age at onset." *Id.* 

<sup>80.</sup> Id.

Clearly, the tools available for chemical risk assessment are quite rudimentary. Gaps in existing knowledge about how chemicals interact with the human body are still quite large, and the hazard identification enterprise is therefore laden with uncertainty. Because there is no such thing as perfect hazard identification, there can be no definitive proof that an environmental agent causes a particular disease.<sup>85</sup> Thus, if definitive proof is the goal, risk assessment is simply the wrong tool.

#### B. Daubert and Its Progeny

As plaintiffs' toxic tort actions moved beyond mesothelioma and other signature diseases, potential defendants and their trade associations began to worry about the possibility of such actions changing the way they do business.<sup>86</sup> Business groups and conservative foundations created "scores of coalitions and task forces"<sup>87</sup> and began funding academic research aimed at limiting frivolous lawsuits.<sup>88</sup> In his well-publicized polemic *Galileo's Revenge*, Manhattan Institute fellow Peter Huber argued that plaintiffs' claims that exposure to toxic substances causes disease are frequently based upon what he termed "junk science."<sup>89</sup> Although the empirical basis for this criticism was never convincingly established,<sup>90</sup> Vice President Dan Quayle's Council on Competitiveness drafted

85. NAS RED BOOK, *supra* note 9, at 19 ("Although the question of whether a substance causes cancer or other adverse health effects is theoretically a yes–no question, there are few chemicals on which the human data are definitive.").

Group C includes "agents with limited evidence of carcinogenicity in animals in the absence of human data." *Id.* at 34,000. Group D includes agents "with inadequate human and animal evidence of carcinogenicity or for which no data are available." *Id.* Group E is limited to "agents that show no evidence for carcinogenicity in at least two adequate animal tests in different species or in both adequate epidemiologic and animal studies." *Id.* 

The guidelines suggest that agents in Groups A and B are the primary candidates for quantitative risk assessment. Agents in Group C are appropriate candidates for quantitative risk assessment on a case-by-case basis. *Id.* at 33,996. In general, "estimates based on adequate human epidemiologic data are preferred over estimates based on animal data." *Id.* at 33,997.

<sup>86.</sup> See Heidi Li Feldman, Science and Uncertainty in Mass Exposure Litigation, 74 TEX. L. REV. 1 (1995).

<sup>87.</sup> Maxwell Glen, Congress Joins the Hue and Cry Over Liability Insurance Crisis, 18 NAT'L J. 380 (1986).

<sup>88.</sup> See Burt Solomon, Finger-Pointing Distinguishes Attempts to Fix Blame for Liability Crisis, 18 NAT'L J. 378 (1986) (identifying the American Tort Reform Association as "a coalition of policyholders assembled last month to lobby for changes in tort law"). The American Tort Reform Association gradually evolved into a fully funded American Tort Reform Foundation with an active website. See http://www.atrafoundation.org (last visited Apr. 6, 2003).

<sup>89.</sup> PETER W. HUBER, GALILEO'S REVENGE: JUNK SCIENCE IN THE COURTROOM (1991); see also W. John Moore, Peter Huber; Free-Lance Critic Hits Shackles of Regulation, 18 NAT'L J. 2797, (1986) (quoting counsel for the Products Liability Alliance, who refers to Huber's work as "the intellectual underpinning" of the tort reform effort of the mid-1980's).

<sup>90.</sup> Professor Huber's mostly anecdotal accounts were heavily relied upon by the tort reformers, but they were harshly reviewed by academics and practitioners. See Robert F. Blomquist, Science, Toxic Tort Law, and Expert Evidence: A Reaction to Peter Huber, 44 ARK. L. REV. 629 (1991); Kenneth J. Chesebro, Peter Huber's Junk Scholarship, 42 AM. U. L. REV. 1637 (1993); John F. Duffy, The FCC and the Patent System: Progressive Ideals, Jacksonian Realism, and the Technology of Regulation, 71 U. COLO. L. REV. 1071, 1076-77 (contrasting Huber's praise of the common law with his criticisms);

fifty proposals for reforming the civil justice system, six of which related to tightening judicial scrutiny of scientific expert testimony.<sup>91</sup> The overtly political purpose of these proposed reforms was to enhance the global competitiveness of U.S. companies by relieving them of the threat of expensive and occasionally ruinous civil liability.<sup>92</sup>

*Daubert v. Merrell Dow Pharmaceuticals, Inc.*<sup>93</sup> gave the Supreme Court an opportunity to address the junk science issue directly.<sup>94</sup> Since nearly all causation claims in toxic tort litigation necessarily rest on expert testimony,<sup>95</sup> one way to ensure the invalidity of such claims—and, consequentially, to reduce the incidence of successful lawsuits—is to raise the bar on admissibility of expert testimony. Unlike the Quayle Commission's tort reform initiative, which precipitated a loud political debate and ultimately failed,<sup>96</sup> the Supreme Court stemmed the daunting flow of resource-intensive toxic tort lawsuits through a politically invisible interpretation of the words "scientific" and "knowledge" in the Federal Rules of Evidence. Many observers believe that this is exactly what the Court had in mind in the *Daubert* and its progeny.<sup>97</sup>

The Supreme Court in *Daubert* declined to employ the *Frye* "general acceptance in the scientific community" standard for admitting expert testimony.<sup>98</sup>

91. PRESIDENT'S COUNCIL ON COMPETITIVENESS, AGENDA FOR CIVIL JUSTICE REFORM IN AMERICA 11, 21-22 (1991), *reprinted in* Dan Quayle, *Agenda for Civil Justice Reform in America*, 60 U. CIN. L. REV. 979 (1992). The Vice President's tort reform initiative relied heavily upon Huber's analysis of the failings of the tort regime. *See* Chesebro, *supra* note 90, at 1645.

92. See Dan Quayle, Protecting America's Greatness, WASH. POST, Dec. 8, 1991, at C7; see also GREEN, supra note 33, at 19-22 (detailing the "contemporary critique" of the tort system).

93. 509 U.S. 579 (1993).

94. See Sanders, supra note 41, at 1389 (suggesting that the "emerging belief that an increase in 'junk science' in the courtroom requires greater judicial vigilance in admitting expert opinion" fueled a sense of emergency that persuaded the Supreme Court to examine the role of expert testimony in toxic tort cases twenty years after the Federal Rules of Evidence had been adopted).

95. See Beecher-Monas, supra note 37, at 1063.

96. See, e.g., M.G., Bumbling on Billions, AM. LAW., April 1992, at 86; Quayle's Crusade, 17 NAT'L J. 344 (1992); Owen Ullmann, President Quayle, WASHINGTONIAN, September 1992, at 69.

97. See Finley, supra note 7, at 347-64; Michael H. Gottesman, From Barefoot to Daubert to Joiner: Triple Play or Double Error? 40 ARIZ. L. REV. 753, 756-59 (1998).

98. See Frye v. United States, 293 F. 1013 (D.C. Cir. 1923).

Jeff L. Lewin, *Calabresi's Revenge? Junk Science in the Work of Peter Huber*, 21 HOFSTRA L. REV. 183, 186 (1993) (book review).

Reacting to the widespread criticism of the way courts and juries were treating scientific evidence, the Carnegie Commission on Science, Technology and Government conducted a multi-year examination of that topic through a task force that included prominent members of the scientific community as well as the bench and bar. CARNEGIE COMM. ON SCL., TECH. AND GOV'T, SCIENCE AND TECHNOLOGY IN JUDICIAL DECISIONMAKING (1993) [hereinafter CARNEGIE COMMISSION REPORT]. The task force's report, published just before the Supreme Court issued its *Daubert* opinion, took a much less pessimistic view of the ability of courts and juries to cope with the complex scientific issues that arise in toxic tort litigation. It pointed out that "many of the criticisms directed at the operation of our court system arise—quite understandably—from misperceptions about the differing methodologies and goals of science and law, and from the consequent failure to comprehend the diverse roles and expertise of 'judge,' 'juror,' and 'scientist.''' *Id.* at 12. While the Carnegie Commission might have been correct in concluding that the existing court system was not broken, this Article takes the position that the federal judiciary's rather extreme response to the criticism, as signaled by *Daubert/Joiner*, has contributed to a crisis of accountability that calls for institutional change.

Instead, it interpreted Rule 104(a) of the Federal Rules of Evidence as requiring trial judges to be gatekeepers of expert testimony, determining the admissibility of "scientific, technical, or other specialized knowledge" under Rule 702.<sup>99</sup> Before allowing a jury to consider expert testimony, trial judges must determine whether that testimony is relevant and reliable. The reliability of scientific proof is determined by its "scientific validity," which is measured by certain criteria, including testability though scientific methods, presence of absence of peer-review scrutiny, known or potential rates of error, and level of acceptance in the scientific community.<sup>100</sup>

Although the Court's terse and ambiguous guidance might have allowed the law to evolve in either direction,<sup>101</sup> the Court's elaboration on the *Daubert* criteria in *General Electric Co. v. Joiner*<sup>102</sup> clarified the trial judge's role of ensuring that any scientific testimony fit the judge's view of the relevant issues, and this had the foreseeable effect of increasing lower court scrutiny.<sup>103</sup> Dicta in *Joiner* suggest that the trial court is obliged to evaluate the scientific validity of an expert's *conclusions*, as opposed to the expert's methodology. The Court's rejection of the data and methodology employed by the *Joiner* expert also accelerated a trend in the lower courts toward aggressive scrutiny of plaintiffs' expert testimony.<sup>104</sup> Finally, Rule 702 has recently been amended to incorporate the *Daubert/Joiner* tests.<sup>105</sup>

<sup>99. 509</sup> U.S. at 589-93.

<sup>100.</sup> Id. at 590 & n.9, 593-95.

<sup>101.</sup> Some early commentators were convinced that *Daubert* would result in greater opportunities for juries to evaluate scientific evidence in toxic tort cases. See, e.g., Kenneth Chesebro, Taking Daubert's "Focus" Seriously: The Methodology/Conclusion Distinction, 15 CARDOZO L. REV. 1745 (1994); Anthony Z. Roisman, Conflict Resolution in the Courts: The Role of Science, 15 CARDOZO L. REV. 1945 (1994). Many early observers believed, however, that Daubert invited the lower courts to play an even more aggressive role in evaluating the scientific validity of expert testimony in cases involving toxic causation. See, e.g., David E. Bernstein, The Admissibility of Scientific Evidence after Daubert v. Merrell Dow Pharmaceuticals, Inc., 15 CARDOZO L. REV. 2139 (1994); Robert F. Blomquist, The Dangers of "General Observations" on Expert Scientific Testimony: A Comment on Daubert v. Merrell Dow Pharmaceuticals, Inc., 82 KY. L.J. 703, 719 (1993-94). See generally David L. Faigman, The Law's Scientific Revolution: Reflections and Ruminations on the Law's Use of Experts in Year Seven of the Revolution, 57 WASH. & LEE L. REV. 661, 663-64 (2000) (noting that "Daubert contained considerable language to support the view that more, rather than less, expert testimony might be the result of the opinion"). For critiques of the Court's guidance in Daubert and Joiner, see Beecher-Monas, supra note 37, at 1051-62; Sanders, supra note 41, at 1391-92.

<sup>102. 522</sup> U.S. 136 (1997).

<sup>103.</sup> The actual holding of *Joiner* was that the courts of appeals should not review district court applications of *Daubert* excluding scientific testimony with any greater scrutiny than other evidentiary rulings.

<sup>104. 522</sup> U.S. at 146; see also D. Alan Rudlin, *The Judge as Gatekeeper: What Hath Daubert-Joiner-Kumho Wrought?* 29 Prod. Safety & Liab. Rep. (BNA) 329 (2001) (arguing that as a result of *Daubert/Joiner/Kumho* federal trial judges now "play an active role in deciding what expert testimony goes to the jury"). Professor Finley reads *Joiner* to express "a normative judgment that judges are to be trusted more than juries (and sometimes more than scientists) in areas where law intersects with science." Finley, *supra* note 7, at 345.

<sup>105.</sup> FED. R. EVID. 702. The Advisory Committee Notes state that Rule 702 was amended "in response to" *Daubert* and "to the many cases applying *Daubert*."

After more than a decade of experience with Daubert, it is now clear that the lower courts have applied it vigorously to exclude expert testimony.<sup>106</sup> Since the plaintiff ordinarily has the burden of proof in tort litigation, this aggressive invocation of the judge's new role as guardian of the purity of scientific evidence has had a disproportionate impact on plaintiffs. With remarkable speed, judges have gone far beyond throwing the clinical ecologists out of the courtroom.<sup>107</sup> Impressed by artful defense counsels' smoke screens, they are now excluding testimony of well-regarded experts.<sup>108</sup> A plaintiff's attorney must prepare not only to establish an expert's qualifications, but also to convince a skeptical trial judge that the testimony supports a scientifically reliable conclusion based upon scientifically reliable data and that the conclusion fits the legal requirements for establishing cause-in-fact.<sup>109</sup> If the plaintiff's attorney fails, everyone goes home, and no one knows whether the plaintiff was a victim of cruel fate or of the defendant's possibly unconscionable conduct. If the attorney succeeds, the judge and jury must sit through days of confusing and conflicting expert testimony, at the end of which the jury may still decide that the plaintiff did not carry the burden of proof.

#### C. The Raul Proposal to Daubertize Judicial Review of Risk Assessment

In a recent American Bar Association-sponsored symposium on "Science, Agencies, and the Courts," Alan Raul proposed that courts reviewing agency

<sup>106.</sup> Jeffry D. Cutler, Implications of Strict Scrutiny of Scientific Evidence: Does Daubert Deal a Death Blow To Toxic Tort Plaintiffs? 10 J. ENVTL. L. & LITIG. 189, 214 (1995) ("[I]t doesn't take a rocket scientist to figure out that a four or five part test including 'general acceptance' as one factor will be more difficult to meet than a test based on 'general acceptance' alone."); Faigman, supra note 101, at 664 (concluding that "[e]xperience soon showed, however, that Daubert had the effect of excluding a lot of evidence that had been admitted previously"); Finley, supra note 7, at 341-42; Joseph Sanders, The Bendectin Litigation: A Case Study in the Life Cycle of Mass Torts, 43 HASTINGS L.J. 301, 391 (1992). For a general critique of clinical ecology and multiple chemical sensitivity, see Carl H. Johnson, When Science is Too Daunting: Multiple Chemical Sensitivity, Federal Courts, and the Struggling Spirit of Daubert, 11 VILL. ENVTL. L.J. 273, 322-30 (2000).

<sup>107.</sup> In the mid-1980s, the science of clinical ecology appeared to be the answer to the causation conundrum for plaintiffs' attorneys. Professor Elliott described the phenomenon as follows:

For a price, some clinical ecologists will testify that exposure to even very small amounts of a wide range of chemicals suppresses the immune system, thereby weakening the body's ability to ward off disease. This weakening, in turn, allegedly makes the plaintiff vulnerable to virtually all diseases known to humankind, including "nervousness," "malaise," and other conditions that present only subjective symptoms.

E. Donald Elliott, *Toward Incentive-Based Procedure: Three Approaches for Regulating Scientific Evidence*, 69 B.U. L. REV. 487, 490 (1989); *see also* Peter Huber, *A Comment on* Toward Incentive-Based Procedure: Three Approaches for Regulating Scientific Evidence by *E. Donald Elliott*, 69 B.U. L. REV. 513, 515 (1989). Even before *Daubert* refined their screening function, the courts never seriously entertained claims based heavily upon the testimony of clinical ecologists. Johnson, *supra* note 106, at 322-24.

<sup>108.</sup> For an excellent example, see Professor Beecher-Monas's thorough and devastating critique of the post-*Daubert* Eighth Circuit Court of Appeals' opinion in *Wright v. Willamette Indus., Inc.,* 91 F.3d 1105 (8th Cir. 1996). Erica Beecher-Monas, *The Heuristics of Intellectual Due Process: A Primer for Triers of Science*, 75 N.Y.U. L. REV. 1563, 1637 (2000).

<sup>109.</sup> See Harvey Brown, *Eight Gates For Expert Witnesses*, 36 HOUS. L. REV. 743 (1999) (detailing eight "gates" through which a proponent of expert testimony must navigate in order to demonstrate that the testimony is admissible); *see also* Beecher-Monas, *supra* note 108.

risk assessment actions employ "regulatory Daubert" as a principle of judicial review—"not as a solution, but as a reform to enhance agency decision-making, to refine judicial review, and to promote accountability."<sup>110</sup> He is perhaps the strongest, and certainly the most articulate, advocate of this position. Convinced that legislative and presidential oversight are insufficient to ensure responsible and accountable decision-making in federal regulatory agencies, Raul believes that judges must assume a more active role.<sup>111</sup> Applying *Daubert* principles to judicial review of agency risk assessments would, in Raul's view, "promote the full disclosure of all of the agency's underlying principles, assumptions, and facts and obligate the agency to come completely clean on the foundation for its scientific decision."<sup>112</sup> Raul believes more stringent judicial scrutiny will induce more agency disclosure to the courts and the public. He is confident that "[t]he documentation will be better, the explanations will be better, the defaults, the policy choices, and the uncertainties will all necessarily be disclosed and subjected to greater scrutiny, because the agency will know that it's not going to survive in court if it doesn't come clean on those factors."113

To the contrary, judicial adoption of regulatory Daubert will likely result in unconstrained regulatory policymaking by unaccountable and scientifically illiterate judges and in a much higher incidence of judicial remand of important regulations. Regulatees will devote greater resources to sponsoring diversionary research. When adverse scientific studies are published, regulatees will hire consultants to fill the scientific literature with critical and contrary commentary that these regulatees will later cite to support claims that the adverse studies are "fatally flawed." Regulatees will attempt to pack advisory committees with sympathetic scientists in the hope of slipping qualifying language into advisory committee reports, and they will send industry scientific consultants to advisory committee meetings to campaign for such qualifying language. In the end, lawyers for the regulatees will bring all of this regulatee-generated information to the attention of credulous judges applying the corpuscular approach required by stringent regulatory-Daubert review to discredit each study relied upon by the agency. The end result will be fewer rules to impede the regulated community and fewer protections for the beneficiaries of congressionally mandated programs.

Although Raul is vague on the details, he believes that the regulatory-*Daubert* approach would "not only... encourage less deference and more probing judicial review but also ... establish more consistent standards."<sup>114</sup> Raul assures us that if the courts adopted that approach, "it wouldn't always be a roll of the dice as to which judicial panel you get, or what appellate or district court you're

<sup>110.</sup> E. Donald Elliott, Alan Charles Raul, Richard J. Pierce Jr., Thomas O. McGarity & Wendy E. Wagner, Dialogue, *Science, Agencies, and the Courts: Is Three a Crowd?* 31 Envtl. L. Rep. (Envtl. L. Inst.) 10,125, 10,129 (2001); *see also* Raul & Dwyer, *supra* note 11.

<sup>111.</sup> Elliott et. al, *supra* note 110, at 10,131.

<sup>112.</sup> Id. at 10,130.

<sup>113.</sup> Id.

<sup>114.</sup> Id.

before, as to how intensive the judicial review of agency science will be."<sup>115</sup> This is apparently because even those courts that were previously willing to defer to the agency resolution of complex science-policy issues would now require that agency explanations meet judicial standards of scientific adequacy. The outcome of appeals would not depend upon the roll of the dice because few courts would defer to agency expertise. All courts would be equally hostile to agency policymaking, and fewer rules would go into effect (although Raul is at pains to avoid pointing this out).

Even if this is not the scenario that Raul envisions, his argument that adopting vague *Daubert* principles of relevance and reliability would bring about consistency in the lower courts is not persuasive. Unquestionably, more cases would get resolved against the agency than under the current "arbitrary and capricious" standard of review, but it is by no means clear that good agency decisions would consistently satisfy courts applying Raul's regulatory-*Daubert* principles, while bad agency decisions would be consistently set aside. The *Daubert* principles by no means constrain judicial discretion within discernable boundaries. Indeed, the essence of the trial judge's gatekeeper function under *Daubert* is the exercise of judicial discretion over expert testimony that goes before the jury. The court of appeals' function under a regulatory-*Daubert* regime is not likely to be less discretionary. Yet, providing wide latitude to reviewing judges is hardly a recipe for judicial consistency.

# D. The Post-*Daubert* "Corpuscular" Approach to Conclusions Based on Scientific Studies

In the wake of *Daubert* and *Joiner*, most courts have adopted a "corpuscular" approach to determining the admissibility of expert testimony in toxic tort cases. Under this approach, the party offering expert scientific testimony must establish the relevance and reliability under the *Daubert/Joiner* criteria of each individual study relied upon by the expert as well as the relevance and reliability of the expert's overall conclusions.<sup>116</sup> If the plaintiff fails to establish the scientific reliability of a sufficient number of individual studies, the trial judge will exclude the expert's testimony and, in the absence of other relevant and reliable expert testimony on causation, grant a defendant's motion for summary judgment before the case ever reaches a jury. In practice, this approach places a heavy, particularized burden on the plaintiff and invites defendants to focus upon flaws in the corpuscles of data underlying the testimony rather than upon the scientific reliability of the expert's overall conclusions.

Epidemiological studies—the primary corpuscles of expert testimony in toxic tort cases—are exceedingly difficult to conduct in a world where health

<sup>115.</sup> Id.

<sup>116.</sup> See Beecher-Monas, *supra* note 37, at 1057, 1067 (noting that the courts have frequently read *Daubert* to require them to sequentially evaluate each study underlying an expert's conclusion to determine admissibility).

and mortality records are notoriously bad, where data must frequently be drawn from human recollections, and where it is impossible to control against every possible confounding factor or source of bias. Consequently, the conclusions of individual epidemiological studies cannot be stated with a high degree of certainty. Indeed, the one thing that can be said with a great deal of confidence about epidemiological studies is that they are likely to contain flaws and potential biases. The corpuscular approach invites parties seeking to exclude expert testimony to search every detail of each epidemiological study for possible flaws and to speculate at great length about confounding factors and other sources of bias. Given the practical impossibility of conducting a perfect epidemiological study, the search is nearly always fruitful.

Perhaps the best example of the corpuscular approach in action is the majority opinion in *Joiner* itself.<sup>117</sup> In that case, a plaintiff with well-documented exposure to PCBs offered the testimony of two experts who concluded that PCB exposure probably caused the plaintiff's small-cell lung cancer. In holding that the appropriate standard of review for a trial court's admissibility determinations was abuse of discretion, the court addressed the trial judge's application of the *Daubert* criteria to the plaintiff's expert testimony. According to the Court, the testimony of both experts was based primarily upon on a laboratory animal study and four epidemiological studies, none of which could validly support a reliable scientific conclusion that PCBs were capable of causing lung cancer in humans.

The animal study could not validly support that conclusion because the animals were young (not middle-aged like the plaintiff), the route of administration was different (direct injection of single doses into the stomach as opposed to continuous dermal and inhalation exposure), the doses the animals received were much larger than the plaintiff's exposure, and the mice developed a different form of cancer. Although the Court was willing to entertain the possibility that a trial court could appropriately admit relevant expert testimony based upon valid animal studies that were relevant to a plaintiff's exposure, the studies in this case "were so dissimilar to the facts presented in this litigation that it was not an abuse of discretion for the District Court to have rejected the experts' reliance on them."<sup>118</sup> In other words, the district court was appropriately performing its *Daubert*-assigned gatekeeper role when it concluded, apparently as a matter of sound science, that the plaintiff's experts could not validly rely upon the animal study.

