

Second-hand Smoke and Cancer

The Research Evidence

JOHN C. LUIK

Of all the questions that might be asked about the problem of environmental tobacco smoke (ETS)—second-hand smoke or passive smoke, as it is called—perhaps the most interesting are these two: since science does not establish that ETS is a risk for lung cancer in non-smokers,

- (1) why does a large majority of the public believe that it is such a risk and
- (2) why do governments, not only in North America, but around the world, regulate public smoking as if it put non-smokers at risk of lung cancer?

The answer to these questions is to be found in the actions of the United States Environmental Protection Agency (EPA) and the anti-smoking movement, both of whom have manufactured a risk and orchestrated a health scare through failing to provide truthful answers to the central questions that should guide public policy about the risks of ETS or indeed about any other risk, namely:

- (1) does second-hand smoke put non-smokers at risk of lung cancer and
- (2) if it does, is the risk substantial enough to worry about?

I want to argue that second-hand smoke provides a splendid example of junk science producing junk public policy.

In order to examine how the EPA and the anti-smoking movement have manufactured the ETS lung-cancer risk, I want to look at two case studies, first, the court case in which the EPA's classification of ETS as a human carcinogen was overturned and, second, the 1998 study by the International Agency for Research on Cancer (IARC) that failed to find a statistically significant link between lung cancer and second-hand smoke. In the first instance, the public-health community and the anti-smoking movement manufactured a health risk and, in the second instance, they attempted to discredit their own scientific study when it failed to support their manufactured risk.

Both of these cases, the EPA court case and the reaction of the anti-smoking and health community to the IARC's ETS study are interesting because they reveal the same key characteristics of junk science—the misrepresentation of scientific findings, the misrepresentation of scientific procedure, and the desire, at all costs, to suppress dissent in the service of junk policy. Let us begin with the EPA court case.

The EPA in court: the Osteen decision

The decision: an analysis

On the face of it, the decision by Judge W.L. Osteen appears to be simply another piece of the seemingly unending stream of tobacco litigation: *Flue-Cured Tobacco Cooperative Stabilization Corporation et al., Plaintiffs, v. United States Environmental Protection Agency, Defendant*. The language is difficult, the arguments complex and technical, and the issue itself appears completely disconnected from the lives and concerns of ordinary citizens. But, behind Judge Osteen's carefully measured words, behind even the specific controversy that the judgment addresses are issues of enormous significance to every citizen of a democratic society who relies on his government to tell him the truth. For the Osteen decision at its core is about truth: it is about how the government uses science to determine whether something constitutes a risk to our health; it is about how the scientific procedures for finding truth and the administrative processes for disseminating truth can be corrupted; and it is about the consequences for public policy of institutionalizing such corrupt science. This analysis begins with a discussion of the concept of corrupt science, then turns to the regulatory processes addressed by the decision, and finally examines the substantive scientific issues of the decision.

Corrupt science

Inasmuch as we wish to argue that the Osteen decision supports the characterization of the EPA's ETS procedure and science as corrupt sci-

ence, it is important to be clear at the outset about what constitutes corrupt science. By corrupt science we mean bogus science, science that knows that its data misrepresent reality and its procedures are deviant but that nonetheless attempts to pass itself off as genuine science. It is science that has an institutionalized motivation and justification for allowing ends extrinsic to science to determine the findings of science, for allowing science to be subject to an agenda not its own, for allowing science to tell lies with clear conscience. It is essentially science that wishes to claim the public-policy advantages of genuine science without conforming to the scientific procedure or doing the work of real science.

There are at least four characteristics of corrupt science. First, corrupt science is science that moves not from hypothesis and data to conclusion but instead from a mandated and acceptable conclusion to selected data back to the mandated and acceptable conclusion. It is science that starts with a conclusion, indeed, starts with a mandated policy and sees its job as that of finding and presenting only that evidence that is considered supportive of that conclusion. That is to say, it is science that fundamentally distorts the scientific procedure through using selected data to reach the “right” conclusion, a conclusion that by the very nature of the data necessarily misrepresents reality.

Second, corrupt science misrepresents the nature of what it seeks to explain. Rather than acknowledging alternative evidence or problems with its evidence that would cast doubt on its conclusions, and rather than admitting the complexity of the issue under review and the limits of the evidence, corrupt science presents what is at best a carefully chosen partial truth as the whole truth necessary for public policy. In effect, public policy is manipulated into reaching certain conclusions on the basis of data that has been fabricated, falsified, misrepresented, or massaged so as to speak in a fashion that is fundamentally at odds with the way things really are. Corrupt science in this sense adopts, according to Teich & Frankel, “the scientific counterparts of what lawyers call ‘sharp practices’ . . . incomplete citation of previously published work; bias in peer review of . . . manuscripts; or skewed selection of data to hide or disguise observations that do not fit the author’s conclusions” (1992: 4).