The four epidemiological studies upon which the plaintiff's experts relied were likewise held scientifically invalid for the purpose of demonstrating that PCB exposure could cause lung cancer in humans. The first study, which looked at workers exposed to PCBs in an Italian capacitor factory, could not validly support the plaintiff's experts' conclusion. Although the rate of lung cancer deaths was "higher than might have been expected" in the exposed

<sup>117.</sup> Gen. Elec. Co. v. Joiner, 522 U.S. 136 (1997).

<sup>118.</sup> Id. at 144-45.

cohort, the authors of the study concluded that "there were apparently no grounds for associating lung cancer deaths (although increased above expectations) and exposure in the plant."<sup>119</sup> Since the authors of the study were unwilling to conclude that the data in that study demonstrated a causal relationship between PCB exposure and lung cancer, the plaintiff's experts could not validly rely upon that study in any way to support a conclusion that there was such a causal relationship, no matter what additional supporting scientific information might be available.

The second study, which examined workers exposed to PCBs at the Monsanto Corporation's Sauget, Illinois, PCB-manufacturing facility, also found the incidence of lung cancer among those workers to be "somewhat higher" than would ordinarily be expected. However, its authors concluded that the increase was not statistically significant; in their judgment the data did not suggest a link between PCB exposure and lung cancer.<sup>120</sup> Neither the trial court nor the Supreme Court elaborated on the degree of statistical significance that would be required to support a valid conclusion that an elevated incidence of lung cancer in PCB-exposed workers was linked to the exposure. They were apparently content to rely upon the judgment of the Monsanto scientists who conducted the study. Therefore, even if they disagreed with the Monsanto scientists as to the level of statistical significance required to support a cause-effect conclusion, the plaintiff's experts could not validly rely upon that study to support an overall conclusion that PCBs cause lung cancer in humans.

Although a third study of Norwegian employees of a cable-manufacturing plant did report an increase in lung cancer that was statistically significant in the authors' view, the plaintiff's experts could not rely upon it either, because it made no mention of PCBs and was limited to the particular mineral oil to which the workers were exposed.<sup>121</sup> The court did not address the fact relied upon by the plaintiff's experts that many kinds of mineral oil contain PCBs. The plaintiff's experts apparently had reason to believe that the mineral oil to which the Norwegian employees were exposed contained PCBs, but because the published study made no mention of PCBs, the Court viewed the study as irrelevant.

The fourth study appeared at first glance to be the Holy Grail for which the Court was apparently searching. The study detected a statistically significant increase in lung cancer deaths in a PCB-exposed human cohort in Japan. But even that study had to be rejected because the "subjects of this study... had been exposed to numerous potential carcinogens, including toxic rice oil that

<sup>119.</sup> Id. at 145 (quoting Alberto Bertazzi, et al., *Cancer Mortality of Capacitor Manufacturing Workers*, 11 AM. J. INDUS. MED. 165, 172 (1987)). Neither the trial court nor the Supreme Court inquired into what might have motivated the authors to conclude that the PCB exposures were not associated with the lung cancers.

<sup>120.</sup> *Id.* Again, neither the trial court nor the Supreme Court inquired into what might have motivated the authors (who probably undertook the study at the behest of the Monsanto Corporation) to conclude that the increase in lung cancer was not statistically significant.

<sup>121.</sup> Id. at 145-46.

they had ingested."<sup>122</sup> Without relying upon any expert statistical analysis, the trial judge concluded, apparently as a scientific matter, that this confounding factor (which is likely to be present in any Japanese cohort) had not been adequately addressed. The study was therefore also invalid and could not support an overall conclusion that PCBs cause lung cancer in humans.

As the *Joiner* case reveals, the corpuscular approach effectively prevents experts in toxic tort cases from applying the cumulative weight-of-the-evidence approach universally employed by regulatory agencies in assessing the human risk posed by toxic substances.<sup>123</sup> The weight-of-the-evidence approach focuses upon the totality of the scientific information and asks whether a cause-effect conclusion is warranted. Given that flaws are inevitable in individual studies and that most studies are not undertaken in anticipation of litigation or the regulatory process, the weight-of-the-evidence approach necessarily involves scientific judgments grounded in scientific expertise; and regulatory agencies are supposed to be repositories of scientific expertise. The corpuscular approach to judicial review focuses upon the inevitable flaws in individual studies and asks whether a sufficient number of relevant studies with sufficiently few flaws remain to support a conclusion that is itself relevant and reliable. Under this approach, a study is either valid or invalid and thus either relevant or irrelevant to the issue the agency must resolve. Both determinations are made by judges who lack scientific expertise.

Courts' *Daubert/Joiner*, corpuscular approach to causation is not only different, but arguably much less scientific,<sup>124</sup> than that of regulatory agencies charged with protecting citizens from toxic risks. A *Daubert* approach to judicial review of agency risk assessments predictably would be similar. Ultimately, agencies would have to defend their regulations in the corpuscular format adopted in *Joiner*. Rather than describe the studies that support a particular conclusion regarding the risk of human exposure to a toxic substance and assess those risks under a weight-of-the-evidence approach, an agency would have to establish the scientific validity of each relevant study, demonstrate its relevance with scientifically valid reasoning, and convince skeptical judges that the overall risk assessment is not arbitrary and capricious.

<sup>122.</sup> Id. at 146.

<sup>123.</sup> See Brown, supra note 109, at 845 (arguing that, although not specifically addressed in Joiner, the Court implicitly rejected the weight-of-the-evidence approach); Gottesman, supra note 97, at 769-70; see also Beecher-Monas, supra note 37, at 1067 ("Although the relevance of a single study may be a fairly straightforward determination, relevance becomes more complicated when a number of studies are involved, each of which is only marginally relevant, but which together purport to form the basis of an expert's conclusions."); Finley, supra note 7, at 336 ("Judges have applied Daubert to subject each item of expert proof proffered by plaintiffs to substantive causation law scrutiny, to see if it, standing alone, would prove both general and specific causation."); Sanders, supra note 41, at 1416-17 (criticizing courts that do not preclude an expert from relying on all of the available evidence). At least one post-Daubert court has explicitly rejected an expert's conclusions based on a weight-of-the-evidence approach. See Allen v. Pa. Eng'g Corp., 102 F.3d 194, 196-98 (5th Cir. 1996).

<sup>124.</sup> See supra note 123.

#### E. The "Hard-Look" Doctrine and the Corpuscular Approach

When federal agencies began exercising their rulemaking powers for the first time in the early 1970s, the U.S. Court of Appeals for the District of Columbia adopted the "hard look" approach to judicial review of informal rulemaking.<sup>125</sup> Under this approach, the courts take a hard look at the administrative record and the agency's explanatory material to determine whether the agency used appropriate analytical methodologies, applied the proper criteria, considered the relevant factors, chose from among the available regulatory options, relied upon allowable policies, and pointed to adequate support in the record for material, empirical propositions. The hard-look approach rapidly attracted the courts of appeals<sup>126</sup> and the legal academy.<sup>127</sup> And, arguably, the Supreme Court adopted a form of hard-look review in *Motor Vehicles Manufacturers Association v. State Farm Mutual Automobile Insurance Co*,<sup>128</sup> in which it stated that a regulation is arbitrary and capricious if

the agency has relied on factors which Congress has not intended it to consider, entirely failed to consider an important aspect of the problem, offered an explanation for its decision that runs counter to the evidence before the agency, or is so implausible that it could not be ascribed to a difference in view or the product of agency expertise.<sup>129</sup>

The first two elements—whether the agency relied on the wrong factors or failed to consider an important aspect of the problem—might be labeled *prescriptive* substantive judicial review.<sup>130</sup> Prescriptive review oversees the manner in which an agency implements its statutory goals as it resolves particular substantive policy issues. It includes the analytical methodologies the agency adopts to resolve certain kinds of issues, the criteria it employs, the factors it considers, the range of options from which it chooses, and the policies it relies upon when existing scientific data do not lead to firm factual conclusions.<sup>131</sup>

<sup>125.</sup> See JOHN E. BONINE & THOMAS O. MCGARITY, THE LAW OF ENVIRONMENTAL PROTECTION 142-45 (2d ed. 1991) (describing the evolution of the hard-look doctrine). Judge Levanthal originally coined the term "hard look" to characterize the scrutiny agencies are expected to give to the contentions of the parties under the Administrative Procedure Act ("APA"), Pikes Peak Broad. Co. v. FCC, 422 F.2d 671 (D.C. Cir. 1969), and to the environmental effects of the parties' activities under the National Environmental Policy Act, Natural Res. Def. Council, Inc. v. Morton, 458 F.2d 827 (D.C. Cir. 1972). Subsequently, the court invoked the hard-look metaphor to characterize the related judicial function of substantive judicial review of agency rulemaking under the APA's "arbitrary and capricious" test. BONINE & MCGARITY, *supra*, at 71; *see* Cabinet Mountains Wilderness/Scotchman's Peak Grizzly Bears v. Peterson, 685 F.2d 678, 685 (D.C. Cir. 1982); Md.-Nat'l Capital Park & Planning Comm'n v. U.S. Postal Serv., 487 F.2d 1029, 1039-40 (D.C. Cir. 1973).

<sup>126.</sup> See supra note 125.

<sup>127.</sup> Id.

<sup>128. 463</sup> U.S. 29, 43 (1983).

<sup>129.</sup> Id. at 42.

<sup>130.</sup> See Thomas O. McGarity, Beyond the Hard Look: A New Standard for Judicial Review?, 2 NAT. RESOURCES & ENV'T. 32 (1986).

<sup>131.</sup> On science-policy questions generally, see NAT'L RESEARCH COUNCIL, NAT'L ACAD. OF SCI., RISK ASSESSMENT IN THE FEDERAL GOVERNMENT: MANAGING THE PROCESS 3 (1983); John S. Applegate, *The Perils of Unreasonable Risk: Information, Regulatory Policy, and Toxic Substances Control*, 91 COLUM. L. REV. 261 (1991); Donald T. Hornstein, *Reclaiming Environmental Law: A Normative Critique of Comparative Risk Analysis*, 92 COLUM. L. REV. 562 (1992); McGarity, *supra* 

The second two elements of the *State Farm* test—whether the agency's explanation runs counter to the evidence or is too implausible—might be called *evaluative* substantive judicial review.<sup>132</sup> This aspect of the *State Farm* test presumes that an agency's "concise general statement of basis and purpose"<sup>133</sup> contains explanations for how it resolved important scientific, economic, and technical questions that arose in the process of promulgating a rule (usually on the basis of assumptions supplied by the agency).<sup>134</sup> It also presumes that the agency responded to material comments by explaining why it accepted or rejected them.<sup>135</sup> The courts' role is to determine whether the agency's technical explanation for how it resolved an important issue, or its response to a critical outside comment, is inconsistent with the evidence in the rulemaking record or is implausible under some appropriate (and judicially ascertainable) measure of plausibility.

The Raul proposal for regulatory-*Daubert* review would intensify both of these evaluative and prescriptive functions of judicial review of agency action. Presumably, the *Daubert* relevance inquiry would be incorporated into the prescriptive aspect of hard-look review; courts would even more carefully examine whether the studies an agency relied upon are relevant to the factors Congress deemed applicable under the particular statutory scheme and might even more carefully examine the appropriateness of agency resolution of science-policy questions.

*Daubert*'s reliability inquiry could easily become incorporated into the courts' evaluative review. Courts would skeptically examine an agency's "concise general statement" (and perhaps other supporting documents) to determine whether the agency relied upon a sufficient number of scientifically reliable studies to support the conclusions in its risk assessment. If not, the agency's explanation presumably would be considered inconsistent with the evidence or otherwise implausible. Again, the inquiry would be more intense than evaluative review has previously been. It would not matter how many studies deemed scientifically invalid by the court purported to observe "fire"; an agency's risk assessment would have to be set aside if the agency had not relied upon a sufficient number of scientifically valid studies in which a cause-effect relationship was observed.

note 58; Wendy E. Wagner, *The Science Charade in Toxic Risk Regulation*, 95 COLUM. L. REV. 1613 (1995).

<sup>132.</sup> McGarity, *supra* note 130.

<sup>133.</sup> The Administrative Procedure Act requires that all final rules produced through informal rulemaking procedures be accompanied by a "concise general statement of [the] basis and purpose" of the rule. *See* 42 U.S.C. § 553(c) (2000).

<sup>134.</sup> See Int'l Harvester Corp. v. Ruckelshaus, 478 F.2d 615 (D.C. Cir. 1973); Kennecott Copper Corp. v. EPA, 462 F.2d 846 (D.C. Cir. 1972).

<sup>135.</sup> See Portland Cement Ass'n v. Ruckelshaus, 486 F.2d 375 (D.C. Cir. 1973).

The recent opinion of the D.C. Circuit in *American Trucking Ass'ns v. EPA*<sup>136</sup> provides a good example of how the current hard-look doctrine differs from the proposed regulatory-*Daubert* approach. In *American Trucking*, various industry groups challenged EPA's newly revised "national ambient air quality standards" ("NAAQS") for ozone and fine particulates. The case eventually reached the Supreme Court. After the Court determined that section 109 of the Clean Air Act<sup>137</sup> did not require or permit EPA to engage in cost-benefit balancing in setting the NAAQS and that, so interpreted, section 109 was not unconstitutional, it remanded the case for resolution of the remaining claims;<sup>138</sup> most of which involved hard-look challenges to the scientific groundings of the agency's new standards. The D.C. Circuit upheld these standards against all pending challenges.

In their comments on the ozone standard, the petitioners pressed the corpuscular approach with particular force, challenging EPA's reliance on "specific field, epidemiological, and clinical studies" they deemed invalid.<sup>139</sup> In EPA's view, these comments "did not reflect an integrative assessment of the evidence—the approach CASAC [the Clean Air Scientific Advisory Committee] has historically urged [the agency] to follow—but rather a piecemeal look at each individual study."<sup>140</sup> The court noted that, for this reason, EPA "dismissed the comments, arguing that such an incremental critique 'tends to miss the strength of the entire body of evidence taken together."<sup>141</sup> In upholding EPA's interpretation of the totality of the evidence on the effects of ground-level ozone, the court allowed EPA to rely upon the weight of the evidence. For example, because the record was "replete with references to studies demonstrating the inadequacies of the old one-hour standard,"<sup>142</sup> the agency was entitled to rely upon the CASAC's consensus that, based upon the weight of the evidence, an eight-hour standard was more appropriate for protecting human health than the old one-hour standard.

#### III

## FLUE-CURED TOBACCO AS A SIGN OF THINGS TO COME

In *Flue-Cured Tobacco Cooperative Stabilization Corp. v. EPA*,<sup>143</sup> the U.S. District Court for the Middle District of North Carolina upheld a tobaccoindustry challenge to EPA's risk assessment for environmental tobacco smoke ("ETS"). The court of appeals concluded that the risk assessment was not ripe

<sup>136. 283</sup> F.3d 355, (D.C. Cir. 2002).

<sup>137. 42</sup> U.S.C. § 7409 (2000).

<sup>138.</sup> Whitman v. Am. Trucking Ass'ns, 531 U.S. 457 (2001).

<sup>139. 283</sup> F.3d at 377.

<sup>140.</sup> Id. (quoting Ozone NAAQS, 62 Fed. Reg. 38,856, 38,866 (July 18, 1997)).

<sup>141.</sup> Id. (citing Ozone NAAQS, 62 Fed. Reg. at 38,866).

<sup>142.</sup> Id. at 378.

<sup>143. 4</sup> F. Supp. 2d 435 (M.D.N.C. 1998), vacated and remanded by 313 F.3d 852 (4th Cir. 2002).

for judicial review and vacated the district court's judgment.<sup>144</sup> But the skepticism the lower court demonstrated toward EPA's scientific assessments, the credulity with which it accepted the assertions of the tobacco industry's scientific consultants, and the corpuscular approach it took respecting studies underlying the risk assessment make *Flue-Cured Tobacco* an example of things to come if the courts adopt a regulatory-*Daubert* approach to judicial review of agency risk assessment. *Flue-Cured Tobacco* is also an ideal case study because of the availability of an extraordinary database of thirty-three million internal tobacco-industry documents resulting from the tobacco litigation that now allows scholars to observe first-hand an industry's attempt to bend science to its litigative and regulatory needs. The following case analysis draws upon this database to explore the industry's decade-long efforts to cast doubt upon EPA's ETS risk assessment initiative.

#### A. The Industry Trashes Hirayama

In January 1981, the *British Medical Journal* published recently compiled and analyzed data from an ongoing prospective cohort study of the relationship between smoking and health conducted by Japanese epidemiologist Takeshi Hirayama. The study tracked the incidence of lung cancer in more than 90,000 nonsmoking wives of Japanese smokers between 1966 and 1979.<sup>145</sup> Hirayama concluded that a smoking husband increased his wife's risk of contracting lung cancer to between one-third and one-half that of a smoker.<sup>146</sup> Hirayama detected no association, however, between spousal smoking and the wife's risk of dying from other diseases like "stomach cancer, cervical cancer, and ischaemic heart disease."<sup>147</sup> To Hirayama, the results indicated "the possible importance of passive or indirect smoking as one of the causal factors of lung cancer."<sup>148</sup>

Shortly after the publication of Hirayama's study, the *International Journal* of *Cancer* published a much smaller case-control study by a team headed by Dimitrios Trichopoulos of the University of Athens School of Medicine.<sup>149</sup> Trichopoulos and his colleagues interviewed fifty-one women with lung cancer

<sup>144.</sup> Flue-Cured Tobacco Coop. Stabilization Corp. v. EPA, 313 F.3d 852 (4th Cir. 2002).

<sup>145.</sup> Takeshi Hirayama, Non-Smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer: A Study from Japan, 282 BRIT. MED. J. 183 (1981). Prior to the Hirayama and Trichopoulos studies, only one of several epidemiological examinations of adverse health effects of smoking in non-smokers had found a statistically significant correlation. In 1979, Drs. White and Froeb published a study in the New England Journal of Medicine concluding that exposure to tobacco smoke in the work-place over a period of twenty years or more caused a dysfunction of the small airways in nonsmokers' lungs, as indicated by measuring forced exhalation volume. James R. White & Herman F. Froeb, Small-Airways Dysfunction in Nonsmokers Chronically Exposed to Tobacco Smoke, 302 NEW ENG. J. MED. 720 (1980). While that study concerned the tobacco industry enough for it to pay for numerous scientific critiques, it did not pose nearly the threat that Dr. Hirayama's study did.

<sup>146.</sup> Id. at 183. The risk was "particularly great in agricultural families when the husbands were aged 40-59" at the beginning of the study. Id.

<sup>147.</sup> Id.

<sup>148.</sup> Id.

<sup>149.</sup> Dimitrios Trichopoulos et al., Lung Cancer and Passive Smoking, 27 INT'L J. CANCER 1 (1981).

and 163 other hospital patients regarding the smoking habits of their husbands. The study concluded that among the nonsmoking women there was a "statistically significant difference between the cancer cases and the other patients with respect to their husbands' smoking habits."<sup>150</sup> The relative risk for nonsmokers with husbands who smoked less than one pack a day was 2.4 compared to a 3.4 relative risk for nonsmoking wives of men who smoked more than one pack per day.<sup>151</sup>

A memorandum prepared for the Tobacco Institute Executive Committee meeting of May 19, 1981, stated the relevance of the two studies to the tobacco industry in stark terms: "The Hirayama study and the Greek study which conclude that smoking causes cancer in nonsmokers may be the most damaging scientific articles in the history of our industry."<sup>152</sup> If smoking posed a risk to nonsmokers, reasonable accommodation was no longer a credible answer to complaints about public smoking, and the tobacco industry was in serious trouble.

1. Initial Response to the Hirayama and Trichopoulos Studies

The industry became aware of both studies several months in advance of publication. On August 13, 1980, an R.J. Reynolds ("RJR") scientist responded to Hirayama's presentation of his results at a scientific gathering in Helsinki:<sup>153</sup> although Hirayama was "the best known of Japan's cancer epidemiologists," the data were "generally considered not to be of high caliber" and could "probably be 'explained' because they were not corrected for 'confounding variables.""154

The industry began drafting plans in advance for responding to the study once published. Warren Donahue, a Philip Morris public relations specialist, warned that the company's response "must not be angry counterattack, but calm, reasoned, and confident statements."155 Soon after the Hirayama and Trichopoulos studies appeared in print, the tobacco industry launched a major initiative to undermine the scientific bases for their conclusions. The Tobacco Institute's Scientific Affairs Division prepared a critical assessment of the Hira-

<sup>150.</sup> Id. The authors noted that a study of Greek women was especially appropriate because for more than twenty years Greek men had smoked heavily while Greek women rarely smoked. Id. at 3. 151. Id.

<sup>152.</sup> J.K.W., Outline for TI Meeting on May 19, 1981 (undated) (Bates No. 680543057). The Bates numbers referenced throughout this Article are unique numbers that are consistent among the various online and print collections of the tobacco-industry documents. These documents are contained in the Legacy Tobacco Documents Library, maintained by the University of California, and all industry documents referenced in this Article are available at that website. See Legacy Tobacco Documents Library, at http://legacy.library.ucsf.edu/ (last visited July 1, 2003).

<sup>153.</sup> Memorandum from Frank G. Colby to Dr. Alan Rodgman (Aug. 13, 1980) (Bates No. 19800813). The industry also had early notification of the Trichopoulos study. Trichopoulos submitted his study for publication on October 15, 1980, and by December the tobacco industry had obtained a copy of the study's galley proofs. See Galley Proofs, Dimitrios Trichopoulos et al., Lung Cancer and Passive Smoking (Dec. 12, 1980) (Bates No. 521027372).

<sup>154.</sup> Memorandum from Frank G. Colby to Dr. Alan Rodgman, *supra* note 153.

<sup>155.</sup> Memorandum from Warren Donahue to Frank Saunders 2 (undated) (Bates No. 2021278875).

yama study highlighting several weaknesses.<sup>156</sup> The mortality statistics, assembled from death certificates (as was the case with most epidemiological studies of that time), were "known to contain many diagnostic inaccuracies."<sup>157</sup> The study did not attempt to determine the length of exposure to tobacco smoke through a measure such as "person years at risk",<sup>158</sup> in other words, the study did not attempt to calculate the "degree of exposure these women had to cigarette smoke."<sup>159</sup> Finally, the Hirayama study did not account for confounding factors like the use of charcoal hibachis for cooking and heating in Japan, the tendency for rural families to spend more time outdoors, the "inadvertent characteristic of Japanese homes (especially in rural areas) to be well-ventilated," or "the fact that a heavy smoker may do most of his smoking on the job or in the local pub rather than at home."<sup>160</sup>

A lengthy internal Philip Morris critique noted "the comparatively large number of *unmarried women with lung cancer*."<sup>161</sup> If the same proportion of unmarried Japanese women smoked as Japanese women in general, the association Hirayama purported to find between lung cancer and passive smoking would "probably be reversed dramatically."<sup>162</sup> Furthermore, it was not clear that Hirayama had corrected for "social class and/or income."<sup>163</sup> The memo also complained that the original data were collected by census takers who were not trained in epidemiology.<sup>164</sup> The goal, the memo concluded, was to obtain and reanalyze Hirayama's raw data. In so doing, the industry might inquire of fellow scientists at the Japan Tobacco and Salt Monopoly whether the data "could be subpoena[e]d and/or whether the Japan and U.S. and/or other Industry organizations could work together 'unraveling' Hirayama's public smoking allegations."<sup>165</sup>

The industry criticisms were potentially valid, but similar concerns could be raised about virtually any epidemiological study. There is always another piece of information a study instrument could have demanded and another potential confounding factor for which a study could have controlled. In many cases, however, the needed information is exceedingly difficult to obtain. Should Hirayama have validated the death certificates he relied upon though some

<sup>156.</sup> Memorandum from Scientific Affairs Division to Horace R. Kornegay, Recent Hirayama Paper (Jan. 26, 1981) (Bates No. 2024955077). The Institute worked the criticisms detailed in the memo into a brief "backgrounder" that it sent out to answer initial press inquiries. Telex from Robert H. Bockman to W. Campbell (Jan. 28, 1981) (Bates No. 2024182984).

<sup>157.</sup> Memorandum from Scientific Affairs Division to Horace R. Kornegay, supra note 156, at 2.

<sup>158.</sup> Id. at 3.

<sup>159.</sup> Id.

<sup>160.</sup> Id. at 4.

<sup>161.</sup> Memorandum, Non-Smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer: A Study from Japan: A Critique 1 (Jan. 27, 1981) (Bates No. 1002647454).

<sup>162.</sup> *Id.* The memo also noted that the conclusion that there was a higher incidence of lung cancer among women in rural versus non-rural areas seemed "contrary to other alleged epidemiological findings." *Id.* at 2.

<sup>163.</sup> Id. at 2.

<sup>164.</sup> Id.

<sup>165.</sup> Id. at 4.

verification tool? Should the instrument have attempted to ascertain how long a wife's husband had been smoking, how much time the husband spent at home, and how frequently the husband smoked when at home? Should the interviewer have asked the wife how frequently she cooked on an hibachi, how drafty her house was, and how much time her husband spent at the pub? That the answer to some, or even most, of these questions might be "yes" does not necessarily undermine Dr. Hirayama's conclusions. Questions that might render an epidemiological study invalid under the corpuscular approach do not preclude its consideration under a weight-of-the-evidence approach, though the number and nature of outstanding questions might suggest how much weight the study should receive.

One of the tobacco-industry consultants who commented on the paper within days of its publication was Peter N. Lee—a statistician retained by the British tobacco industry's trade association, the Tobacco Advisory Council ("TAC"), to prepare unsolicited analyses of relevant scientific studies as they appeared in the literature.<sup>166</sup> Lee noted that "[t]he importance of Hirayama's paper... [was] obvious[,] especially as it [came] from an epidemiologist of repute and [was] based on a very large data base of over 265,000 adults followed for 14 years."<sup>167</sup> Lee observed, however, that the analyses were standardized for the age of the husband but not the wife. If the nonsmoking wives of smokers tended to be older than nonsmoking wives of nonsmokers in Japan, age alone might have accounted for the increased lung cancer in the first group. Yet if this were the case, the effect should have shown up for all age-related diseases and not just lung cancer.<sup>168</sup> The bottom line was that "Hirayama's estimate of the significance of [the] risk ratio using the Mantel-extension [Chi] test appear[ed] correct."<sup>169</sup> Despite its shortcomings, it was "difficult to argue that the study was fundamentally flawed."<sup>170</sup>

Nevertheless, Lee found Hirayama's conclusion that passive smoking caused lung cancer "hard to accept [considering the] relative dose of smoke constituents taken in by active and passive smokers."<sup>171</sup> Even if the data supported the conclusion, Lee questioned its applicability to the Western world, with its markedly different mortality patterns.<sup>172</sup> Finally, Lee noted that Lawrence Garfinkel, an epidemiologist working on the American Cancer Society's ongoing large-scale epidemiological study, had told Lee that his data showed no adverse

<sup>166.</sup> P.N. Lee, Paper by Dr. T. Hirayama in the British Medical Journal, 17th January 1981 "Non-Smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer: A Study from Japan": First Comments (Jan. 20, 1981) (Bates No. 521024694).

<sup>167.</sup> Id. at 1.

<sup>168.</sup> *Id.* at 2.

<sup>169.</sup> *Id.* Lee disagreed, however, with application of the Mantel test to support the conclusion that "the risk associated with passive smoking is particularly marked in wives of young agricultural workers." *Id.* 

<sup>170.</sup> Id. at 4.

<sup>171.</sup> Lee, supra note 166, at 3.

<sup>172.</sup> Id.

effect from passive smoking.<sup>173</sup> Lee speculated that "Garfinkel's findings, based on a larger study[] and taking into account a wide range of co-factors[,] may turn out to be of greater scientific value."<sup>174</sup>

Although Lee was on the payroll of the British tobacco industry, his analysis was surprisingly balanced. He suggested that the Hirayama study would not fare well when compared to the larger Garfinkel study, which by its nature would be more relevant to the American and British populations, but he also found many reasons to give the study at least modest weight. While cautioning that Hirayama's study should not be taken authoritatively without additional evidence, he nevertheless refused to conclude that it was fundamentally flawed from a statistical perspective. A carefully nuanced critique was not, however, what the U.S. tobacco industry wanted to hear.

On January 27, 1981, Lee reported to the Research Committee of the British Tobacco Advisory Council that neither Dr. Garfinkel nor Dr. Nicholas Wald, a well-regarded Oxford epidemiologist, had any "solid evidence on which to refute [Hirayama's] conclusions."<sup>175</sup> The committee members noted that "in addition to any criticisms of a purely scientific nature, it was necessary to take account of the marked differences in the social life of the Japanese compared with countries in the West."<sup>176</sup> In addition, they cautioned Lee that "from a public relations point of view, [it was important] to widen the issues by taking account of other factors likely to be involved in the different patterns of cancer in the different communities."<sup>177</sup>

The U.S. tobacco industry also sought Lee's advice. An analysis of data prepared by Frank C. Colby, an RJR statistician, with the help of Dr. Marvin Kastenbaum,<sup>178</sup> chief statistician for the U.S. Tobacco Institute ("TI"), concluded that if unmarried nonsmoking Japanese women were included in the

<sup>173.</sup> Id.

<sup>174.</sup> *Id.* Five days later, in a paper entitled "Some Further Comments," Lee noted that the overall increase in lung cancer among all women in Japan indicated that other factors (perhaps air pollution) "must be of substantial importance in Japanese lung cancer aetiology... In any event the large contribution of nonsmoking factors would appear to add to the reason for not taking Hirayama's findings as gospel without other evidence." P.N. Lee, Paper by Dr. T. Hirayama: Some Further Comments (Jan. 25, 1981) (Bates No. 504886135).

<sup>175.</sup> Minutes of the 11th Meeting of the Research Committee of T.A.C. Held at 11:00 a.m. on Tuesday, 27th January 1981 at Glen House, Stag Place, London SWIE 5AG, at 4 (Jan. 27, 1981) (Bates No. 2025017011).

<sup>176.</sup> Id.

<sup>177.</sup> Id. On February 10, 1981, Lee prepared another analysis in which he concluded that "if the passive smoking really does lead to a doubling in the risk of lung cancer, then it is equivalent to active smoking of at least three cigarettes a day in the Japanese situation." P.N. Lee, Relative Effects of Active and Passive Smoking: Some Mathematical Considerations (Feb. 10, 1981) (Bates No. 504867953). To Lee, this seemed "very high... when viewed against evidence on smoke uptake in extreme passive smoking situations." Id. This led Lee to the "strong suspicion that some biasing factor is involved, though what this could be is not at all clear." Lee speculated that the search for this biasing factor "may lead to something which might explain part of the smoking/lung cancer relationship itself. An intriguing thought!" Id. An intriguing thought, perhaps, but not one that the tobacco industry was anxious to pursue.

<sup>178.</sup> See Memorandum from Marvin Kastenbaum to Frank Colby (Feb. 20, 1981) (Bates No. 504886141).

category of nonsmoking women who were married to nonsmokers, the difference in lung cancer rates disappeared.<sup>179</sup> Asked to comment on Colby's memo, Lee observed that adding unmarried women to the class of women married to nonsmokers would be inappropriate unless the data were carefully adjusted for age because men die faster than women.<sup>180</sup> Furthermore, Dr. Lee disagreed with Colby's calculations, and he provided detailed calculations of his own.<sup>181</sup> Apparently, Dr. Lee's rebuttal was sufficient to dissuade Dr. Colby from pursuing the matter further. Dr. Kastenbaum, however, was not so easily discouraged.

The TI prepared a quick response to the Trichopoulos study.<sup>182</sup> Noting the small number of subjects included in the study, TI scientists found it impossible to draw valid inferences without better information about the sample populations.<sup>183</sup> Since at least eighteen of the fifty-one lung cancers had been clinically diagnosed without laboratory verification, it was impossible to tell whether they were primary lung cancers or had metastasized from some other part of the body.<sup>184</sup> In addition, Trichopoulos failed to control for all possible confounding factors, including "genetic predisposition and family history of disease, occupational exposures, socio-economic status and stress."<sup>185</sup> Finally, the study failed to determine the actual number of cigarettes smoked in the home and ignored the possibility that husbands might have limited their smoking to places primarily outside the home such as "the local taverna."<sup>186</sup>

2. Confronting Hirayama Directly

On March 2, 1981, Kastenbaum wrote Dr. Hirayama directly, requesting answers to sixteen specific questions.<sup>187</sup> Some of the demands were altogether reasonable. For example, Kastenbaum asked for a copy of the Hirayama's questionnaire, further amplification on some definitional issues, and clarification as to whether certain categories in his tables included nonmarried women.<sup>188</sup> Other requests would have required Hirayama to expend a great deal of effort. For example, Kastenbaum asked for fifteen years' worth of information concerning who gathered the data, the type of training they received, and

188. *Id.* at 1-3.

<sup>179.</sup> Memorandum from Frank C. Colby to Max H. Crohn, Jr. 2 (Feb. 3, 1981) (Bates No. 506641713). A Philip Morris statistician reached the same conclusion. *See supra* text accompanying note 161.

<sup>180.</sup> P.N. Lee, Hirayama Passive Smoking Study: Some Comments on the Critique by Colby 1 (Feb. 19, 1981) (Bates No. 504886335).

<sup>181.</sup> *Id.* at 1-5.

<sup>182.</sup> Tobacco Institute, Background for Response to Trichopoulos Paper (Feb. 6, 1981) (Bates No. 1002623175).

<sup>183.</sup> *Id.* at 1.

<sup>184.</sup> Id. at 2.

<sup>185.</sup> *Id*.

<sup>186.</sup> *Id.* Dr. Colby also examined the Trichopoulos study and concluded that it was "based on even flimsier data than the Hirayama study." Memorandum from Frank C. Colby to Max H. Crohn, *supra* note 179, at 5.

<sup>187.</sup> Letter from Marvin A. Kastenbaum to Takeshi Hirayama (Mar. 2, 1981) (Bates No. 503947528).

the location of all demographic histories.<sup>189</sup> Additionally, Kastenbaum requested a copy of all of Hirayama's raw data, preferably in computer-friendly form.<sup>190</sup> Many of the questions were plainly argumentative in nature and were not asked with any realistic expectation of response.<sup>191</sup>

The German Verband, Germany's equivalent of the Tobacco Institute, arranged for Peter Lee and Dr. Franz Adlkofer, a German tobacco-industry scientist, to meet with Hirayama during the last week of April, 1981.<sup>192</sup> Adlkofer and Lee first met for an hour with Hirayama's boss, Dr. Sugimura.<sup>193</sup> Dr. Sugimura was "remarkably frank" about Hirayama's work on ETS, suggesting that Hirayama was "over ready to jump into publication without adequate scientific discussion first."<sup>194</sup> He speculated that the dose Japanese women would have received from their husbands' smoke would be very low because Japanese married men were typically at home for only ten hours per day, eight of which they spent sleeping.<sup>195</sup>

Adlkofer and Lee then met for seven hours with Hirayama himself. Hirayama impressed Lee "as being more of an open-minded scientist than might have been expected."<sup>196</sup> He was "very frank and answered all our questions very fully and without any hint that he was at all annoyed by our difficulties in accepting the conclusions of his study."<sup>197</sup> Significantly, Hirayama "did not consider it proved that passive smoking increased the risk of lung cancer."<sup>198</sup> He believed that his paper, together with the Trichopoulos publication, generated "a hypothesis that required further confirmation before it could be accepted."<sup>199</sup> Hirayama welcomed the forthcoming Garfinkel paper and hoped it might remove some of the "media pressure" and "allow further research to be carried out more peacefully."<sup>200</sup> He did not believe, however, that it refuted his own work.<sup>201</sup>

192. Letter from Takeshi Hirayama to Dr. D. Schmahl (Feb. 16, 1981) (Bates No. 1000120121).

- 197. *Id.* at 2.
- 198. *Id.*
- 199. Id.
- 200. Id.
- 201. Id.

<sup>189.</sup> *Id.* at 2. Kastenbaum demanded "a record of deaths by age, sex and other available characteristics" and "the age distribution of [his] subjects by sex and smoking class, as of 1965 and at any other point in time." *Id.* 

<sup>190.</sup> Id. at 1.

<sup>191.</sup> For example, Kastenbaum asked Hirayama how he reconciled his reliance on death certificates with other Japanese studies on the accuracy of such death-certificate based studies, how he assessed the reliability of self-reporting of smoking habits in light of serious questions that had been raised in the scientific literature on self-reporting, *id.* at 1-2, and how he reconciled his 8.7 per 100,000 mortality rate for nonsmoking wives of nonsmoking husbands with the 5.7 per 100,000 overall Japanese mortality as determined by the American Cancer Society, *id.* at 3.

<sup>193.</sup> P.N. Lee, Notes on Visit to Japan: April 24th to May 1st, 1981 (Apr. 5, 1981) (Bates No. 504798208).

<sup>194.</sup> *Id.* at 1-2.

<sup>195.</sup> Id. at 2.

<sup>196.</sup> Id.

None of Hirayama's responses indicated "any major biasing or confounding factors which might explain the association seen between passive smoking and lung cancer."<sup>202</sup> In particular, social class, a factor strongly alluded to by many of the tobacco-industry scientists, "did not alter the picture materially."<sup>203</sup> Similarly, Hirayama adequately controlled for air pollution and population density.<sup>204</sup> Hirayama also had good answers to questions about the frequency of smoking among Japanese husbands and the reliability of self-reported smoking habits.<sup>205</sup> Lee further divined from his conversation with Hirayama that industry scientists had misunderstood Hirayama's data and had erroneously concluded that death rates of unmarried nonsmoking women were considerably higher than those of married, nonsmoking women.<sup>206</sup> Although Lee still believed that Hirayama's conclusion that passive smoking caused lung cancer was "implausible,"<sup>207</sup> he noted that "no possible biasing or confounding factor considered so far" explained the results.<sup>208</sup>

3. Letters to Scientific Journals

Soon after the publication of Hirayama's paper, the medical journals began to receive letters from tobacco-industry consultants.<sup>209</sup> On March 21, 1981, the *British Medical Journal*, the journal that had originally published Hirayama's paper, published a detailed letter from German epidemiologist Martin Rutsch, who purported to find "apparent inconsistencies" within the subpopulation of unmarried women and the "alleged" difference between urban and rural populations.<sup>210</sup> In light of these discrepancies, he found Hirayama's conclusions to be

209. Some of the letters consisted of broad complaints, rather than detailed critiques. For example, in a letter to the British journal *New Scientist*, tobacco-industry consultant H.J. Eysenck opined that until Hirayama supplied additional information on his studies, he would "hesitate to come to any firm decision." H.J. Eysenck, *More Smoke*, 89 NEW SCIENTIST 494 (1981). At the time that he wrote this letter, Dr. Eysenck was one of the scientists receiving payments from "Special Account # 4," which was administered by the Committee of Counsel of the U.S. Tobacco Industry. He had been paid \$14,421 for the six months ending on February 28, 1981. Greenspan & Yellon, P.C., Audit of Jacob, Meidinger & Finnegan, Special Account # 4 (Feb. 28, 1981) (Bates No. 2015042074).

210. Martin Rutsch, Non-Smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer, 282 BRIT. MED. J. 985 (1981).

<sup>202.</sup> Id. at 4.

<sup>203.</sup> Id. at 8.

<sup>204.</sup> Id.

<sup>205.</sup> Id. at 4.

<sup>206.</sup> Id. at 7.

<sup>207.</sup> P.N. Lee, Notes on Visit to Japan: April 24th to May 1st, 1981, Summary of Our Main Findings (Apr. 5, 1981) (Bates No. 504798226).

<sup>208.</sup> *Id.* Adlkofer's impressions of the meeting were somewhat less positive than Lee's. Memorandum from Frank G. Colby, Telephone Call from Prof. Adlkofer on May 11, 1981 to FGC on Hirayama Public Smoking Study (May 26, 1981) (Bates No. 503681397). Although he believed that Hirayama's philosophy was "very prejudicially anti-smoking," he did not believe that Hirayama was such a "fanatic" that he would "actually falsify figures." *Id.* at 1. Adlkofer believed that Hirayama was "a very rigid epidemiologist" who was unprepared to weigh "physiological and biological realities" in interpreting his raw data. *Id.* 

"problematic."<sup>211</sup> Hirayama later published a response to Rutsch in which he demonstrated that lung cancer rates were virtually identical in married and unmarried nonsmoking women, thus refuting Rutsch's allegations of inconsistencies.<sup>212</sup>

The *British Medical Journal* also published a critical letter from long-time tobacco-industry consultant Theodor D. Sterling, complaining that Hirayama had failed to consider the exposure of Japanese women to indoor air pollution from household heating and cooking equipment.<sup>213</sup> Since smoke from wood fires had "been suggested as a factor in lung cancer etiology" and since cooking with kerosene stoves had "been associated with lung cancer in women in Hong Kong," Hirayama's data would be suspect if more nonsmoking wives of smoking husbands employed those fuels for heating and cooking than nonsmoking wives of nonsmoking husbands. Sterling thought there was good reason to believe this to be the case because a greater proportion of Japanese smokers belonged to the lower classes.<sup>214</sup>

Although Hirayama agreed that Sterling's suggestion "should be explored further," he noted that a recent Japanese study concluded that "there was no increased use of kerosene stoves in households where the husbands smoked."<sup>215</sup> Moreover, he controlled for socio-economic class and found that there was a slightly higher incidence of smoking among males in the higher socio-economic classes.<sup>216</sup> Thus, Hirayama demonstrated that it can be much easier to come up with plausible-sounding theories about confounding factors than it is to produce hard evidence demonstrating their real-world existence. Sterling, however, knew how the doubt-casting game was played, and for many years he maintained that confounding factors accounted for the association between primary smoking and lung cancer.<sup>217</sup>

<sup>211.</sup> *Id.* After his meeting with Hirayama, Peter Lee concluded that Rutsch's first point was based upon a misinterpretation of Hirayama's data and was not well taken. Lee, *supra* note 193, at 7. Lee did not, however, attempt to correct Rutsch's mistake publicly.

<sup>212.</sup> Takeshi Hirayama, Letter to the Editor, 282 BRIT. MED. J. 1393 (1981).

<sup>213.</sup> Theodor D. Sterling, Communication, 282 BRIT. MED. J. 1156 (1981).

<sup>214.</sup> Id.

<sup>215.</sup> Hirayama, *supra* note 212. Although the tobacco industry continued to make much of the cooking habits of Japanese women, other scientists working for the tobacco industry regarded "the kerosene stove 'issue" to be "merely a debating point, and not an important criticism." Memorandum from Frank G. Colby to Max H. Crohn, Jr., Critique of Dr. Hirayama's Comments on Letters to the Editor Critiquing his Paper on Lung Cancer in Non-smoking Wives of Japanese Smokers (May 21, 1981) (Bates No. 504886999).

<sup>216.</sup> Hirayama, supra note 212.

<sup>217.</sup> Dr. Peter J. Burch of the Department of Medical Physics at the Leeds General Infirmary offered a unique critique of the Hirayama study. He suggested that the results were explainable by the hypothesis that "women who marry smokers are more likely to be genetically predisposed to lung cancer than those who marry non-smokers." Peter J. Burch, *Passive Smoking and Lung Cancer*, 282 BRIT. MED. J. 1393 (1981). This hypothesis would, in his view, explain both the data and the Rutsch critique. Because the habit was "openly allowed in the society," he could "conceive of no influence on the selection of partners." *Id.* Hirayama responded that Burch's theory—that the choice of a marriage partner involves one or more factors that are associated with smoking—was apparently not true in Japan "where mating has surely been random at least in terms of smoking." Hirayama, *supra* note 212.

Not all the commentary on the Hirayama study was critical. In a review article for Environmental Research featuring the Hirayama and Trichopoulos studies, E. Cuyler Hammond of the American Cancer Society and Irving J. Selikoff of the Mount Sinai School of Medicine opined that Hirayama adequately controlled for confounding factors.<sup>218</sup> Although the authors worried that the index Hirayama used to estimate passive-smoke exposure was suspect because some husbands probably smoked more at home than elsewhere,<sup>219</sup> any inaccuracy was more likely to weaken the association than enhance it.<sup>220</sup> Similarly, while Hirayama failed to describe his age-standardization method, any resulting bias would likely have tended to reduce the appearance of an upward trend.<sup>221</sup> Finally, although Hirayama did not verify his subjects' cause of death, there was no reason to suspect that error in recording the cause of death on the death certificates of the deceased would be connected with the smoking habits of their husbands.<sup>222</sup> Hammond and Selikoff could identify "no sound reason" to suspect Hirayama's findings "were due entirely to errors, biases, or confounding factors of some sort."223 Still, they wanted more positive evidence though "perhaps not a great deal more"-before they were willing to conclude that passive smoking causes lung cancer.<sup>224</sup>

4. The Mantel Critique and the Garfinkel Study

Two independent developments came together in June 1981 to give the tobacco industry a unique opportunity for deconstructing the Hirayama and Trichopoulos studies. First, Dr. Lawrence Garfinkel of the American Cancer Society published a long-anticipated study. Analyzing the huge multi-year database assembled by E. Culyer Hammond, the industry's arch-nemesis, Garfinkel found no statistically significant increase in lung cancer among nonsmoking wives of smokers. Second, Dr. Kastenbaum of the Tobacco Institute discovered another anomaly in the Hirayama paper that led him to conclude that Hirayama had made a statistical error. The TI then hired Dr. Nathan Mantel, a former National Cancer Institute statistician who had developed the statistical methodology used by Dr. Hirayama, to examine whether Kastenbaum's critique appeared reasonable. The tobacco industry's strategic use of the Garfinkel study and Dr. Mantel's expertise is an ideal example of the corpuscular

<sup>218.</sup> E. Cuyler Hammond & Irving J. Selikoff, *Passive Smoking and Lung Cancer with Comments on Two New Papers*, 24 ENVTL. RES. 444 (1981).

<sup>219.</sup> Id. at 449.

<sup>220.</sup> Id.

<sup>221.</sup> Hammond & Selikoff, supra note 218, at 449.

<sup>222.</sup> Id.

<sup>223.</sup> *Id.* The Trichopoulos study, according to Hammond and Selikoff, was "a small but very interesting case-control study." *Id.* at 450. They found the trends to be "in general agreement with findings." *Id.* The procedures for classifying women by the smoking habits of their husbands seemed reasonable, and the control group appeared to be well chosen. *Id.* at 450-51. Beyond the obvious implications for public policy, the authors noted that the passive-smoking data supported the hypothesis that the carcinogenesis dose-response curve was, at very low doses, not linear but logistic in shape. *Id.* at 452.

<sup>224.</sup> Hammond & Selikoff, supra note 218, at 452.

approach in action.

The industry got wind of Garfinkel's conclusions long before their June publication.<sup>225</sup> Concluding that the paper could, if properly publicized, kill the troublesome passive-smoking issue,<sup>226</sup> the TI's public relations director solicited "the best and senior brains" at the public relations firm Burson-Marsteller to come up with a plan to "off-set the recent[,] widely publicized findings of Hirayama."<sup>227</sup> On the day of publication, the TI's public relations department would distribute a lawyer-approved press release to the media.<sup>228</sup> The TI's federal and administration departments had tested media-pickup possibilities by conducting dry runs in the April and May issues of the Journal of the National Cancer Insti*tute.*<sup>229</sup> The TI's state-relations department would develop a "field distribution plan" that would target state legislators who relied upon the Hirayama study to support passive-smoking legislation.<sup>230</sup> It would also work with volunteers from the industry's grassroots Tobacco Action Network to solicit letters to the editors of local and regional newspapers "underscoring how the new report cancels the old one."231 The industry's Council on Tobacco Research and the Shook Hardy law firm would provide scientists to underscore in the media the significance of the Garfinkel study.<sup>232</sup> A select group of these scientists would accompany the TI's "media tour [of] ten major markets to merchandise [the] Garfinkel findings."233

Meanwhile, a discussion with Professor Chris P. Tsokos, a statistician and industry consultant at the University of South Florida, led Dr. Kastenbaum to conclude that the Hirayama paper contained a "fundamental error."<sup>234</sup> Applying the "Mantel-extension chi test" to the Hirayama data, Kastenbaum concluded there was "not a statistically significant dose-response relationship between lung cancer rates for nonsmoking wives and the level of smoking in their husbands."<sup>235</sup> Kastenbaum speculated that Hirayama had forgotten to take the square root of a critical number and had therefore reported a chi-squared value, rather than the required chi value. Given this serious blunder, Hira-

<sup>225.</sup> See Memorandum from Frank C. Colby to Max H. Crohn, Jr., *supra* note 179, at 5 (noting that Garfinkel had submitted his study for publication in January, 1981, and that Garfinkel came "exactly to *the opposite conclusions as Dr. Hirayama*").

<sup>226.</sup> Letter from William Kloepfer, Jr. to Jim Carr (May 1, 1981) (Bates No. 521024170).

<sup>227.</sup> Id.

<sup>228.</sup> *Id.* at 1. The press release was ultimately approved by both Shook Hardy and Covington & Burling. Memorandum from Samuel D. Chilcote, Jr. to Members of the Executive Committee 1 (May 29, 1981) (Bates No. TIFL 0062181).

<sup>229.</sup> Memorandum from Samuel D. Chilcote, Jr. to TI Operations Committee 1 (May 11, 1981) [hereinafter Chilcote-TI Memorandum].

<sup>230.</sup> Id.

<sup>231.</sup> Id. at 2.

<sup>232.</sup> Id. at 2.

<sup>233.</sup> Id.

<sup>234.</sup> Memorandum from Marvin A. Kastenbaum to Horace Kornegay, Meeting with Prof. C. Tsokos Concerning the Hirayama Paper (June 1, 1981) (Bates No. TI0427-2891); Memorandum from Marvin A. Kastenbaum to Horace Kornegay, Suspected Error in the Hirayama Paper (June 1, 1981) (Bates No. TIBR0016526) [hereinafter Suspected Error Memorandum].

<sup>235.</sup> Suspected Error Memorandum, supra note 234, at 1.

yama's conclusions were "not scientifically or statistically sound."<sup>236</sup> To be on the safe side, however, Kastenbaum decided to consult with Dr. Nathan Mantel, the creator of the statistical test, "to seek an independent evaluation of the results."<sup>237</sup>

Although unwilling to conclude that Hirayama had committed a mathematical error, Mantel agreed that the published report at least raised the question "whether Hirayama has conducted a more refined analysis about which he is giving us no clues, or he has mistakenly interpreted his chi-square[d] value as a chi value."<sup>238</sup> Although a "[m]uch more careful analysis of the data would be needed" to know for certain, Mantel was at least willing to entertain the possibility that Hirayama had made a critical mistake.<sup>239</sup> It was by no means a stinging critique, but it came from a credible source.<sup>240</sup>

Without attempting to contact Dr. Hirayama for an explanation, the Tobacco Institute initiated an aggressive public relations blitz "to discredit Dr. Hirayama's [paper]."<sup>241</sup> The TI's public relations department planned a "one-two punch"<sup>242</sup> strategy under which Burson-Marsteller would publicize Dr. Kastenbaum's allegations on Friday for the weekend press and then initiate a second wave of press releases to accompany the publication of the Garfinkel study, which was expected soon thereafter.<sup>243</sup>

The Tobacco Institute threw the first punch on June 10, when its chairman, Horace Kornegay, sent a telegram to Hirayama's boss, Dr. Sugimura, notifying him of the "very grave" error Dr. Kastenbaum had discovered.<sup>244</sup> Copies of the telegram were provided to Dr. Hirayama, the *British Medical Journal*, and the press.<sup>245</sup> Simultaneously, a lawyer-approved press release announced that Drs. Mantel and Tsokas had "confirmed the existence of a fundamental mathematical error" in the Hirayama study.<sup>246</sup> The release stated that the study had "been used to support unnecessary limitations on individual liberties."<sup>247</sup> The TI public relations department also distributed a packet on the Mantel critique to the

241. Memorandum from the Tobacco Institute to Executive Committee and Stan Temko (June 1, 1981) (Bates No. 500651593).

242. Telex from William Kloepfer, Jr. to Mary Covington (June 10, 1981) (Bates No. 500875018).

243. Memorandum from Sam Chilcote to James C. Bowling et al. (June 11, 1981) (Bates No. TI1190-3142).

244. Telegram from Horace Kornegay to Dr. T. Sugimura (June 10, 1981) (Bates No. TINY0020599).

245. Id.

246. News Release from the Tobacco Institute for Immediate Use Accompanied by Text of Cablegram to Japan and Memorandum from Dr. Mantel 1 (June 10, 1981) (Bates No. 500651585).

247. Id. at 2.

<sup>236.</sup> Id.

<sup>237.</sup> Id. at 4.

<sup>238.</sup> Letter from Nathan Mantel to Marvin A. Kastenbaum (June 5, 1981) (Bates No. TINY0020382).

<sup>239.</sup> Id. at 2.

<sup>240.</sup> Professor Tsokos agreed with Dr. Kastenbaum that Hirayama made an erroneous calculation. Tsokos's application of the Mantel procedure to the data as he interpreted them "fail[ed] to confirm the conclusion that non-smoking wives of heavy smokers have a higher risk of lung cancer." Letter from Chris P. Tsokos to Horace R. Kornegay 2 (June 9, 1981) (Bates No. TI0427-2884).

field force it had assembled to distribute the TI's message on the Garfinkel study. The packet contained orders for the field force to "personally visit more than 200 news organizations nationwide" on Friday, June 12.<sup>248</sup> About 100 video news clips were sent to local television stations, and 400 audiocassettes were sent to large radio stations. Finally, the TI staff made personal visits to elected and appointed officials.<sup>249</sup>

At this point, events took an unexpected turn. A copy of the TI press release found its way to Peter Lee, who realized the industry was about to make a serious mistake. Lee immediately sent a telex to Stuart Alexander of the Tobacco Institute telling him that "Mantel's suspicions that Hirayama has used chi squared instead of chi are in my opinion definitely unfounded."<sup>250</sup> Lee further cautioned that Mantel's letter to Kastenbaum "only suggests there might be [an error] and that more careful analysis is needed."<sup>251</sup> Lee had spoken with Mantel, who communicated that he felt "it probable that the best analysis methods would not significantly alter Hirayama's conclusions."<sup>252</sup>

Lee related that the data Dr. Kastenbaum had given Mantel were not "agestandardized." Although Hirayama had not made this clear in his paper, the data he used were, in fact, standardized for age and occupation. Lee then provided a series of calculations using Hirayama's age-standardized data that yielded a chi (not chi-squared) value of 3.35, which was "very similar to quoted 3.299."<sup>253</sup> The difference was attributable to Hirayama's having broken the table down into five age groups in making his calculations but not having broken down the data that far in his published tables. In any event, it was "clear he did not make any grave error."<sup>254</sup> Lee urged Alexander to persuade the TI not to issue the press release.<sup>255</sup> Otherwise, once someone demonstrated that Hirayama was right, the TI would "be in danger of implicitly agreeing passive smoking causes lung cancer."<sup>256</sup>

Lee's telex created a serious dilemma for the TI. It could have called halted the public relations blitz pending a thorough investigation of Lee's concerns. Instead, it decided to do whatever it could to keep Mr. Lee quiet. On the

<sup>248.</sup> Letter from Samuel D. Chilcote, Jr. to Ernest Pepples (June 11, 1981) (Bates No. TI1190-3139).

<sup>249.</sup> Tobacco Institute, Promotion of Hirayama and Garfinkel Stories, at tab A (June, 1981) (Bates No. 03739559).

<sup>250.</sup> Telex from Peter Lee to Stuart Alexander (June 12, 1981) (Bates No. 517000679).

<sup>251.</sup> Id. at 1.

<sup>252.</sup> Id.

<sup>253.</sup> Id.

<sup>254.</sup> *Id.* at 2. Lee further noted that had Mantel or Kastenbaum "bothered to work out a chi value corrected only for age in two groups" using Hirayama's published data, they would have been interested to note that the chi rose from a non-significant 1.82 to a value of 2.71," which was quite significant. *Id.* Indeed "it would have risen further with finer age groups." *Id.* Lee allowed that Mantel and Kastenbaum "may not have known that Hirayama did not take into account time of death in the analysis by considering every individual year of life" as Mantel had assumed. In his experience, however, "this would only have altered the value marginally tending usually to understate rather than over state differences." *Id.* 

<sup>255.</sup> Telex from Peter Lee to Stuart Alexander, *supra* note 250, at 2.

<sup>256.</sup> Id.

morning of December 12, the day the blitz began, Horace Kornegay reported to the TI Executive Committee that Kastenbaum had spoken with Lee on the telephone, but could not evaluate Lee's concerns because "Lee had not done the calculations."<sup>257</sup> In fact, Lee had performed several detailed calculations and had made them quite explicit in his telex. As far as Kornegay was concerned, however, "Lee's criticism was without merit."<sup>258</sup>

Kornegay further reported that Kastenbaum had just spoken with Dr. Mantel who reported that Lee had called him to discuss the matter and had suggested "that [t]he Tobacco Institute was 'using' him." Incensed, Kornegay declared that "this unwarranted intervention" by Mr. Lee was "regarded by the management of [t]he Institute as officious and without precedent."<sup>259</sup> Kornegay therefore sent a cablegram to Sir James Wilson, the chairman of the TAC, to inform him that the "highly officious actions on the part of your consultant, Mr. Peter Lee, [were] deeply resented by the management of [t]he Tobacco Institute."<sup>260</sup> Kornegay related that he had instructed TI staff that "there shall be no further advanced information supplied [to the TAC] until further notice."<sup>261</sup>

Wilson replied that he was "deeply distressed that our good relations should have been put in jeopardy in the way you describe,"<sup>262</sup> and he promised to institute "a full inquiry at this end."<sup>263</sup> Kornegay "acknowledge[d] with thanks" Wilson's promise and explained why Dr. Kastenbaum believed Lee's calculations (which he apparently now acknowledged) were erroneous. In Wilson's absence, Hugh Grice of the TAC responded that he had asked Lee "to reconsider his calculations in the light of your message."<sup>264</sup>

On another front, Ernest Pepples, general counsel of British American Tobacco Company, called Richard Ely, a scientist at Brown & Williamson Tobacco ("B&W") on June 15 to complain about Lee.<sup>265</sup> Pepples asked Ely "to ensure that Peter Lee was carefully advised not to go direct[ly] to outsiders and to consider very carefully any further statement that he himself would make in order not to compromise his own reputation."<sup>266</sup> Ely noted that Hugh Grice of the TAC had already "made arrangements to put these points across to Peter Lee."<sup>267</sup> In the meantime, B&W would hold up distribution of the TI's public relations package.<sup>268</sup>

261. *Id*.

263. Id.

- 266. Id.
- 267. Id.
- 268. Id.

<sup>257.</sup> Memorandum from Horace R. Kornegay to Executive Committee (June 12, 1981) (Bates No. 03554397).

<sup>258.</sup> Id.

<sup>259.</sup> Id.

<sup>260.</sup> Cablegram from Horace R. Kornegay to Sir James Wilson (June 12, 1981) (Bates No. TI0427-2875).

<sup>262.</sup> Cable from James Wilson to Horace R. Kornegay (June 15, 1981) (Bates No. 03029457).

<sup>264.</sup> Cable from Hugh Grice to Horace R. Kornegay (June 16, 1981) (Bates No. TI10150530).

<sup>265.</sup> Richard Ely, Note for File (June 17, 1981) (Bates No. 521024595).

On June 17, Lee provided a detailed four-page response to the TI's critique of his earlier cable.<sup>269</sup> After probing three possible theories under which the data failed to demonstrate a statistically significant increase in lung cancer among nonsmoking wives of smokers, Lee concluded once again that the chi values in the published paper "seem just about right as far as I can see."<sup>270</sup> Another B&W statistician also examined the Hirayama data in light of Mantel's critique and related that his calculations "agree with Hirayama and Lee."<sup>271</sup>

On June 26, Sir James Wilson wrote to Kornegay to provide the results of the TAC's internal investigation.<sup>272</sup> Wilson was "deeply concerned, whatever the merits of the statistical issue, lest any action by [the TAC] should have made the task of T.I. more difficult."<sup>273</sup> On the other hand, Wilson was "completely satisfied that Peter Lee had no intention of embarrassing the T.I. in any way."<sup>274</sup> Wilson conceded, however, that "in his anxiety to reach the truth" (a goal that the TI was apparently not as anxious to attain), Lee had "somewhat exceeded the proprieties." Wilson assured Kornegay that in the future, messages would "be formally cleared through [the TAC] rather than referred directly to your consultants."<sup>275</sup> However, Wilson by no means conceded that Lee was wrong on the merits. Indeed, after Lee subsequently presented his calculations to the TAC Research Committee, its members unanimously "confirmed their confidence in Mr. Lee's interpretation of the statistics."<sup>276</sup>

Soon thereafter, German tobacco-industry scientist Franz Adlkofer let it be known that he agreed with Lee and believed that Mantel and Kastenbaum were wrong.<sup>277</sup> Adlkofer had met with Hirayama, and Hirayama had sent new data confirming his conclusions. Adlkofer invited Dr. Kastenbaum and Dr. Tsokos to Germany to review the data,<sup>278</sup> but TI chairman Horace Kornegay, "gave a forceful veto" to that idea.<sup>279</sup> Adlkofer later maintained that "Hirayama was correct, that the TI knew it and . . . [the] TI published its statement about Hirayama knowing that the work was correct."<sup>280</sup>

Lee's allegations notwithstanding, the TI's public relations blitz had its intended effect. As planned, stories began to appear over the weekend in major newspapers across the country with titles like "Miscalculation Reported in

<sup>269.</sup> Letter from Peter N. Lee to D.G.I. Felton (June 17, 1981) (Bates No. 521024597).

<sup>270.</sup> Id. at 4.

<sup>271.</sup> Memorandum from A.I.B. to H.B.W. (June 18, 1981) (Bates No. 521024587).

<sup>272.</sup> Letter from James Wilson to Horace R. Kornegay (June 26, 1981) (Bates No. TIBR0016428).

<sup>273.</sup> Id.

<sup>274.</sup> Id.

<sup>275.</sup> Id.

<sup>276.</sup> Minutes of the 14th Meeting of the Research Committee of T.A.C. Held at 11:00 a.m. on Tuesday, 21st July 1981 at Glen House, Stag Place, London, SWIE 5AG (July 21, 1981) (Bates No. 1000089356).

<sup>277.</sup> Memorandum from J.K. Wells, III to E. Pepples, Smoking and Health—Tim Finnegan 2 (July 24, 1981) (Bates No. 521028146).

<sup>278.</sup> Id.

<sup>279.</sup> Id.

<sup>280.</sup> Id.

Study On Cancer in Wives of Smokers,"<sup>281</sup> "Math Mistake Claimed in Smoking-Peril Study,"<sup>282</sup> and "Tobacco Unit Raps Smoking Study."<sup>283</sup> Local newscasts devoted brief segments to the news about the Mantel letter and the TI's unequivocal statement that "an arithmetical error invalidates this study."<sup>284</sup> Sometime later, in a more carefully researched article, the *Medical World News* interviewed Dr. Mantel to determine whether the TI had accurately characterized his opinion.<sup>285</sup> Mantel suggested that Hirayama "either made a more refined analysis, of which he's giving us no details, or perhaps he made an arithmetic error." When presented with the TI press release stating that his memo "invalidate[d]" Hirayama's study, Mantel demurred: "I didn't say that—the Tobacco Institute did."<sup>286</sup> Later, Mantel told *Time* that, while he had raised questions about the study, he had not concluded that it was invalid, as suggested by the TI. He related that the TI had "put words in my mouth."<sup>287</sup>

Dr. Sugimura was traveling when Kornegay's letter arrived,<sup>288</sup> and he therefore had no immediate response. As it happened, he was in Washington, D.C. when he read about the Mantel criticism in the *New York Times*.<sup>289</sup> At a meeting with the TI's public relations director, Sugimura said that the vice-director of his institute had discussed the Mantel critique with Hirayama, who was "very much concerned."<sup>290</sup> Hirayama promised to investigate the matter and send the results of that investigation to Kornegay. Sugimura understood that scientists should "be willing to admit a mistake, but also should challenge a critic if the critic is wrong."<sup>291</sup> Sugimura confided that he "was skeptical of the conclusion of the study, right from the start."<sup>292</sup>

On June 19, 1981, the long-awaited publication of the Garfinkel paper in the *Journal of the National Cancer Institute* provided the opportunity for the second round of the TI's program to discredit Hirayama's paper.<sup>293</sup> Garfinkel's paper examined trends for lung cancer in nonsmokers over a twelve-year period using data from an ongoing prospective epidemiological study initiated by the American Cancer Society ("ACS") in 1960. Acknowledging the Hirayama and

<sup>281.</sup> *Miscalculation Reported in Study on Cancer in Wives of Smokers*, N.Y. TIMES, June 15, 1981, at B7.

<sup>282.</sup> Math Mistake Claimed in Smoking-Peril Study, CHI. TRIB., June 15, 1981.

<sup>283.</sup> Tobacco Unit Raps Smoking Study, BOSTON GLOBE, June 16, 1981.

<sup>284.</sup> Radio TV Reports, Inc., Broadcast Excerpt, WINS-New York, June 14, 1981 (Bates No. TIMN0019450).

<sup>285.</sup> On Passive Smoking: Cancer Society and Tobacco Institute in Rare Unity, MED. WORLD NEWS, July 6, 1981, at 30.

<sup>286.</sup> Id.

<sup>287.</sup> Tobacco Wars: Is Passive Smoking Harmful?, TIME, July 6, 1981, at 43.

<sup>288.</sup> Cable from Ms. Kimura to Horace Kornegay (June 12, 1981) (Bates No. TI1015-0550).

<sup>289.</sup> Memorandum from William Kloepfer to Wilson Wyatt et al. (June 17, 1981) (Bates No. TI0427-2866).

<sup>290.</sup> Id.

<sup>291.</sup> Id.

<sup>292.</sup> Id.

<sup>293.</sup> Lawrence Garfinkel, *Time Trends in Lung Cancer Mortality Among Nonsmokers and a Note on Passive Smoking*, 66 J. NAT'L CANCER INST. 1061 (1981).

Trichopoulos results, Garfinkel undertook a "similar analysis of nonsmokers" identified in the ACS study, despite his concern that classifying nonsmoking women on the basis of the smoking habits of their husbands was not an accurate measure of their exposure to ETS.<sup>294</sup> His analysis showed that the ratio of adjusted lung cancer deaths in women whose husbands smoked fewer than twenty cigarettes per day to those women whose husbands never smoked was 1.37, but the ratio with respect to wives of husbands who smoked more than twenty cigarettes per day was only 1.04.<sup>295</sup> Neither of the differences was statistically significant, and the trend was clearly in the wrong direction to support a causation hypothesis.<sup>296</sup>

Although these results by no means demonstrated that environmental tobacco smoke was incapable of causing cancer in nonsmokers, the facts that the Garfinkel study was based on a larger data set than the Hirayama study and that the data were assembled in the United States, suggested that the conclusions of the Hirayama and Trichopoulos studies were of questionable relevance to the health risks of ETS in the United States.<sup>297</sup> Yet Trichopoulos agreed that neither his study nor Hirayama's provided definitive information on the health effects of ETS.<sup>298</sup>

The Tobacco Institute issued its pre-arranged press release stating that the industry was "convinced from the outset of the highly questionable nature of the Japanese finding that smokers create a lung cancer risk in their nonsmoking spouses."<sup>299</sup> The TI was therefore pleased "to learn of the new American Cancer Society study contradicting that finding."<sup>300</sup> This, coupled with the TI's discovery of the error in the Hirayama study "and the many scientific criticisms of it which have been published in the *British Medical Journal*, fully support[ed] our misgivings."<sup>301</sup> The TI regretted that the Hirayama study "led to so many news stories, editorials and other demands for smoking prohibitions from those who unfortunately accepted it at face value."<sup>302</sup>

Like the Mantel critique, the Garfinkel story received a great deal of attention in the press. A *New York Times* headline read: "Research Casts Doubt on

<sup>294.</sup> Id. at 1064.

<sup>295.</sup> Id.

<sup>296.</sup> Id. at 1065. Peter Lee reviewed the Garfinkel paper in April, prior to its publication. P.N. Lee, "Time Trends in Lung Cancer Mortality Among Nonsmokers and a Note on Passive Smoking," Some Comments on the Paper by L. Garfinkel to Appear in the Journal of the National Cancer Institute, (Apr. 3, 1981) (Bates No. 2025016956). Lee found no reason to believe that the Garfinkel data were materially biased. Id. at 3. Lee found that "Garfinkel's findings are not consistent with the estimated doubling of risk in heavy smokers given by Hirayama." Id. at 5. Thus, despite the consistent findings of the Hirayama and Trichopoulos papers, "more evidence is needed for a number of reasons." Id. at 1.

<sup>297.</sup> Garfinkel, supra note 293, at 1966.

<sup>298.</sup> Tobacco Wars, supra note 287.

<sup>299.</sup> Tobacco Institute, For Release (June 19, 1981) (Bates No. TI1185-1418).

<sup>300.</sup> Id.

<sup>301.</sup> Id.

<sup>302.</sup> Id.

Danger of Cancer To Wives of Smokers."<sup>303</sup> Most of the stories also made reference to the Mantel critique.<sup>304</sup> In total, more than 684 newspaper and magazine articles appeared during June and July on the Mantel critique, the Garfinkel study, or both.<sup>305</sup> Tobacco-industry representatives appeared on seventyfive news and talk shows to relate "the doubts raised about the Japanese study."<sup>306</sup>

To be absolutely certain its message did not escape the public's attention, the industry launched a major advertising campaign in which it placed two fullpage advertisements in twenty major newspapers during early August, 1981.<sup>307</sup> The ads were titled "Here's What's *Now* Being Said about Tobacco Smoke in the Air" and featured headlines from several newspapers on the Mantel letter and the Garfinkel study.<sup>308</sup> The text of the ads claimed that "several eminent biostatisticians found an apparent statistical error in the Japanese calculations raising serious questions about the study.<sup>309</sup> The ad campaign, however, was the last straw for Dr. Mantel. He told the press that he had "been offended by the way they represented what I have done.<sup>310</sup> Furthermore, in his view, Garfinkel's results were "not inconsistent with [those of] Hirayama.<sup>3311</sup>

Meanwhile, the scientific debate continued to rage in the professional journals. The *British Medical Journal* responded to Kornegay's alarming letter to Sugimura by offering to publish it and the Mantel correspondence as letters to the editor.<sup>312</sup> It published those letters and another criticism of Hirayama's study by Dr. Eleanor J. MacDonald, another long-time tobacco-industry consultant, in the October 1981 issue.<sup>313</sup> In the same issue, the journal published a

307. Tobacco Industry Ad Campaign Ignites Dispute, S.F. CHRON., Aug. 14, 1981, at 4.

308. Tobacco Institute, Here's What's *Now* Being Said about Tobacco Smoke in the Air (undated) (Bates No. TINY0011832).

311. Id.

<sup>303.</sup> Research Casts Doubt on Danger of Cancer to Wives of Smokers, N.Y. TIMES, June 20, 1981, at A1.

<sup>304.</sup> Id.

<sup>305.</sup> Public Relations Department, Tobacco Institute, The Hirayama Controversy: An Analysis of Media Activity, June 15-July 31, 1981 (Bates No. TITX0027702).

<sup>306.</sup> Tobacco Institute, Public Relations Division 1981 Highlights (December 10, 1981) (Bates No. TIMN0017335, TIMN0017341) [hereinafter 1981 Highlights]. Having been informed that the Garfinkel paper would be published in the June issue of the *Journal of the National Cancer Institute*, the Tobacco Institute had already launched a "Garfinkel Paper/Letter-to-Editor Program" through its nationwide Tobacco Action Network ("TAN"). Memorandum from Jack Kelly to TAN Corporate Coordinators, Garfinkel Paper/Letter-to-Editor Program (June 1, 1981) (Bates No. TIFL0062177). A memorandum to the TAN Corporate Coordinators predicted that the Garfinkel study would indicate "that the Hirayama study ... does not apply to American women." *Id.* at 1. Since the study "spurred significant legislative efforts to restrict smoking," it was important "that public knowledge of the conflicting ACS results be as widespread as possible." *Id.* The Tobacco Institute hoped that TAN could accomplish this through a massive nationwide campaign of letters to the editors of local newspapers. *Id.* The memo suggested language that might be included in the editorials. *Id.* at 2.

<sup>309.</sup> Id.

<sup>310.</sup> Tobacco Industry Ad Campaign Ignites Dispute, supra note 307.

<sup>312.</sup> Letter from Daphne Gloag to Horace R. Kornegay (June 15, 1981) (Bates No. TI0427-2865).

<sup>313.</sup> Horace J. Kornegay & Marvin A. Kastenbaum, Letter, 283 BRIT. MED. J. 914 (1981); Eleanor

J. MacDonald, Letter, 283 BRIT. MED. J. 915 (1981); Nathan Mantel, Letter, 283 BRIT. MED. J. 914 (1981); see Letter from Patrick M. Sirridge to Thomas F. Ahrensfeld, et al. (Oct. 7, 1981) (Bates No.

critique of the Mantel letter by two MIT statisticians and Hirayama's own defense of his paper.<sup>314</sup> Hirayama provided a table containing the age- and occupation-specific data Kastenbaum had demanded, and he noted that his calculations had been confirmed by the two MIT statisticians and by statisticians at the National Cancer Institute.<sup>315</sup> He believed Dr. MacDonald had simply "misunderstood" the statistical method he had employed.<sup>316</sup> Finally, Hirayama was not surprised by the Garfinkel study's conclusions. He noted that the "discrepancy could be partly due to different methodology"<sup>317</sup> and partly due to the fact that Garfinkel obtained a much lower response rate because he had relied on volunteer surveyors.<sup>318</sup>

\* \* \*

The publication of the Hirayama study generated a legitimate scientific debate over the extent to which the reported data justified Hirayama's conclusions. For example, *Newsweek* reported that Dr. Nicholas Wald of Oxford University "voiced surprise" at the findings because, by his calculations, a nonsmoker's indirect exposure from a one-pack-per-day smoker would be less than a single cigarette per day.<sup>319</sup> Similarly, Dr. Garfinkel, who was no friend of the tobacco industry, concluded that, despite the Hirayama study, ETS "did not appear to be an important problem in the overall picture."<sup>320</sup> The tone of the initial scientific debate, however, was cautious and nuanced by comparison with the industry's highly orchestrated attempt to trash Hirayama's work in both the scientific literature and the lay media.

319. Matt Clark & David Shapiro, *Secondhand Smoke and Lung Cancer*, NEWSWEEK, Jan. 26, 1981, at 63. The Tobacco Institute worked Dr. Wald's skeptical observations into a brief "backgrounder" that it sent out soon after the publication of the study for use in answering initial press inquiries. Telex from Robert H. Bockman to W. Campbell (Jan. 27, 1981) (Bates No. 2504013075).

320. Garfinkel, supra note 293, at 1066.

<sup>01336493) (</sup>carrying a recommendation from Shook Hardy to the Committee of Counsel that funding for Dr. MacDonald be continued and attaching a copy of her letter to the *British Medical Journal*).

<sup>314.</sup> Jeffrey E. Harris & William H. DuMouchel, Letter, 283 BRIT. MED. J. 915 (1981); Takeshi Hirayama, Letter, 283 BRIT. MED. J. 916 (1981).

<sup>315.</sup> Hirayama, supra note 314.

<sup>316.</sup> Id.

<sup>317.</sup> Id.

<sup>318.</sup> Id. The scientific debate continued in the British Medical Journal, which published the Tsokos critique and a brief critique by Peter Lee in its November issue. Tsokos maintained that Hirayama had still not adequately explained how he derived his chi value of 3.29, and he related that no one had been able to come up with that exact number using published data. Chris P. Tsokos, Letter, 283 BRIT. MED. J. 1464 (1981). Lee first noted that Mantel had not taken age and occupation into account in his criticism of the Hirayama paper, but he also pointed out that Hirayama had apparently made a minor statistical error in his response to Mantel in the previous issue. The very small dose of ETS that nonsmokers received led Lee to "seriously doubt whether the elevated lung cancer risk seen in nonsmoking wives of smokers, statistically significant as it may be, is really caused by the passive smoke exposure." Peter N. Lee, Letter, 283 BRIT. MED. J. 1465, 1466 (1981). Lee viewed it as "far more likely that the explanation lies in some hitherto undiscovered confounding or biasing factor." Id. Hirayama agreed with Lee that his most recent table contained errors, and he corrected them in an accompanying table. They did not, however, "influence the substance" of his earlier letter. As to the issue of dose, Hirayama suggested that the "only way to answer such questions must be by carrying out in-depth studies of the chronic toxicity of sidestream smoke and of health consequences resulting from prolonged exposure to passive smoking." T. Hirayama, Letter, 283 BRIT. MED. J. 1466 (1981).

The press release on the Mantel letter is a classic example of the rhetoric of the corpuscular approach to scientific evidence. The industry spun Dr. Mantel's rather tentative conclusion that Kastenbaum's analysis raised questions about the Hirayama study into a confirmation of the existence of a fundamental mathematical error invalidating Hirayama's conclusions. The unconfirmed possibility of a mathematical error rendered Hirayama's analysis scientifically worthless and therefore entirely ignorable, despite the supporting evidence of the Trichopoulos study. Ignoring Peter Lee's convincing demonstration that Kastenbaum's criticism was misplaced, the industry steadfastly maintained its position on the Hirayama study in legislative and administrative settings for the next two decades, even as additional published studies suggested an association between passive smoking and lung cancer. As each important study was released, the industry hauled out the scientific blunderbuss and its effective public relations apparatus and attempted to blow it away.

In the final analysis, the scenario Lee envisioned when he warned the TI not to go forward with its public relations blitz did play out in the scientific literature. Hirayama was able to demonstrate through a letter to the editor that he had in fact used the chi test, and Mantel made it clear that he felt he had been used by the Tobacco Institute, as Lee had suggested. Yet the TI did not recant. Instead, it steadfastly maintained for the next twenty years that the study had been the subject of many "highly critical" letters to scientific journals and was therefore "scientifically questionable."<sup>321</sup>

The industry regarded its efforts to discredit the Hirayama paper as a huge success<sup>322</sup> and held them up as a model for future scientific debates. A late-1981 Liggett & Meyers memorandum argued: "Our recent experience with the Hirayama Report demonstrate[s] the need for promptly critiquing research *to determine its validity* in order to identify the appropriate response to negative research, be it scientific or statistical in nature."<sup>323</sup> The industry's goal was to attack the scientific underpinnings of individual studies as they became available. The studies were then to be rejected as "junk science" and, as such, irrelevant to the debate over the dangers of smoking.

Most of the original skeptics who were not industry consultants later changed their views as more evidence became available. Dr. Wald, for example, later authored a report for a committee empanelled by the National Research Council concluding that the studies demonstrated a "highly significant association" between ETS and lung cancer that could not be explained by sys-

<sup>321.</sup> See, e.g., Scientific Affairs Division, Tobacco Institute, Public Smoking in Perspective 2 (Sept. 28, 1981) (Bates No. TNWL0039291) (observing that "many highly critical letters about the . . . study appeared shortly after its publication").

<sup>322.</sup> See 1981 Highlights, *supra* note 306; Memorandum from E.A. Horrigan to J. Tylee Wilson et al., Tobacco Institute-Hirayama/Garfinkel Publicity (June 30, 1981) (Bates No. 503947352) (noting that "the end result was one of the best executed programs to come out of the Institute in a long time and the successful achievement of major newspaper, radio and TV publicity for 'our side'").

<sup>323.</sup> Memorandum from Liggett Meyers, Tobacco Institute Proposal—New Directions (undated) (Bates No. 502484395) (emphasis added).

tematic bias.<sup>324</sup> The paid industry consultants never changed their minds and continued to attack the studies as they came out. When EPA prepared its risk assessment, the industry recruited these same consultants to trash that document, and they were happy to oblige.

#### B. EPA Prepares a Draft Risk Assessment

In the years immediately following the publication of the Hirayama, Trichopoulos, and Garfinkel studies, additional case-control studies on the adverse health effects of exposure to ETS began to appear. In 1986, Surgeon General C. Everett Koop issued a report concluding that the cancer risks of smoking could "extend to those who inhale tobacco smoke emitted into the air."<sup>325</sup> Noting that sidestream smoke was "qualitatively similar to the mainstream smoke inhaled by the active smoker," the report concluded that "nonsmokers are exposed to levels of environmental tobacco smoke that would be expected to generate a lung cancer risk."<sup>326</sup> Adopting a weight-of-the-evidence approach, and drawing upon both the Hirayama and Garfinkel studies, the report declared that "[t]he relative abundance of data reviewed ..., their cohesiveness, and their biologic plausibility allow a judgment that involuntary smoking can cause lung cancer in nonsmokers."<sup>327</sup>

During the same year, the Committee on Passive Smoking appointed by the National Research Council of the National Academy of Sciences ("NAS") published a report based upon a comprehensive review of the scientific literature.<sup>328</sup> Although noting that the "physiochemical nature" of ETS differed from that of mainstream smoke, the Committee pointed out that ETS contained many of the same toxic chemicals as mainstream smoke.<sup>329</sup> A causal connection between ETS and disease was therefore "biologically plausible."<sup>330</sup> For the Committee, "[t]he weight of evidence derived from epidemiologic studies," including both the Hirayama and Garfinkel cohort studies and several case-control studies, demonstrated an association between ETS exposure and lung cancer that was "unlikely to be due to chance or systematic bias."<sup>331</sup> The Committee therefore

<sup>324.</sup> Eliot Marshall, Tobacco Science Wars, 236 SCIENCE 250, 250 (1987).

<sup>325.</sup> OFFICE ON SMOKING & HEALTH, U.S. DEP'T OF HEALTH & HUMAN SERVS., THE HEALTH CONSEQUENCES OF INVOLUNTARY SMOKING: A REPORT OF THE SURGEON GENERAL, ix (1986) [hereinafter SURGEON GENERAL REPORT].

<sup>326.</sup> Id. at x.

<sup>327.</sup> *Id.* The report noted that the Hirayama study had "been critically discussed in correspondence since its initial publication." *Id.* at 74. In particular, Hirayama's "failure to specifically describe the methods for age standardization in the initial report led to speculation that the statistical methods used were incorrect," but "the calculations were later confirmed." *Id.* at 76

<sup>328.</sup> COMM. ON PASSIVE SMOKING, NAT'L ACAD. OF SCI., ENVIRONMENTAL TOBACCO SMOKE: MEASURING EXPOSURES AND ASSESSING HEALTH EFFECTS (1986).

<sup>329.</sup> Id. at 7-8.

<sup>330.</sup> Id. at 11.

<sup>331.</sup> Id. at 245.

concluded that "[c]onsidering the evidence as a whole, exposure to ETS increases the incidence of lung cancer in nonsmokers."  $^{332}$ 

In addition to activity on the scientific front, on October 17, 1986, Congress enacted the Radon Gas and Indoor Air Quality Research Act of 1986 ("the Radon Act").<sup>333</sup> Pursuant to that statute,<sup>334</sup> in 1988, EPA's small indoor-air staff asked the agency's Office of Research and Development ("ORD") to prepare a risk assessment on ETS.<sup>335</sup> The project proceeded slowly, in part because of the lively debate it precipitated between ORD and the Air Program's Criteria and Assessment Office in the spring of 1990.<sup>336</sup>

Neither the Surgeon General's nor the NAS report dissuaded the tobacco industry from sustaining its attack on the Hirayama study. The industry continued its references to the Mantel critique and the media reports about that critique.<sup>337</sup> The tobacco industry was also well aware of the significance of EPA's forthcoming ETS risk assessment. A February 1990 briefing paper prepared by RJR scientist Guy B. Oldaker warned that the ETS issue represented "the single strongest attack" tobacco opponents had ever launched against the industry.<sup>338</sup> The threat, however, was based upon epidemiology, an inexact science. Since the opponents would never be able to prove as a purely scientific matter that ETS caused lung cancer in nonsmokers, Oldaker predicted that the "driving force will ultimately shift to the public domain," which is occupied by institutions such as EPA and the Occupational Safety and Health Administration ("OSHA") that seek to "maintain their existence and . . . grow" by filling power vacuums resulting from environmental disputes.<sup>339</sup> The industry, Oldaker argued, could respond in three ways to the ETS science problem: "We can

339. *Id.* at 1, 3.

<sup>332.</sup> *Id.* at 8.

<sup>333.</sup> Pub. L. No. 99-499, §§ 401-405, 100 Stat. 1758.

<sup>334.</sup> Id. at § 403(c). Enacted on October 17, 1986, the Radon Act required EPA to "establish a research program with respect to radon gas and indoor air quality." Id. at § 403(a). The program was to include "research and development concerning the identification, characterization, and monitoring of the sources and levels of indoor air pollution" and "research relating to the effects of indoor air pollution and radon on human health." Id. at § 403(b)(1-2). EPA was to assemble an interagency committee of federal officials from agencies dealing with indoor air pollution and an advisory committee of individuals from the states, the scientific community, industry, and public interest organizations to assist the agency in implementing the program. Id. § 403(c). However, the Act specifically declined to authorize EPA to "carry out any regulatory program or any activity other than research, development, ... related reporting, [and] information dissemination." Id. at § 404.

<sup>335.</sup> Letter from William K. Reilly to Thomas J. Bliley, Jr. (June 19, 1991) (Bates No. TI00951497).

<sup>336.</sup> See Memorandum from Chris DeRosa to William H. Farland, Review of "Lung Cancer Hazards and Other Respiratory Effects Due to Exposure to Environmental Tobacco Smoke," (Apr. 27, 1990) (Bates No. 515705640) (concluding that the risk assessment was "most comprehensive, thorough and well-written," representing "a conscientious and detailed effort on a complex subject," and "the culmination of a solid analysis," but suggesting various improvements in using risk assessment data on mainstream smoke to assess ETS risks and in how the risk assessment addressed a major meta-analysis of the ETS data).

<sup>337.</sup> See Tobacco Institute, Tobacco Smoke and the Nonsmoker: Scientific Integrity at the Cross-roads 27-31 (Oct. 28, 1986) (Bates No. 2021002344).

<sup>338.</sup> Guy B. Oldaker, III, Briefing Paper on Environmental Tobacco Smoke (ETS) 1 (Feb. 15, 1990) (Bates No. 513000665).

ignore the issue.... We can mount a vigorous protest to the assaults on our industry.... We can match the efforts of the opposition at every practical opportunity and from all levels of our company and industry."<sup>340</sup> The industry adopted the third approach.

On June 25, 1990, EPA made its draft risk assessment ("DRA") available for public review and comment.<sup>341</sup> Relying heavily upon the analyses of the scientific literature contained in the NAS and Surgeon General's reports for its analysis of the pre-1986 studies, the DRA provided a comprehensive analysis of the literature published between 1986 and 1989 and integrated the results of all of the studies into a single qualitative and quantitative whole.<sup>342</sup>

The DRA reached three important and controversial conclusions. First, ETS was a Group A human carcinogen.<sup>343</sup> According to EPA's carcinogen assessment guidelines, this meant there was "sufficient evidence from epide-miologic studies to support a causal association between exposure to [ETS] and cancer."<sup>344</sup> Second, using a conservative quantitative risk-assessment model, EPA estimated that exposure to ETS caused approximately 3,800 lung cancers per year.<sup>345</sup> Third, EPA concluded that exposure of infants and young children to ETS from parental smoking was associated with increased prevalence of acute lower respiratory tract infections (bronchitis and pneumonia), respiratory symptoms of irritation (cough, sputum, wheeze), and middle-ear effusions (a sign of chronic middle-ear disease), decreased lung function in childhood, and a small reduction in the rate of pulmonary growth and development.<sup>346</sup>

At the heart of the DRA was a meta-analysis of the combined data from nineteen case-control studies and the data from the three cohort studies: the Hirayama study, the Trichopolous study, and the Garfinkel study.<sup>347</sup> The

<sup>340.</sup> Id. at 4.

<sup>341.</sup> U.S. Envtl. Protection Agency, Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children; External Review Draft, 55 Fed. Reg. 25,874 (June 25, 1990) [hereinafter EPA Risk Assessment]. Although it contains a May 1990 date, the draft risk assessment was not released until June 25, 1990. The primary author was EPA consultant Kenneth G. Brown, who wrote all of the chapters of the DRA and Appendix B. *Id.* at xiv.

<sup>342.</sup> *Id.* at x.

<sup>343.</sup> *Id.* at 1-3.

<sup>344. 1986</sup> Final Guidelines, *supra* note 9, at 34,000. EPA concluded that if ETS did not cause cancer in nonsmoking wives, then four of the nineteen existing case-control studies were false positives, and the probability of that happening was 0.013. The analysis further concluded that more than half of the nineteen studies had p-values of 0.1 or less, an outcome that would occur with the probability of less than 0.001 if cancer incidence in nonsmoking wives of smokers was unrelated to ETS exposure. EPA Risk Assessment, *supra* note 341, at 3-21. Moreover, the trends in the fourteen studies that allowed for dose-response supported the conclusion that "the observed association between exposure to ETS and increased occurrence of lung cancer deaths [was] statistically significant." *Id.* at 3-25.

<sup>345.</sup> EPA Risk Assessment, supra note 341, at 1-5.

<sup>346.</sup> Id. at 1-5 to 1-9.

<sup>347.</sup> *Id.* at 4-24. The DRA noted that the Hirayama study had received much critical scrutiny over the years, but Hirayama had "adequately addressed [the criticisms] as described in the [Surgeon General] and [NAS] reports." *Id.* at 3-34. The DRA also cited the published results of a roundtable discussion at a 1984 symposium for the proposition that "previous challenges to Hirayama's work regarding data analysis and other issues appear to have been resolved, aside perhaps from the issue of mis-

result was an overall relative risk of 1.41, somewhat larger than the relative risk of 1.32 for females reported in the NAS study. Since the EPA meta-analysis included more data, its confidence interval was somewhat smaller, a reflection of the reduced statistical uncertainty.<sup>348</sup> The 1.42 relative risk for the case-control studies was almost identical to the 1.39 relative risk for the cohort studies, so EPA concluded that "the results of one type of study reinforced the outcome from the other type."<sup>349</sup>

The authors of the DRA understood that the studies probably reflected some misclassification bias. They applied a complicated formula to account for this bias, which reduced the relative risk for the meta-analysis to 1.28.<sup>350</sup> The numbers had to be adjusted upward again, however, to reflect nonsmoking women's exposure to background levels of ETS outside of the home.<sup>351</sup> This resulted in a relative risk of 1.48 for the combined case-control and cohort data,<sup>352</sup> a figure in the same range as the 1.34 relative risk reported in the NAS study, which had used eight fewer case-control studies.

Finally, the conclusion that ETS was a Group A carcinogen was biologically plausible. The DRA cited numerous scientific studies demonstrating that the sidestream smoke ("SS") emitted from a smoldering cigarette between puffs—the main component of ETS—contained many of the same carcinogenic compounds scientists had identified in mainstream smoke ("MS").<sup>353</sup> EPA understood that nonsmoker exposure to these carcinogens varies because the composition of chemicals in ETS and their relative distribution between vapor and particulate phases change over time.<sup>354</sup> Moreover, passive exposure to the chemical constituents of ETS occurs at much lower concentrations than active exposure to the same constituents in MS.<sup>355</sup> Nevertheless, since any level of exposure to a carcinogen carries a potential cancer risk, the excess risk reported in many of the epidemiologic studies made sense biologically.<sup>356</sup>

#### C. Review by EPA's Science Advisory Board

Tobacco industry scientists and dozens of consultants spent the summer and early fall of 1990 preparing more than twenty-five densely detailed critiques of

356. Id. at 1-2.

reported smoking habits." *Id.* (citing G. Lehnert, *Roundtable Discussion from Symposium "Medical Perspectives on Passive Smoking*," 13 PREVENTIVE. MED. 732, 732-33 (1984)).

<sup>348.</sup> Id.

<sup>349.</sup> Id.

<sup>350.</sup> EPA Risk Assessment, *supra* note 341, at 4-25. The formula used to reduce the relative risk was explained in a detailed appendix.

<sup>351.</sup> *Id.* at 4-28. The DRA noted that after the NAS panel adjusted its estimate of the increased risk associated with spousal smoking from thirty-four to twenty-five percent to reflect misclassification bias, it made a second adjustment to take background exposures into account, and this raised the increased risk back up to forty-two percent. *Id.* at 3-32.

<sup>352.</sup> Id. at 4-24.

<sup>353.</sup> *Id.* at 1-2.

<sup>354.</sup> Id. at 2-1.

<sup>355.</sup> EPA Risk Assessment, supra note 341, at 1-2, 2-1.

the DRA. The critiques were submitted to the agency's Science Advisory Board ("SAB"), which agreed to review the document for scientific accuracy.<sup>357</sup> Philip Morris, RJR, and the TI coordinated their responses so that at least one submission touched upon every conceivable aspect of the DRA, and so that every submission forcefully made the same important general points.<sup>358</sup>

On October 1, 1990, the industry submitted massive filings containing thousands of pages of comments and criticisms.<sup>359</sup> The TI's cover letter suggested that the entire EPA effort was a waste of time and resources because the existing studies provided "no basis on which to conduct a risk assessment."<sup>360</sup> Philip Morris maintained that the DRA's classification of ETS as a Group A carcinogen was "based solely on uncritical acceptance of several human epidemiological studies" that had been heavily criticized in the scientific literature.<sup>361</sup> Finding EPA's risk assessment "analogous to a lawyer's brief," RJR argued that EPA's "[p]olicy preferences should not be foisted upon the lay public disguised as science."<sup>362</sup>

The bulkiest submission was a 220-page letter by Gary Huber purporting to discuss every published study on ETS and human disease, including "several

362. Id. at 81.

<sup>357.</sup> Tobacco Institute, Public Affairs Management Plan Progress Report, Public Smoking Issue 1 (Aug. 1990) (Bates No. TIDN0017470) [hereinafter August Management Report]. The TI's Public Smoking PR staff continued to work on "approximately 25 scientific reviews to be submitted to EPA at the close of its comment period and to the SAB in advance of the Board's hearing on the risk assessment and policy guide." The SAB is a formal EPA advisory committee composed of distinguished scientists, engineers, and economists from around the country. Congress created EPA's SAB in the Environmental Research, Development and Demonstration Act of 1978, 42 U.S.C. § 4365 (2000). According to the statute, the SAB was to be composed of at least nine independent members who would provide scientific advice to the administrator. The SAB was to review proposed criteria documents and provide advice and comments to the administrator regarding the scientific and technical basis of proposed criteria, standards, limitations or regulations. EPA has traditionally limited the SAB to about sixty permanent members who serve for staggered terms of one to four years. More than 250 additional scientists and engineers serve as consultants to the SAB on various issues where additional expertise is deemed desirable. The SAB has a staff of about twenty EPA employees.

<sup>358.</sup> *Id.* (stating that the TI staff "continued coordinating the industry's submissions on both draft documents with member companies who planned to submit independent comments."); Tobacco Institute, Public Affairs Management Plan Progress Report, Public Smoking Issue 1 (July 1990) (Bates No. TIDN0017433) ("We continued coordinating the industry's submissions on both draft documents with two member companies who planned to submit independent comments."). This was not inexpensive. For the month of July, TI's legal expenses for the public smoking issue came to more than \$308,000. During the same month, TI spent more than \$1,300,000 on professional fees for the "concerted activity of scientists on ETS research and the EPA risk assessment." August Management Report, *supra* note 357, at 7.

<sup>359.</sup> Philip Morris, Inc., EPA Review Draft: Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children, Comments of Philip Morris, Inc. (Sept. 28, 1990) (Bates No. 2028557177) [hereinafter Philip Morris Comments]; R.J. Reynolds Tobacco Co., Comments of R.J. Reynolds Tobacco Company on Health Effects of Passive Smoking—Assessment of Lung Cancer in Adults and Respiratory Disorders in Children (Oct. 1, 1990) (Bates No. 508142133) [hereinafter RJR Comments]; Tobacco Institute, Comments of the Tobacco Institute on Health Effects of Passive Smoking—Assessment of Lung Cancer in Adults and Respiratory Disorders in Children (Oct. 1, 1990) (Bates No. 508142139) [hereinafter TI Comments].

<sup>360.</sup> Letter from Samuel D. Chilcote, Jr. to the Project Officer for Environmental Tobacco Smoke (Oct. 1, 1990) (Bates No. 503246519).

<sup>361.</sup> Philip Morris Comments, *supra* note 359, at 1-2.

thousand more articles than were cited in the EPA review draft."<sup>363</sup> The product of an entire year of analysis at the tobacco industry's expense, Huber's criticism that literature cited in the DRA was "selective and . . . not complete"<sup>364</sup> had an authoritative ring. Since EPA staff declined to provide a detailed description of every published study of any conceivable relevance to the association between ETS and lung cancer, instead relying on the work already done in support of previous reports by a committee of the National Research Council and the Surgeon General, Huber could plausibly claim that the DRA would have reached a substantially different conclusion if only its scientists had matched his efforts.

Apparently willing to be used again by the TI, Nathan Mantel wrote a fiftytwo-page critique of the DRA that was very critical Hirayama's most recent report. Dr. Mantel believed Hirayama had not satisfactorily answered a 1983 guest editorial in which Mantel had pointed out "the highly defective nature of the Hirayama study and analysis, including its failure to take into account properly the cohort nature of the study."<sup>365</sup> Mantel asserted that the high relative risk in the Hirayama study "could well reflect a greater misclassification bias, with married women denying that they were in fact smokers, *i.e.*, giving the seemingly correct answer rather than the factual answer."<sup>366</sup> The frequently repetitious submissions of several other industry consultants also criticized the Hirayama study.<sup>367</sup> Even Peter Lee had something negative to say about its exposure index's reliance upon whether the spouse currently smoked, rather than the more appropriate index of whether the spouse ever smoked.<sup>368</sup>

The tobacco industry and its consultants had a number of criticisms of EPA's meta-analysis techniques and the ETS data it had selected the analysis. First, they maintained that EPA improperly applied recognized meta-analysis techniques.<sup>369</sup> Dr. Mantel warned that if several of the individual studies

<sup>363.</sup> Letter from Gary Huber to the Project Officer for Environmental Tobacco Smoke 1 (Sept. 28, 1990) (Bates No. 525012050).

<sup>364.</sup> Id. at 1.

<sup>365.</sup> Nathan Mantel, Commentary 10 (Sept. 18, 1990) (Bates No. 2023475261).

<sup>366.</sup> *Id.* at 10-11.

<sup>367.</sup> See Earl Jonas, Comments on the EPA Review Draft: "Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children" 5 (Sept. 28, 1990) (Bates No. 87655618); S. James Kilpatrick, Comments on Dr. Hirayama's Record Linkage Study of Japanese Adults in "Epidemiological Evidence of Lung Cancer From ETS" 10 (Sept. 24, 1990) (Bates No. 87662455); Maurice E. LeVois & Maxwell W. Layard, Summary of Major Criticisms of EPA's Draft Risk Assessment: Health Effects of Passive Smoking 11 (Sept. 25, 1990) (Bates No. 876656106); Karl Uberla, Comments on the Draft Document—Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children and Environmental Tobacco Smoke: A Guide to Workplace Smoking Policies 3-11 (Sept. 21, 1990) (Bates No. 876666437); Lawrence M. Wexler, Comments on the EPA Draft Document Entitled "Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children" 11 (Sept. 26, 1990) (Bates No. 2046090141).

<sup>368.</sup> P.N. Lee, "Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disease in Children": A Commentary on Issues Relating to Lung Cancer in the May 1990 EPA External Review Draft 10 (Sept. 19, 1990) (Bates No. 507971697).

<sup>369.</sup> RJR Comments, supra note 359, at 12-13.

contained similar biases, then meta-analysis could allow those biases "to reinforce each other."<sup>370</sup> Thus, even though the bias in any single study might be too small to be statistically significant, the biases could combine in a meta-analysis "to give rise to an overall appearance of statistical significance" that was scientifically unjustified.<sup>371</sup>

Second, the industry argued that EPA improperly included and excluded data from individual studies. EPA should have limited the meta-analysis to studies undertaken in the United States, thus eliminating the Hirayama study from the meta-analysis.<sup>372</sup> At the same time, it should have included a study published in a dissertation that found no link between ETS and cancer.<sup>373</sup> The drafters of the report had considered the dissertation, but failed to include the data in the meta-analysis because the author had died, and his advisor could not be reached to verify the otherwise unpublished data.<sup>374</sup>

Third, the industry's scientists criticized the meta-analysis for combining data from studies that were not sufficiently similar to yield accurate comparisons.<sup>375</sup> For example, the industry argued that combining case-control and cohort studies was inappropriate because different durations of observation could introduce bias into the combined analysis.<sup>376</sup> The demand for compara-

373. Luis R. Varela, Assessment of the Association Between Passive Smoking and Lung Cancer (May 1987) (Bates No. 2023382403); *see* Butler, *supra* note 372, at 3; Lee, *supra* note 368, at 9; NCF Comments, *supra* note 372, at 1; Philip Morris Comments, *supra* note 359, at 58; RJR Comments, *supra* note 359, at 23-24; Springall, *supra* note 372, at 4; Uberla, *supra* note 367, at 3; Wexler, *supra* note 372, at 6.

374. Philip Morris undertook a large public relations campaign criticizing EPA for missing the study. *See* Richard Kluger, ASHES TO ASHES (1996). Janerich later published a complete analysis of his data that concluded that a statistically significant association did exist. *See* Dwight T. Janerich et al., *Lung Cancer and Exposure to Tobacco Smoke in the Household*, 143 NEW ENG. J. MED. 632 (1990).

375. Philip Morris Comments, *supra* note 359, at 2; *see also* Letter from J.W. Daniel to Project Officer for Environmental Tobacco Smoke 2 (Sept. 14, 1990) (Bates No. 87655009) (arguing that the EPA meta-analysis was "unreliable because the individual ETS studies vary significantly in study design, methodology, sample population, confounding risk factors, etc."); Wexler, *supra* note 367, at 5 (arguing that "the extreme differences in data collection, methodology, and measure of ETS exposure among the 21 studies make any test for statistical homogeneity meaningless").

376. RJR Comments, *supra* note 359, at 15; *see also* E. Lee Husting, An Epidemiological Review of the EPA Report: Health Effects of Passive Smoking, Assessment of Lung Cancer in Adults and Respiratory Disorders in Children 13 (Sept., 1990) (Bates No. 87663772) (arguing that studies with different

<sup>370.</sup> Mantel, *supra* note 365, at 13; *see also* LeVois & Layard, *supra* note 367, at 8 ("Since the possible effects of bias in confounding are not adequately controlled in the individual studies, pooling the studies in a meta-analysis greatly increases the probability that the systematic effects of these errors will be significant and introduces the additional possibility that publication bias has restricted attention mainly to positive results.").

<sup>371.</sup> Mantel, *supra* note 365, at 13.

<sup>372.</sup> Letter from W.H. Butler to the Project Officer for Environmental Tobacco Smoke 3 (Sept. 26, 1990) (Bates No. 2023474857); LeVois & Layard, *supra* note 367, at 8; National Chamber Federation, ETS Comments 1 (Aug. 15, 1990) (Bates No. TIMN 343325) [hereinafter NCF Comments]; Letter from Petr Skrabenek to the Project Officer for ETS 1 (Sept. 18, 1990) (Bates No. 2023475720); A. Springall, Submission of Comments on the Draft EPA Report: Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children 5 (Sept. 1990) (Bates No. 507972473); Memorandum from Paul Switzer to the Project Officer for Environmental Tobacco Smoke, Comments on the EPA Review Draft Health Effects of Passive Smoking 5 (Sept. 27, 1990) (Bates No. 503246500); R.L. Tweedie & K.L. Mengersen, Lung Cancer and Exposure to Environmental Tobacco Smoke: Appendix 1: Review of Individual Studies 10 (1990) (Bates No. 507758436).

bility was easy for the industry to make—and difficult for EPA to satisfy because there are few fully agreed-upon epidemiological protocols. If the industry was demanding that all epidemiological studies make all observations and adjust for all confounding factors in identical ways, then no meta-analysis would ever be possible. The question was essentially whether EPA should have undertaken a meta-analysis at all.

Fourth, the industry was not happy with the way EPA adjusted its relative risk calculations upward to reflect background exposures to ETS that nonsmoking wives receive outside the home because that adjustment was appropriate only if ETS exposure in fact causes lung cancer.<sup>377</sup> The industry argued that this constituted circular reasoning: EPA was assuming that ETS caused lung cancer as part of its attempt to determine whether ETS caused lung cancer. However, given that several studies independently demonstrated a positive association between ETS exposure and lung cancer, it was not necessarily circular for EPA to assume the association existed for purposes of cumulating data from many studies, several of which alone lacked the statistical power to support relative risk determinations at low relative risk ratios.

Finally, the industry was especially critical of EPA's willingness to draw cause-effect conclusions from a meta-analysis that demonstrated, at best, only a very weak statistical association between ETS exposures and lung cancer in humans.<sup>378</sup> Although EPA's guidelines specified no quantitative threshold for strength of association, the industry commenters argued that relative risks of 2.0 to 5.0 were required.<sup>379</sup> The strength of association necessary to support a statistically significant conclusion that a causal relationship exists is ultimately a policy question dependent upon the epidemiologist's willingness to be wrong. It is not surprising that the industry was more risk averse in this regard than EPA. The DRA, however, left it to the reader to decide whether the statistical association was sufficiently powerful to justify conclusions about carcinogenic risks posed by ETS.

durations of observation may not be combined in a meta-analysis or serious bias might result because, for example, air pollution levels could have changed over the years).

<sup>377.</sup> RJR Comments, *supra* note 359, at 32; *see also* Mantel, *supra* note 365, at 7 (arguing that background adjustment "presupposes that there is indeed a passive smoking effect").

<sup>378.</sup> Philip Morris Comments, *supra* note 359, at 1 ("[T]he notion of strength of association [was] not applied to the epidemiologic studies on ETS."); Tweedie & Mengersen, *supra* note 372, at 8 (citing EPA guidelines for proposition that strength-of-association is one of the standard criteria for evaluating an epidemiological study).

<sup>379.</sup> Daniel, *supra* note 375, at 2 (stating that a relative risk value of less than 3.0 is "generally considered to represent an extremely weak association"); Husting, *supra* note 376, at 17 (arguing that an association of less than 3.0 relative risk is weak); Mantel, *supra* note 365, at 3 (stating that "relative risks should exceed 1.50 to be taken seriously"); Philip Morris Comments, *supra* note 359, at 8 ("[A]n association is generally considered weak if the odds ratio is under 3.0 and particularly when it is under 2.0."); RJR Comments, *supra* note 359, at 49–50 (noting that EPA's staff at a September 1989 workshop on EPA's guidelines suggested that a relative risk below 5.0 might be considered weak); Tweedie & Mengersen, *supra* note 372, at 8 (arguing that a relative risk of greater than 3.0 required); Wexler, *supra* note 367, at 7 ("[R]elative risks of less than 2.0 are generally considered to be weak.").

The industry and its contractors were also unimpressed by EPA's biological plausibility conclusion.<sup>380</sup> They argued that ETS was a poorly characterized and dynamic mixture of thousands of compounds that differed both qualitatively and quantitatively from both mainstream smoke and sidestream smoke.<sup>381</sup> Many factors affected the composition of ETS, including dilution, room volume, ventilation, temperature, humidity, lighting, and absorption by surfaces.<sup>382</sup> The distribution between the particulate and vapor phases of mainstream smoke differed substantially from the distribution in ETS.<sup>383</sup> Indeed, that the cancers in smokers caused by mainstream smoke (squamous-cell and small-cell carcinomas) were by and large not the same as the cancers allegedly caused in non-smoking wives by ETS (adenocarcinomas) appeared to undercut any argument based upon biological plausibility.<sup>384</sup>

The industry argued that dosimetric considerations also augered against a finding that ETS exposure caused lung cancer. The concentrations of potential carcinogens in ETS were orders of magnitude smaller than those measured in mainstream smoke.<sup>385</sup> The very modest exposures to any carcinogenic constitu-

382. Reasor & Will, supra note 381, at 6.

383. Alan Rodgman, A Comparison of the Chemical, Physical, and Biological Properties of Cigarette Mainstream Smoke (MS), Cigarette Sidestream Smoke (SS), and Environmental Tobacco Smoke (ETS) (1991) (Bates No. 2026125296). For example, nicotine in the more acidic MS occurred almost exclusively in the particulate phase, whereas nicotine in ETS occurred almost exclusively in the vapor phase. *Id.* This difference in phases might or might not affect toxicity.

385. Philip Morris Comments, *supra* note 359, at 13-14; *see also* Daniel, *supra* note 375, at 2 (noting that "exposure of non-smokers to ETS is two or more orders of magnitude less than the exposure of

<sup>380.</sup> Edward L. Alpen, A Critique of EPA External Review Draft 600/6-90/006A, May 1990, "Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children" 5 (Feb. 1991) (Bates No. 2026125439) (stating that the DRA's biological plausibility approach "cannot stand close scientific scrutiny"); Butler, *supra* note 372, at 4 (arguing that the assumption that ETS has roughly the same constituents as mainstream smoke is "probably incorrect"); Lee, *supra* note 368, at 17 (arguing that the DRA failed to discuss properly "the plausibility of the association seen between spousal smoking and lung cancer"); Mantel, *supra* note 365, at 2 (rejecting the reasoning that "if cigarette smoking can cause lung cancer, then passive smoking is also likely to cause lung cancer").

<sup>381.</sup> Philip Morris Comments, *supra* note 359, at 13-14; *see also* Statement of John Wesley Clayton, Jr. 8 (undated) (Bates No. 503244740) (pointing to "profound qualitative and quantitative differences among (1) mainstream smoke, (2) side-stream smoke, and (3) ETS"); Husting, *supra* note 376, at 45-46 (citing numerous references for the proposition that ETS is a complex mixture that is not identical to mainstream smoke); Mark J. Reasor & James A. Will, Evaluation of the EPA Draft Report Health Effects of Passive Smoking: Assessment of Lung Cancer and Respiratory Disorders in Children 4 (Sept. 1990) (Bates No. 2026188180) (arguing that "ETS is insufficiently characterized relative to mainstream smoke, that the exposure conditions involving the two materials differ significantly, and that extrapolation from mainstream smoke exposure to ETS exposure accordingly are problematic"); RJR Comments, *supra* note 359, at 60; Skrabanek, *supra* note 372, at 1-2 (rejecting the concept of "incriminating a complex mixture of substances, whose chemical and physical properties are poorly understood without reference to concentrations and exposure times"); John A. Todhunter, Review of the Draft EPA Document Entitled "Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children" 38 (Sept. 1990) (Bates No. 503246583) (noting that mainstream smoke and ETS are very different in composition and behavior).

<sup>384.</sup> Letter from F. Adlkofer to Project Officer for ETS Policy Guide 2-3 (Sept. 26, 1990) (Bates No. 503244675); Butler, *supra* note 372, at 3-4; Guy Crepat, A Commentary on Issues Relating To Lung Cancer in the 1990 EPA External Review Draft 3 (Sept. 25, 1990) (Bates No. 2026127628); Letter from D. Schmahl to the Project Officer for ETS Policy Guide 1 (July 25, 1990) (Bates No. 508136421); Skrabanek, *supra* note 372, at 1.

ents encountered by nonsmokers in ETS were, in the industry's view, well below the thresholds necessary to invoke a carcinogenic response.<sup>386</sup> In addition to challenging the standard nonthreshold model of chemical carcinogenesis, the industry even challenged EPA's base assumption that "smoking is a causative factor for lung cancer in humans."<sup>387</sup>

Although ETS probably contained as many compounds as mainstream smoke, no one knew what they were. Thus, the industry could plausibly argue that chemical similarity could not support the drawing of conclusions about the carcinogenicity of ETS from existing studies on mainstream smoke. The argument that there is a threshold exposure level to most carcinogens, below which as a practical matter the carcinogen will cause no tumors, was a familiar one to regulatory agencies, and the industry added nothing new to the debate in that regard. Although it is certainly possible that thresholds do exist for some carcinogens, no federal regulatory agency has been willing to assume this is the case, and the tobacco industry by no means demonstrated that thresholds existed for the carcinogenic compounds identified by EPA and the NAS in environmental tobacco smoke.

At the end of a two-day meeting in December 1990, at which most of the tobacco industry's criticisms were aired, the SAB committee sent the DRA

active smokers to mainstream smoke"); Huber, supra note 363, at 2 (arguing that ETS constituents are "highly diluted by a factor of 100 to 1000 or more relative to mainstream tobacco smoke"); Husting Comments, supra note 376, at 45 (noting "serious questions about the dosages of ETS received by a non-smoker and smoker"); LeVois & Layard, supra note 367, at 10 (arguing that a cigarette equivalent risk two or three orders of magnitude lower than EPA's estimate based upon epidemiological data alone "challenge[d] the biological plausibility of the effect observed in the ETS epidemiologic studies"); Rodgman, supra note 383, at iv (positing "MS/ETS dilution ratios" for nicotine (ranging from 57,000 to 7,200,000), acrolein (1500 to 2800), acetone (240 to 2000), benzene (112 to 7000) and benzo(a)pyrene (68 to 40,000)); Springall, supra note 372, at 5 (noting that dosimetry-based estimates of lung cancer deaths were an order of magnitude lower than EPA's estimates based on the epidemiology studies); Theodor D. Sterling et. al., Evaluation of a Report by the U.S. Environmental Protection Agency on: Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disorders in Children 2-3 (Sept. 20, 1990) (Bates No. 2026133314) (arguing that the "number of lung cancers associated with ETS by the use of the cigarette-equivalent model is less by more than an order of magnitude than the number of lung cancers that can be ascribed to ETS using the epidemiologic model").

<sup>386.</sup> Daniel, *supra* note 375, at 2-3 (arguing that the assumption that there is no safe level for carcinogens is controversial); Jonas, *supra* note 367, at 2 (arguing that EPA's assumption that any level of exposure to a carcinogen carried a potential risk of cancer was "far from proven" and was "contradicted by a considerable body of evidence"); Comments by George B. Leslie, FRC Path., on May 1990 EPA External Review Draft: "Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults and Respiratory Disease in Children" 1-2 (1990) (Bates No. 2026133012) (arguing that EPA's view was "contrary to the view of many toxicologists, and is also contrary to common human experience"); Rodgman, *supra* note 383, at iii (arguing that "a practical threshold limit value does exist for known tumorigens" and that "[t]his has been demonstrated not only for MS . . . but also for several of the MS components on [EPA's] list of 43"); Skrabanek, *supra* note 372, at 1-2 (rejecting presumption that ETS constituents lack thresholds); Todhunter, *supra* note 381, at 15 ("[D]ata in the observed range are as consistent with there being no effect at very low exposures as there are with there being an effect.").

<sup>387.</sup> RJR Comments, *supra* note 359, at 58. Rodgman, *supra* note 383, at iv (arguing that tumor-inhibiting agents in MS and ETS offset tumorigenic compounds).

back to EPA for further work.<sup>388</sup> The committee concurred with EPA's ultimate conclusion that ETS was a Class A carcinogen,"<sup>389</sup> but it advised the agency to place greater weight on the "biological considerations and the extensive experience with active smoking to support the classification."<sup>390</sup> The committee agreed that spousal smoking was an appropriate indicator of ETS exposure and that epidemiological studies from other countries were relevant to the analysis.<sup>391</sup> Furthermore, meta-analysis was "an appropriate tool to summarize the epidemiological studies," and EPA had adequately accounted for confounding factors in its meta-analysis.<sup>392</sup> However, the DRA had over-emphasized the meta-analysis and shortchanged the biological considerations related to respiratory carcinogenesis that provided "compelling evidence that it is consistent with the results of the meta-analysis."<sup>393</sup> Since the document was "incomplete in many respects," the committee recommended organizational changes and offered suggestions for buttressing its technical content.<sup>394</sup>

Seventeen months later, EPA circulated for comment a much more comprehensive risk assessment in advance of a second SAB meeting on July 21 and 22, 1992. The tobacco industry once again submitted hundreds of pages of criticisms of the new draft. This time, the SAB committee concluded that the revised draft had an "improved presentation, discussion, and analysis of ETS as a lung carcinogen, providing a firmer basis for the designation of ETS as an EPA Class A Carcinogen," a classification it unanimously endorsed.<sup>395</sup> The committee was confident the risk assessment would be "an invaluable source of information for health professionals and policymakers for years to come."<sup>396</sup>

Having received the SAB's endorsement, EPA published its final risk assessment ("FRA") in December 1992.<sup>397</sup> Once again it concluded that ETS was a human carcinogen, and this time it estimated that ETS was "responsible for approximately 3,000 lung cancer deaths annually in U.S. nonsmokers."<sup>398</sup> The FRA relied upon the biological plausibility of the conclusion that ETS caused lung cancer, given "the established causal relationship between lung

<sup>388.</sup> Letter from Raymond Loehr & Morton Lippman to William K. Reilly (Apr. 19, 1991) (Bates No. 2026096384); *see also* Indoor Air Quality and Total Human Exposure Comm., Envtl. Protection Agency, Environmental Tobacco Smoke Review, at 165 (Dec. 5, 1990) (Bates No. 87209741).

<sup>389.</sup> *Id*.

<sup>390.</sup> Id.

<sup>391.</sup> Id. at 2-3.

<sup>392.</sup> Id. at 3-4.

<sup>393.</sup> Id.

<sup>394.</sup> Id.

<sup>395.</sup> Letter from Raymond C. Loehr & Morton Lippmann to William K. Reilly 2 (Nov. 20, 1992) (Bates No. 515926835).

<sup>396.</sup> Id.

<sup>397.</sup> Office of Atmospheric and Indoor Air Programs, Envtl. Protection Agency, Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders, EPA/600/6-90/006F (Dec. 1, 1992), *available at* http://oaspub.epa.gov/eims/eimsapi.dispdetail?deid=2835 (last visited Apr. 12, 2003) [hereinafter FINAL RISK ASSESSMENT].

<sup>398.</sup> Id. at 1-1.

cancer and active smoking."<sup>399</sup> Since several new epidemiological studies, including the Janerich study, had become available in the interim, the FRA now incorporated thirty studies rather than twenty-two.<sup>400</sup> Nine of the thirty individual case-control and cohort studies demonstrated an association between lung cancer and spousal ETS exposure at the 90% confidence level; the probability of this happening by chance was less than one in ten thousand.<sup>401</sup> Reacting to tobacco-industry criticism, EPA elected not to undertake an overall meta-analysis of all the epidemiological studies. Instead, it performed smaller meta-analyses of groups of studies arranged by country and exposure level.<sup>402</sup> The FRA found increased risks for the highest exposure groups without exception, and the relative risk estimates "pooled within countries [were] all statistically significant and range[d] from 1.38 to 3.11."<sup>403</sup>

#### D. The Court's Opinion

Six months after EPA issued the final risk assessment, the Flue-Cured Tobacco Cooperative Stabilization Corporation and several tobacco companies sued the agency in the U.S. District Court for the Middle District of North Carolina.<sup>404</sup> They alleged that EPA violated the Radon Act,<sup>405</sup> the Administrative Procedure Act,<sup>406</sup> and the Due Process Clause of the U.S. Constitution.<sup>407</sup> The group sought a declaratory judgment that EPA's classification of ETS as a human carcinogen was arbitrary and capricious, and prayed for a permanent injunction requiring EPA to withdraw its risk assessment.<sup>408</sup> The group claimed that the publication of the risk assessment caused harm to the tobacco industry because it damaged the reputation of its products and because it would predictably result in additional attempts to regulate smoking in public places.<sup>409</sup>

The district court first addressed the industry's contention that EPA violated the Radon Act both substantively and procedurally.<sup>410</sup> The parties agreed that the meaning of the Statute was plain, so the dispute could be resolved without resorting to step two of the prescribed *Chevron* analysis,<sup>411</sup> but they dis-

401. *Id*.

405. 42 U.S.C. § 7401 nt. (2000).

- 407. U.S. CONST. amend. V.
- 408. Flue-Cured Tobacco, 1995 U.S. Dist. LEXIS 7521, at \*9-10.

409. Id. at \*12.

<sup>399.</sup> *Id.* at 1-7.

<sup>400.</sup> Id. at 1-6.

<sup>402.</sup> FINAL RISK ASSESSMENT, supra note 397, at ch. 5.

<sup>403.</sup> *Id.* at 5-62.

<sup>404.</sup> Flue-Cured Tobacco Coop. Stabilization Corp. v. EPA, No. 6:93CV00370, 1995 U.S. Dist. LEXIS 7521 (M.D.N.C. May 23, 1995).

<sup>406.</sup> Administrative Procedure Act, 5 U.S.C. §§ 701-706 (2000).

<sup>410.</sup> Flue-Cured Tobacco Coop. Stabilization Corp. v. EPA, 4 F. Supp. 2d 435, 439-40 (M.D.N.C. 1998), vacated and remanded by 313 F.3d 852 (4th Cir. 2002).

<sup>411.</sup> In *Chevron, U.S.A., Inc. v. Natural Resources Defense Council, Inc.*, 467 U.S. 837 (1984), the Supreme Court articulated a two-part test for judicial review of agency interpretations of the statutes that they administer:

agreed on the Statute's plain meaning. The court rejected the industry's substantive argument that the Radon Act does not authorize EPA to prepare an ETS risk assessment. In the court's view, the Statute's requirement that EPA conduct research on the adverse health effects of indoor air pollutants and disseminate those findings plainly empowered EPA to prepare a formal risk assessment of ETS and to classify ETS according to its pre-existing carcinogen classification scheme.<sup>412</sup>

The court found that EPA had erred procedurally, however, when, instead of assembling a separate advisory committee under the Radon Act, it had allowed a special committee of its existing Scientific Advisory Board ("SAB") to perform the advisory role the Act envisioned.<sup>413</sup> The court found two problems with EPA's procedural shortcut. First, the Radon Act required EPA to establish a representative advisory committee. The use of the word "establish" suggested that Congress meant for EPA to create a new committee, not borrow an existing standing committee. The second problem was that the Radon Act also provided a role for the existing SAB in reviewing EPA's broad indoor-air research plan. Had Congress intended for a committee of the SAB to double as the statutory advisory committee, it presumably would have said so in the Radon Act.<sup>414</sup> Although perhaps insufficiently deferential to the agency's interpretation of its own statute, the court's statutory analysis was by no means unreasonable.

However, the court did not simply remand to the agency with instructions to appoint a new advisory committee. The tobacco industry persuaded the court to rule on whether EPA's modest procedural impropriety affected the assessment itself. To answer this question and to address the plaintiffs' substantive claims, the court engaged in a lengthy, detailed, and unnecessary inquiry into the scientific merits of the FRA that is an indicator of things to come should the

412. 4 F. Supp. 2d at 442-43.

414. 4 F. Supp. 2d at 445 ("Had Congress meant SAB when requiring a representative advisory group, Congress would have specified SAB as it did in the subsequent paragraph.").

When a court reviews an agency's construction of the statute which it administers, it is confronted with two questions. First, always, is the question whether Congress has directly spoken to the precise question at issue. If the intent of Congress is clear, that is the end of the matter; for the court, as well as the agency, must give effect to the unambiguously expressed intent of Congress. If, however, the court determines Congress has not directly addressed the precise question at issue, the court does not simply impose its own construction on the statute, as would be necessary in the absence of an administrative interpretation. Rather, if the statute is silent or ambiguous with respect to the specific issue, the question for the court is whether the agency's answer is based on a permissible construction of the statute.

<sup>467</sup> U.S. at 842-43.

<sup>413.</sup> This was an especially odd contention for the industry to make because at the time EPA was preparing the DRA, the industry was strongly insisting that EPA have the document and the associated workplace policy statement reviewed by the SAB. Letter from Clausen Ely to Robert Axelrad 2 (Apr. 23, 1990) (Bates No. 2023989033). At that time, the industry and its lawyers did not mention their concern that the SAB was not composed of representatives of the various interest groups. In fact, it strongly opposed one appointment to the committee, Dr. David M. Burns, on the ground that his views were representative of those of the anti-smoking groups. Letter from Samuel D. Chilcote to William K. Reilly (Aug. 10, 1990) (Bates No. 2023388016).

courts adopt a regulatory-*Daubert* approach to judicial review of agency risk assessment.

1. Biological Plausibility

The court first examined the agency's biological plausibility finding: that ETS is likely to cause lung cancer because mainstream smoke is a well-known lung carcinogen. The industry had argued that ETS and mainstream smoke are not sufficiently similar in chemical composition to permit EPA to draw conclusions about the health effects of ETS from data concerning mainstream smoke.<sup>415</sup> This argument appeared to be supported in one FRA conclusion that "ETS is rapidly diluted into the environment, and consequently, passive smokers are exposed to much lower concentrations of these agents than are active smokers."416 But this observation rested on EPA's rejecting the argument that it could quantitatively assess health risks posed by ETS by expressing the nonsmoker dose as a cigarette-equivalent dose. As the court noted, EPA had rejected cigarette equivalence because the absolute and proportional quantities of the components and physical states of ETS and mainstream smoke differ substantially, because the agency was uncertain which tobacco-smoke constituents actually cause cancer, and because it did not know whether there were significant differences in the way humans metabolize ETS versus mainstream smoke.417

The court noted the industry argument that the same uncertainties beclouding cigarette equivalence also undermined EPA's reliance on data regarding the health effects of mainstream smoke. Additionally, the industry claimed that EPA paid insufficient attention to several industry-commissioned analyses of the biological plausibility issue<sup>418</sup> and that it had failed to identify its criteria for determining chemical similarity.<sup>419</sup> Finally, the court observed the industry's challenge to EPA's assumption that there was no threshold concentration below which exposure to mainstream smoke would not cause human carcinogenic effects.<sup>420</sup>

Id.

419. *Id.* at 453. In this regard, the industry noted that EPA had previously declined to use chemical similarity to classify chemical substances as human carcinogens. *Id.* 

<sup>415. 4</sup> F. Supp. 2d at 451-52. The court believed this to be a matter of some importance to the overall ETS risk assessment:

The importance of Plaintiffs' arguments is that the biological plausibility analysis establishes Chapter 5's "a priori hypothesis" that ETS is a Group A carcinogen. EPA uses this hypothesis to justify the use of one-tailed significance tests, which the Agency in turn relies upon to switch from a 95% to 90% confidence interval.

<sup>416.</sup> FINAL RISK ASSESSMENT, supra note 397, at 2-8.

<sup>417. 4</sup> F. Supp. 2d at 452.

<sup>418.</sup> *Id.* at 453 nn.24-25. In this regard the court referred to several hundred pages of analysis contained in the following submissions: Comments of Cronan (JA 6,188); Comments of Gori (JA 10,839); Comments of Todhunter (JA 10,072); Comments of Flamm (JA 10,633-34); Comments of Newell (JA 10,660-61); Comments of Reasor (JA 10,786); Comments of the Tobacco Institute (JA 9,537-38, 9,543); Comments of Reasor (JA 10,789-90); Comments of R.J. Reynolds (JA 5,841-58); and Comments of Philip Morris (JA 10,012, 10,024).

<sup>420.</sup> Id.

The court then restated EPA's response that is had never suggested ETS and mainstream smoke were chemically identical. It had, in fact, repeatedly noted the dissimilarities and pointed out that the chemical constituents of ETS are diluted by the surrounding air. The FRA's drafters had nevertheless also carefully analyzed exposure and biomarker studies of the extent humans inhale and metabolize the chemical constituents of ETS.<sup>421</sup> The industry's argument, the court observed, ignored the distinction EPA had consistently drawn between qualitative hazard assessment conclusions and quantitative exposure assessment: statements made in one context were not necessarily inconsistent with those made in the other.<sup>422</sup> EPA had repeatedly stressed that risk assessment is an art that cannot be bound by hard-and-fast rules.<sup>423</sup>

Following its carefully balanced recapitulation of these arguments, the court proceeded to analyze them in a most confused manner. EPA had made clear that the apparent discrepancies the industry seized upon stemmed from EPA making *qualitative* comparisons of the chemical constituents of mainstream smoke and ETS for the purpose of *qualitative* hazard assessment, while refuting the industry's attempt to use mainstream smoke and ETS exposure comparisons for the purpose of *quantitative* exposure assessment.

The FRA included a thorough and convincing rationale for rejecting the industry's suggested cigarette-equivalence approach. The agency noted that "[t]he differences in size distribution for [mainstream smoke] and [sidestream smoke] particles, as well as the different breathing patterns of smokers and non-smokers, have implications for deposition of the produced particle contaminants in various regions of the respiratory tract."<sup>424</sup> Yet "[d]espite quantitative differences and potential differences in phase distributions, the air contaminants emitted in [mainstream smoke] and [sidestream smoke] are qualitatively very similar in their chemical composition because they are produced by the same process."<sup>425</sup> Indeed, "the available data indicate that tobacco combustion results in the emission of a large number of known toxic compounds and that many of these will be released at rates that are higher in [sidestream smoke] than in [mainstream smoke]."<sup>426</sup>

With respect to exposure assessment, the agency had observed that environmental concentrations of ETS constituents in indoor air resulted from a "complex interaction" of nine variables, variations in any one of which could have "a marked impact" on the ETS constituent concentrations.<sup>427</sup> With so

425. Id.

<sup>421.</sup> Id. at 455.

<sup>422.</sup> Id. at 454.

<sup>423. 4</sup> F. Supp. 2d at 454.

<sup>424.</sup> FINAL RISK ASSESSMENT, supra note 397, at 3-3.

<sup>426.</sup> Id. at 3-7.

<sup>427.</sup> Id. at 3-12. The nine factors included:

<sup>1)</sup> the generation rate of the contaminant(s) from the tobacco (including both [sidestream smoke] and exhaled [mainstream smoke] emissions), 2) location in the space that smoking occurs, 3) the rate of tobacco consumption, 4) the ventilation or infiltration rate, 5) the concentration of the contaminant(s) in the ventilation or infiltration air, 6) air mixing in the space,

many factors at play in assessing ETS exposures, it is not surprising that EPA was uncomfortable with crude attempts to quantify ETS risks through cigaretteequivalence estimates. However, EPA did cite numerous studies demonstrating that "1) many of the contaminants of health interest found in [sidestream smoke] are also found in ETS; 2) ETS contaminants are found above background level in a wide range of indoor environments in which smoking occurs; and 3) the concentrations of ETS contaminants indoors can be highly variable."<sup>428</sup>

The court recognized EPA's point that, in addressing the four components of quantitative risk assessment—hazard identification, dose-response assessment, exposure assessment, and risk characterization—it is sometimes useful to examine qualitative aspects of the data as well as quantitative aspects.<sup>429</sup> In particular, it was not inconsistent to stress the chemical similarities in concluding that ETS was probably carcinogenic in its hazard assessment while at the same time refusing to rely upon those similarities by taking a cigarette-equivalence approach to quantifying nonsmoker exposures in the quantitative exposure assessment. Thus, it was simply erroneous for the court to conclude that "EPA apparently used a different risk assessment methodology for each chapter."<sup>430</sup> EPA's treatment of mainstream smoke and ETS was not at all inconsistent from a scientific perspective given the important distinction between qualitative hazard assessment and quantitative exposure assessment and the recognition that risk assessment is a multi-step process.

Having apparently misunderstood what EPA was attempting to accomplish in the report, the court continued *ad hominem*, suggesting "the ugly possibility that EPA [had varied its] methodology ..., without explanation, based on the outcome sought in that chapter."<sup>431</sup> The court found it "striking that [mainstream smoke] and ETS were similar only where such a conclusion promoted finding ETS a carcinogen."<sup>432</sup> However, this assessment of the science was not at all striking and was entirely within the range of scientific plausibility. From the standpoint of precautionary risk assessment, EPA's approach was not only warranted but desirable.

The court was also troubled by EPA's apparent failure to identify in advance the criteria that would guide its determination whether ETS was chemically similar to mainstream smoke.<sup>433</sup> Noting that EPA conceded that large uncertainties beclouded its attempts to draw conclusions about the car-

Id.

<sup>7)</sup> removal of contaminants by surfaces or chemical reactions, 8) re-emission of contaminants by surfaces, and 9) the effectiveness of any air cleaners that may be present.

<sup>428.</sup> FINAL RISK ASSESSMENT, supra note 397, at 3-13.

<sup>429. 4</sup> F. Supp. 2d at 454-55.

<sup>430.</sup> *Id.* at 456.

<sup>431.</sup> *Id*.

<sup>432.</sup> *Id.* at 457.

<sup>433. 4</sup> F. Supp. 2d at 457. ("The record presents no evidence of EPA establishing similarity criteria before the Assessment.").

cinogenicity of ETS from the known carcinogenicity of mainstream smoke,<sup>434</sup> the court wondered how "EPA could still classify ETS a known human carcinogen based on similarities between [sidestream] and [mainstream smoke]."<sup>435</sup> Apparently, the burden was on EPA to undertake the intensive scientific analysis of chemical similarities required to draw firm conclusions before writing a report that used such similarities as a basis for drawing conclusions about the hazards of ETS. The court thus rewarded the tobacco industry's strategy of casting doubt, but never undertaking the scientific studies necessary to resolve the underlying question.

The court further faulted EPA for relying upon the epidemiology studies to buttress its bioplausibility conclusion. The court believed that it was "circular for EPA to . . . argue the epidemiology studies support the agency's a priori [bioplausibility] theory" because "[w]ithout the theory, the [epidemiological] studies would likely have done no such thing."<sup>436</sup> A risk assessment is, of course, more convincing if the risk assessor can point to a theoretical biological explanation for the statistical association the epidemiological studies reveal. The current state of biological science does not, however, provide theoretical confirmation for empirical observations, especially for exposures to complex mixtures like ETS. At best, EPA could conclude from chemical similarity and other considerations that a cause-effect relationship was sensible as a matter of biology. EPA was not arguing in circles; it was making the subtler point, consistent with the weight-of-the-evidence approach, that the epidemiological studies and the chemical similarity studies were mutually reinforcing.

Finally, the court alluded to dissenting voices within EPA to bolster its conclusion that the FRA lacked a sound scientific basis. Scientific disagreement is probably less commonplace in an agency than in the scientific community in general, but differences in judgment about whether the scientific data support a particular classification are not at all unusual. If unanimity were required every time a conclusion must to be reached on the basis of sketchy data and in the face of large uncertainties, conclusions would never be possible. The fact that some EPA scientists believed ETS belonged in the Group B-1 category did not especially undermine the agency's conclusion that ETS belonged in the next higher category. The upper-level decisionmakrs within EPA apparently believed the agency scientists supporting the Group A classification had the better internal argument. Although Judge Osteen, the judge who presided over the *Flue-Cured Tobacco* case, might have resolved the internal agency dispute differently, Judge Osteen was not the administrator of EPA.

<sup>434.</sup> Id. at 457 n.31.

<sup>435.</sup> *Id.* at 457. The court also noted that "[t]he record also fails to explain whether or how EPA determined that, because some components of ETS may be absorbed, questions raised in other areas of the assessment about the carcinogenic potential of ETS were no longer relevant." *Id.* 

<sup>436.</sup> Id. at 456.

#### 2. EPA's Meta-analysis of the Epidemiological Data

The court paid careful attention to the scientific validity of EPA's metaanalysis. Since the meta-analysis was fundamental to the risk assessment, any rejection of that analysis was sufficient to invalidate EPA's Group A carcinogen conclusion and it therefore would have ratified the tobacco industry's claim that ETS had not been shown to cause cancer. The court first evaluated industry claims that EPA cherry-picked favorable studies for inclusion in the metaanalysis. It then evaluated the scientific validity of EPA's choice of confidence intervals and several other aspects of the agency's statistical analysis.

*a.* Choice of studies. At the time EPA released its FRA, the scientific literature contained thirty-three studies of nonsmoking female spouses (two of which had not yet been completed and one of which was available only in preliminary form), twelve studies of nonsmoking females in workplaces where smoking was allowed, and thirteen studies of females exposed to ETS during childhood. EPA pooled the data reported in thirty completed and one partially completed spousal studies into a single meta-analysis. The industry argued that EPA did not adequately explain why it included the thirty-one spouse studies in its meta-analysis, while excluding the two incomplete studies and all of the childhood and workplace studies.<sup>437</sup> The court agreed with the industry.<sup>438</sup>

EPA had concluded that the data from the excluded studies were generally less reliable because they contained less exposure information. Without citing or quoting from the record, the court found that "EPA's three citations to the record do not support this assertion."<sup>439</sup> If the purpose of a meta-analysis is to combine data from studies involving similarly exposed individuals, however, it would be sensible to exclude from any data from studies containing less exposure information. But to a technically unsophisticated judge unfamiliar with the context, EPA's terse explanation appeared conclusory and wanting. Apparently, EPA had the high burden of explaining every step it took in assembling and analyzing supporting data in sufficient detail to persuade a skeptical judge.<sup>440</sup>

Another reason cited by EPA for excluding the workplace and childhood studies from the metaanalysis was the presence of confounding factors in those studies that rendered them less reliable. The court found that this rationale also lacked record support. *Id.* at 459. Indeed, the court found that the

<sup>437.</sup> *Id.* at 459.

<sup>438.</sup> Id. at 459-60.

<sup>439.</sup> Id. at 459.

<sup>440.</sup> EPA concluded that the workplace studies should be excluded from the meta-analysis because only two of those studies made any attempt to classify subjects by ETS exposure. *Id.* As the court read EPA's risk assessment, this explanation appeared to be "targeted only at workplace data contained within the spousal smoking studies and does not address the Agency's decision to disregard workplace and childhood exposure data reported outside spousal studies." *Id.* at 460. Since the court did not quote the language in the risk assessment that it relied upon in drawing this conclusion, it is difficult to evaluate whether the court's conclusion is correct. The court made no further attempt to explain its conclusion that EPA's otherwise reasonable explanation was limited to workplace data in the spousal studies. Apparently, the court did not hold itself to the same obligation as the agency to explain its conclusions.

The court faulted in particular EPA's decision to exclude the two most recent spousal studies from the meta-analysis. At the time it prepared the report, EPA stated it did not consider the two studies in its meta-analysis because they were submitted after the comment period closed, and the agency had to draw the line somewhere; otherwise it would never be able to complete the required report. The agency gave three reasons for its apparently inconsistent inclusion of data from one of the three unpublished studies: 1) interim results of that study had been published, 2) it was the largest study of its kind, and 3) its methodology was superior to that of any of the other studies.

To the court, however, the record did not support the agency's seemingly plausible distinction between the included study and the two excluded, incomplete studies.<sup>441</sup> The court did not accept EPA's conclusion that the included study employed a superior methodology because the administrative record did not include a full discussion of the criteria by which EPA determined such superiority. The court also noted that "[e]ven if EPA provided criteria, comparison would not be possible since EPA provides no discussion on the two U.S. spousal studies excluded."<sup>442</sup> The court might have concluded that the agency should have waited for all three studies to be published, but EPA did not know at the time whether they would be completed within a reasonable period.

*b.* The agency's use of statistics. Although demurring that it was "neither necessary or desirable to delve further into EPA's epidemiological web," the court nevertheless addressed two additional industry criticisms of the EPA epidemiologists' statistical meta-analysis techniques.<sup>443</sup> First, the court restated the industry's allegation that EPA used a 90% confidence interval in its meta-

Advisory Committee Report cited by EPA supported the opposite conclusion. *Id.* Once again the court offered only a single conclusory statement to support its assessment of EPA's reasoning process. *Id.* 

Finally, in rejecting the childhood studies, EPA cited the fact that the studies were heavily dependent upon childhood memories to estimate childhood exposures. *Id.* at 460. Although EPA explicitly stated its conclusion that "recall of ETS exposure in childhood is more difficult than recall of such exposure in adulthood," and cited a published study in support of this conclusion, the court did not view this as sufficiently supporting EPA's decision to exclude the childhood data from its meta-analysis. *Id.* Again the court offered no explanation for its conclusion.

<sup>441.</sup> The court further found that the decision to exclude the two recent studies conflicted with the Radon Act's requirement that "EPA's [indoor air risk assessment] program shall 'gather data and information on *all aspects* of indoor air quality." 4 F. Supp. 2d at 460 (quoting Radon Act § 403(a)(1)). The quoted language from the statute requires the agency to consider all aspects of indoor air quality. Aspects of indoor air quality would presumably include the chemical composition of various indoor air environments, ventilation and air flow considerations, exposure patterns, and the toxicity of various indoor air pollutants. The statute plainly did not require the agency to consider all available *data* on all aspects of indoor air pollution, whether or not the relevant studies were completed and had survived peer review and were published in a scientific journal. The statute did not preclude EPA from considering unpublished data, but it certainly did not require it either. The court's one-sentence explanation was as follows: "In conducting a risk assessment under the Act, EPA deliberately refused to assess information on all aspects of indoor air quality." *Id.* The court neither explained how it interpreted the statute, nor how the statutory language applied to EPA's report. In particular, the court failed to point to a single aspect of indoor air quality that EPA deliberately refused to assess.

<sup>442.</sup> *Id.* at 460. The court did not address the other two reasons for including the Fontham study. 443. *Id.* at 460-61.

analysis, rather than the 95% interval deemed appropriate by the industry and used in the DRA, "to enhance the likelihood that its meta-analysis would appear statistically significant."<sup>444</sup> The court then noted EPA's explanation that using the 90% interval avoided what would have been an apparent statistical discrepancy.<sup>445</sup>

Second, the court noted the industry's complaint that EPA classified ETS as a Group A carcinogen, in large part based on a relative risk of only 1.19, without adequately explaining why the agency required every other Group A carcinogen to exhibit a much higher relative risk, or why EPA recently found relative risks of 2.6 and 3.0 insufficient to classify other such agents as Group A carcinogens. In the industry's view, EPA had singled out ETS for Group A ranking. To this contention, EPA had responded that the data on ETS differed from the data on some other chemicals in both the number of studies showing a positive correlation between exposure and cancer and the strength of the correlation that had been shown.<sup>446</sup>

The court concluded that the studies relied upon by EPA would not yield a statistically significant correlation between ETS and cancer if standard statistical methodologies were employed.<sup>447</sup> The court did not identify those methodologies it deemed "standard," nor did it explain why it was inappropriate to employ a 90% confidence interval. Indeed, it is not at all clear from the opinion that the court understood the purpose of confidence intervals and levels of statistical significance in epidemiological analysis. Once again, the court put the burden on the agency to educate and explain why it employed the methodologies that it did.

The court was also troubled that answers to many of the industry's concerns came from the agency's lawyers rather than the FRA itself. The court perceived that the agency was changing its explanation mid-stream and therefore had a heightened duty to explain itself.<sup>448</sup> But the court gave no reasons for

<sup>444.</sup> *Id.* at 461. One member of the SAB committee had raised the same complaint. *Id.* (quoting Geoffrey Kabat, Comments on EPA's Draft Report: "Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders" (July 28, 1992)). The industry also noted that previous ETS studies by the National Academy of Sciences, the International Agency for Research on Cancer, and the Surgeon General employed a 95% confidence interval. *Id.* 

<sup>445.</sup> *Id.* at 461. EPA's concern was that "use of the 95 percent confidence interval with the onetailed test... would have produced an apparent discrepancy: study results that were statistically significant using the standard p-value of .05 might nevertheless have a 95 percent confidence interval that included a relative risk of 1." *Id.* (quoting Conformed Memorandum Supporting EPA's Cross Motion for Summary Judgment at 96, 4 F. Supp. 2d 435.).

<sup>446. 4</sup> F. Supp. 2d at 462.

<sup>447.</sup> *Id.* This was because "[a]nalysis conducted with a .05 significance level and 95% confidence level included relative risks of 1."

<sup>448.</sup> The court cited several cases to support its conclusion that the agency record must support its action and that agencies have a heightened duty to explain changes in methodologies. *Id.* (citing Motor Vehicle Mfrs. Ass'n, Inc. v. EPA, 768 F.2d 385, 399 (D.C. Cir. 1985); W. States Petroleum Ass'n v. EPA, 87 F.3d 280, 284 (9th Cir. 1996); Natural Res. Def. Council, Inc. v. EPA, 859 F.2d 156, 205-11 (D.C. Cir. 1988)). All of these cases involved informal rulemaking proceedings in which the outcome was a binding rule enforceable with fines and even criminal sanctions. In each case, the agency's attorneys were heavily involved in drafting the text of the rule, in analyzing the comments on the proposed

believing that EPA's position had changed between the time the FRA was published and the filing of briefs. Not surprisingly, the agency felt obliged to brief the arguments raised by the industry in its voluminous filings. That the agency risk assessors had not anticipated the industry lawyers' subsequent contentions did not suggest a shift in agency position; they were writing for a largely technical audience. The fact that the agency brief addressed specific contentions more thoroughly than the risk assessment itself is easily explained by the highly unusual posture in which the agency found itself when the court denied its motion to dismiss. The court's decision allowing the industry leave to file a lengthy brief filled with flyspeck criticism of a scientific report drafted without the aid of agency attorneys forced the agency to respond in kind.

#### E. Assessment of the Flue-Cured Tobacco Opinion

Although the court "vacated" chapters 1 through 6 and the relevant appendices of the FRA, the consequences of a court's vacating an agency risk assessment are far from clear. The report could not be erased or retroactively deleted from the agency's computers, and the degree of its persuasiveness should not be affected by one judge's assessment of the validity of its scientific conclusions. Should another agency rely upon the report in regulating ETS exposure, it is not at all clear that such reliance would jeopardize the validity of that agency's conclusions. Another reviewing court would not have to defer to the *Flue-Cured Tobacco* court's conclusions regarding the validity of the scientific analyses in the report. Presumably, it would review that agency's action on the basis of the rulemaking record, in light of that agency's explanations.

Nevertheless, the court's rejection of EPA's meta-analysis is a fine example of the corpuscular approach in action. The court placed the burden on EPA to justify every contested detail of the analysis through reference to contemporaneous statements made on an official record compiled during the risk assessment process. The agency had to explain why it included some studies in the meta-analysis and excluded others, and its explanations had to be plausible to the untrained and technically unsophisticated judge. A brief explanation that would have satisfied scientists familiar with the context appeared conclusory to a judge whose education on the relevant issues was presumably limited primarily to the briefs prepared by the parties' lawyers.

The court resolved the issues of scientific validity on the informal, nonlegal record before it, with very little deference to the agency's expertise, and with very little appreciation for the subtleties of the science. In doing so, the court

rule, and in crafting the preambles in which the agency provided its explanation for its rule and cited the evidence from the rulemaking record. The agency action before the court in *Flue-Cured Tobacco* was not a formal rule, but rather a scientific report that could perhaps be cited in future rulemaking endeavors. It was intended to survive the sort of scientific criticism from the scientific community that such reports typically undergo, rather than the sort of legal analysis typically required of agency rules. Unlike the industry, which viewed the proceedings as a legal battle and enlisted the support of dozens of lawyers from the outset, EPA regarded the matter as a scientific one, to be resolved by an advisory committee of scientists, with minimal input from agency attorneys.

limited its attention to briefed arguments and did not take the time to view or perhaps even read the entire risk assessment. The court clearly put the burden on EPA to rebut each industry assertion by citing evidence in the record of sufficient credibility to convince the court that the industry's arguments were misplaced. This legalistic approach prevented the agency from relying on a holistic assessment under a weight-of-the-available-evidence standard illuminated by the current state of scientific knowledge. While lawyers are trained to document assertions with citations to precedent and available evidence, without regard to whether the assertions meet an overall test of scientific plausibility, scientists do not think or act in this way. Reports prepared by the National Research Council do not provide citation to a "scientific record" for every critical fact found and inference drawn. Yet, those reports are often enormously influential in public policymaking.

The court's demand for scientific-record support for various contentions in the agency's brief apparently reflects its misunderstanding of the risk assessment preparation process as a relatively formal administrative proceeding with an identifiable record. Although not required by the Radon Act or the Administrative Procedure Act, EPA employed notice-and-comment rulemaking procedures for the purpose of maximizing public input into the drafting and redrafting process. There was a record of sorts, but it certainly was not prepared with judicial review in mind. The agency prepared a transcript of the SAB proceedings, sworn testimony, and cross-examination, but the SAB procedures did not require mandatory document production. By requiring the agency to cite record support for arguments raised in response to the industry's briefs, the court effectively turned the process ex post facto into a formal administrative proceeding. It is not surprising that EPA failed to satisfy a judge demanding a great deal of record explanation from a record never intended to be scrutinized under judicial review.

The industry's strategy from the outset was to employ legal arguments and analysis to characterize the risk assessment as a legal document and therefore subject to legal challenge. With virtually unlimited resources, the industry's scientific consultants and lawyers spent hundreds of hours poring over the report and the relied-upon studies to find gaps in the agency's explanations. The industry then demanded that the agency rebut its specific challenges, and the court placed that burden upon the agency. The legal strategy was successful in the district court, and if EPA had been authorized to take any action against indoor smoking, it would have been prevented from doing so. Yet the agency had never asserted authority to do anything other than prepare and make available a report on the risks posed by ETS.

One unresolved mystery of *Flue-Cured Tobacco* is why the court devoted the vast bulk of its written opinion to a *Daubert*-like analysis of the industry's challenge to the factual basis of EPA's report. The court's resolution of EPA's failure to appoint a properly representative advisory committee was outcome

determinative. If the court's legal analysis was correct, it was not necessary to devote such considerable attention to the merits of the case.

One explanation is that the court feared EPA would simply march its soldiers down the hill and march them back again without encountering a solid critique of the report's substance. Essentially, EPA would prepare a new report with the same deficiencies—this time by a properly constituted advisory committee—reaching the same conclusion: that ETS is a Group A carcinogen. The court might have felt its own deep foray into the merits in search of record support was necessary to ensure that EPA paid sufficient attention to industry representatives on the new advisory committee.

Alternatively, the court might have been determined to ensure that the ultimate outcome of the process reflected sound science. The industry briefs were highly critical of many aspects of EPA's analysis, and many of the industry critiques appeared plausible to the court. The court might have seized upon those critiques as an opportunity to teach the agency a lesson in how an administrative record should support a scientific conclusion. Scientists draw conclusions based upon the exercise of scientific judgment, without lengthy side-explanations about why they exercised their judgment the way they did. The court, accustomed to abundant legal documentation and explanation, might have found it necessary to give EPA a lesson in how to craft a scientific conclusion with legal-type support. Had the court's holding not been overturned on ripeness grounds, EPA's legal counsel would no doubt have played a much larger role in writing the next ETS report.

Finally, the court might have wanted to limit the practical impact the risk assessment would have on other decision-making entities such as OSHA, local governments, and private employers. A simple remand to run the report through a properly constituted advisory committee would not undermine the substance of the risk assessment in the minds of future decisionmakers, and it would certainly not persuade entities that had already imposed smoking bans to lift them. A judicial opinion attacking the substance and scientific validity of the report, on the other hand, could seriously undermine efforts to ban smoking in public areas. Industry documents never intended for public review reveal beyond cavil that the industry's strategy from the outset was to bolster its political efforts, blunting the force of anti-smoking efforts across the United States through judicial challenge. The court delivered an opinion in line with the industry's political needs. Whether this alignment was by design or merely the unintended consequence of an exceedingly successful strategy is a question only the judge can answer.

#### IV

#### THE DANGERS OF DAUBERTIZING JUDICIAL REVIEW OF RISK ASSESSMENT

In the past, courts have resisted efforts by the regulated community to Daubertize judicial review of risk assessment. For example, the D.C. Circuit rejected the industry's invitation to adopt a *Daubert*-like standard of in reviewing OSHA's standard for worker exposure to ethylene oxide (EtO) under the substantial evidence test. The court observed of the industry's arguments:

[The industry] attacks each piece of evidence, suggesting that no individual piece proves a relationship between EtO exposure and various adverse health effects. This approach disregards the marginal contribution that each piece of evidence makes to the total picture. While some of OSHA's evidence suffers from shortcomings, such incomplete proof is inevitable when the Agency regulates on the frontiers of scientific knowledge... OSHA need not "prove" its assertions in the manner AEOU demands .... Rather, OSHA need only gather evidence from which it can reasonably draw the conclusion it has reached. Our function ... is only to search for substantial evidence, not proof positive.<sup>449</sup>

Similarly, in reviewing EPA's decision to phase tetraethyl lead out of gasoline, the D.C. Circuit stated:

Contrary to the apparent suggestion of some of the petitioners, we need not seek a single dispositive study that fully supports the Administrator's determination. Science does not work that way; nor, for that matter, does adjudicatory factfinding. Rather, the Administrator's decision may be fully supportable if it is based, as it is, on the inconclusive but suggestive results of numerous studies. By its nature, scientific evidence is cumulative: the more supporting, albeit inconclusive, evidence available, the more likely the accuracy of the conclusion... Thus, after considering the inferences that can be drawn from the studies supporting the Administrator, and those opposing him, we must decide whether the cumulative effect of all this evidence, and not the effect of any single bit of it, presents a rational basis for the ... regulations.<sup>450</sup>

More recently, in upholding EPA's revisions to the ozone and fine particulate national ambient air quality standards, the court reaffirmed that:

It is not our function to resolve disagreement among the experts or to judge the merits of competing expert views. Our task is the limited one of ascertaining that the choices made by the [EPA] Administrator were reasonable and supported by the record. That the evidence in the record may also support other conclusions, even those that are inconsistent with the Administrator's, does not prevent us from concluding that [her] decisions were rational and supported by the record.<sup>451</sup>

Thus far, the courts have sensibly refrained from acting as gatekeeper with respect to the scientific information agencies must rely upon in preparing risk assessments. This judicial reticence is sensible for several reasons.

First, the courts have played a prominent role in the ossification of informal rulemaking and the consequent inability of regulatory agencies to implement their statutory missions. One partial solution to the ossification problem is for the courts to back off.<sup>452</sup> To reduce judicial scrutiny, a "pass/fail professor" test should replace the hard-look doctrine.<sup>453</sup> Daubertizing judicial review of risk assessment would be a move in precisely the opposite direction.

<sup>449.</sup> Pub. Citizen Health Research Group v. Tyson, 796 F.2d 1479, 1495 (D.C. Cir. 1986).

<sup>450.</sup> Ethyl Corp. v. EPA, 541 F.2d 1, 37-38 (D.C. Cir. 1976) (en banc) (footnote omitted).

<sup>451.</sup> Am. Trucking Ass'ns v. EPA, 283 F.3d 355, 362 (D.C. Cir. 2002) (quoting Lead Indus. Ass'n, Inc. v. EPA, 647 F.2d 1130, 1160 (D.C. Cir. 1980).

<sup>452.</sup> Thomas O. McGarity, *Some Thoughts on "Deossifying" the Rulemaking Process*, 41 DUKE L.J. 1385, 1453 (1992) (recommending that the courts replace the "hard look" doctrine with "a more deferential image").

<sup>453.</sup> Id. The "pass/fail professor" test is described as follows:

Second, judges do not always have a good sense for what is relevant in complex risk assessments. The essence of the blunderbuss attack strategy against an agency risk assessment is the search for any inconsistency or instance in which the agency did not analyze a particular matter in sufficient depth. The petitioners' arguments, which are the primary indicators of the importance of particular issues, tend to elevate trivial issues to the level of importance required to convince the court that the error, failure, or omission warrants remand. Not surprisingly, many reviewing judges demonstrate a "remarkable instinct for the capillary" in reviewing agency rules.<sup>454</sup> Unaware of which issue might ultimately doom a rulemaking initiative, agencies will be compelled to over-analyze every issue, no matter how trivial, wasting scarce analytical resources responding to minutiae. Under regulatory *Daubert*, the blunderbuss attack stands a good chance of producing the desired result—a malfunctioning regulatory process.<sup>455</sup>

Third, judges who come to the task with an anti-government ideological perspective are not always as concerned with the quality of agency decisions as they are with ensuring that agencies do not encroach too deeply upon private markets.<sup>456</sup> Assigning a gatekeeper role to the courts under a regulatory-*Daubert* regime would provide an opportunity for judges intent on reducing the federal government's role in commercial activities to force those agencies to become more timid in carrying out their statutory missions. Although it is unclear whether Daubertizing judicial review will enhance the quality of agency decision-making, it almost certainly will result in regulations that are less

A better metaphor may be that of the "pass-fail prof" who must determine whether a research paper on a topic with which he is vaguely familiar meets the minimum standards for passable work. His disagreement with the paper's conclusions will certainly not cause him to flunk the student. Even a poor analysis will not cause the paper to fail, if it is at least plausible. A check of the citations may reveal that the student could have found more sources or that he may have mischaracterized one of the cited sources, and still the paper will pass. Only where there is an inexcusable gap in the analysis, an obvious misquote, or evidence of intellectual dishonesty, will the pass-fail prof give the student an "F" and order the student to try again.

Id.

<sup>454.</sup> Richard J. Pierce, Jr., Unruly Judicial Review of Rulemaking, 5 NAT. RES. & ENV., Fall 1990, at 23, 24.

<sup>455.</sup> For example, common-law courts applying *Daubert* principles in common-law torts cases routinely exclude testimony based upon meta-analysis of epidemiological data as scientifically unreliable, despite the fact that epidemiologists routinely employ meta-analysis to enhance their understanding of epidemiological data. *See* Beecher-Monas, *supra* note 37, at 1072 (concluding that "the courts' wholesale rejection of meta-analysis has little scientific justification").

<sup>456.</sup> See Gulf S. Insulation v. Consumer Prod. Safety Comm'n, 701 F.2d 1137 (5th Cir. 1983); see also DEVRA LEE DAVIS, THE "SHOTGUN WEDDING" OF SCIENCE AND LAW: RISK ASSESSMENT AND JUDICIAL REVIEW 67, 85 (1985) ("The decision stands simply as a remarkable judicial probe of an agency's record on a narrow question."); Nicholas A. Ashford et al., A Hard Look at Federal Regulation of Formaldehyde: A Departure From Reasoned Decisionmaking, 7 HARV. ENVTL. L. REV. 297, 368 (1983) ("[W]e find the Fifth Circuit's analysis to be unpersuasive in its evaluation of CPSC's cancer risk assessment for formaldehyde."); Howard A. Latin, Good Science, Bad Regulation, and Toxic Risk Assessment, 5 YALE J. ON REG. 89, 131 (1989) ("The court's opinion reflects ... a fundamental misunderstanding of the limited evidence on which most risk assessments of carcinogens are based."); Richard A. Merrill, The Legal System's Response to Scientific Uncertainty: The Role of Judicial Review, 4 FUNDAMENTAL & APPLIED TOXICOLOGY S418, S425 (1984) ("The opinion's close scrutiny of an exercise that is fraught with uncertainty, but yet promises improvement in regulation of health hazards, is disconcerting.").

burdensome to regulatees. This is precisely the outcome that most regulatees, and many judges, desire.

Fourth, Daubertizing judicial review of agency risk assessment will pervert the process of health and environmental risk assessment by encouraging lawyerdominated attempts to bend science to the will of regulated industry. From the moment the tobacco industry learned that an epidemiological study would be published suggesting an association between exposure to ETS and lung cancer, the industry and its lawyers launched a crusade to discredit that study and subsequent studies. Industry consultants were hired to flood the publishing journal with letters critiquing the study. Public relations consultants filled the media with attacks on the studies and suggestions that the question of the ETS health risk was still very much undecided. Industry lawyers and sympathetic politicians attempted to manipulate the composition of the agency's advisory committee, and the industry deluged the agency in a flood of industry-funded comments and criticisms of the agency's early drafts. All of this was undertaken with the expectation that the agency would ultimately retreat and write a more equivocal document, but it was also done with an eye toward the litigation that would follow if the agency did not.

Finally, and most importantly, by encouraging such blunderbuss attacks, regulatory-*Daubert* judicial review will ultimately distort the science underlying agency risk assessment. Conversely, a weight-of-the-evidence approach makes good sense as a scientific matter. No study is perfect, and scientists are forced to make assessments about how the universe works on the basis of incomplete scientific information. Risk assessments are necessarily tentative and can frequently be stated with greater confidence as additional studies help strengthen the underlying scientific foundation. If agency risk assessments are to be based upon science, agency risk assessors must be permitted, perhaps with the help of qualified experts, to look at the entirety of the scientific database. Their analyses should not be confined to those studies likely to be deemed scientifically reliable by a judge, at the end of an adversarial proceeding.

#### V

#### CONCLUSION

The predictable consequence of Daubertizing judicial review of agency risk assessment will be an overall reduction in the protections government provides to those at risk. At the very least, more stringent judicial review will result in more remands. If agencies react as regulatory-*Daubert* advocates hope they will, risk assessments will contain better supported and more cautious conclusions. To the extent agencies in turn rely upon risk assessments to support regulatory actions, more cautious risk assessments will yield less stringent regulations. Thus, while Daubertizing judicial review might or might not produce more scientifically sound agency risk assessments, it will almost certainly yield fewer and less stringent regulations. Like the regulatory reformers of the 104th Congress, the not-so-hidden agenda of regulatory-*Daubert* advocates is most likely regulatory relief. The courts should resist the invitation to play such an overtly political role in the way society addresses health and environmental risk.