Third, corrupt science not only misrepresents reality but also misrepresents its own procedures in arriving at its conclusions. Instead of acknowledging the selectivity of its procedures and the official desire for demonstrating predetermined conclusions, it invests both its procedures and its conclusions with a mantle of indubitability. It hides, as it were, behind what both scientists and the public believe scientific procedure to be and, in doing so, it builds an aura of respectability

around a decidedly disrespectful procedure. The results appear to be reliable because the procedure appears to be objective, open, and candid—in short, scientific. The selective, the arbitrary, the irrational, and the contrived appear to be certifiably absent since the procedure is “scientific.” Substance and procedure are thus mutually supporting and, taken together, the scientific findings that result from the scientific procedure present a formidable barrier to public policy dissent.

Fourth, whereas legitimate science creates a climate in which debate and dissent is welcome, in which disagreement is dealt with on the basis of the quality of its evidence and argument and in which *ad hominem* argument is considered inappropriate, corrupt science seeks to create formidable institutional barriers to dissent through excluding dissenters from the process of review, characterizing dissent as working against the public interest, and contriving to silence dissent not by challenging its scientific merits but by questioning its character and motivation.

These four characteristics of corrupt science manifest themselves in a variety of ways which include: claiming that a statistical association is a causal relationship; a highly selective use of data; fabrication of data; falsification of data; misrepresentation of data; selective citation and referencing; claiming that a risk exists regardless of exposure level; claiming that a large number of statistically non-significant studies constitute a significant evidentiary trend; claiming that a series of inconclusive or weak studies justify a strong conclusion; relaxing generally accepted statistical conventions without compelling reasons; being unwilling to consider non-conforming data seriously; implying that the status of an authority justifies its evidence independently of the strength of that evidence; suggesting that weak evidence warrants decisive regulatory action; claiming that a finding based on one population is necessarily true of a different population; suggesting that certain risks are exempt from the normal regulatory and public-policy process; and conjoining the roles of the public-policy advocate and scientist.

The Issues

The issues in dispute in the litigation before Judge Osteen centred on the EPA's 1992 report, *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders*, in which the EPA, using the authority provided to it under the Radon Gas and Indoor Air Quality Research Act of 1986, examined the health effects of ETS and classified it as a Group A carcinogen (USEPA 1992). Classification as a group A carcinogen meant that the scientific evidence supported the conclusion that ETS causes lung cancer in human beings. The plaintiffs argued that both the procedure used by the EPA to examine the health effects of ETS and its finding that ETS causes cancer in human beings were flawed. Specifi-

cally, the plaintiffs alleged that: the EPA exceeded its statutory authority under the Radon Research Act; the EPA failed to follow the Radon Research Act's procedural requirements; the EPA violated established administrative law procedures by reaching a conclusion about the health effects of ETS before conducting its scientific examination of the evidence; and the EPA's classification of ETS as a Group A carcinogen was not the product of reasoned science and decision-making.

Issues about the procedure

Questions about the procedure involved at least three separate issues: the extent of the EPA's authority under the Radon Research Act; the nature of the Radon Research Act's procedural requirements and the EPA's conformity to these requirements; and the question of whether the EPA reached a decision about the health effects of ETS before beginning the scientific procedure, and whether such a decision violated administrative law procedures.

The EPA's authority under the Radon Research Act

The language of the Radon Research Act appears, at least within the statutory context, to be relatively straightforward. Yet, as Judge Osteen observed, the parties reading the same plain language of the Act, came "to opposite conclusions" as to its meaning (Osteen 1998: 8). The Act authorizes the EPA to establish a research program on radon and indoor air quality that has three components: research and development concerning the identification, characterization, and monitoring of the sources and levels of indoor air pollution; research relating to the effects of indoor air pollution and radon on human health, and dissemination of information to assure the public availability of this research (Osteen 1998: 4). The Act does not authorize the EPA to establish any regulatory program based on the research conducted under the Act. In order to assist it in discharging its responsibilities under the Act, the EPA is required to create two advisory groups, one of which is to be made up of representatives of federal agencies that are concerned with indoor air quality, and the other of which is to be made up of "individuals representing the States, the scientific community, industry, and public interest organizations" (Osteen 1998: 5). As the court notes, the purpose of the Act was to have the EPA provide United States Congress and the public with clear, objective information about indoor air quality and the effects of indoor air quality on human health (Osteen 1998: 10). The Act was not intended to provide regulatory authority to the EPA: the EPA's role was neither that of advocate of certain positions nor of public-policy maker. Rather, the EPA was to create a research program that would result in clear, neutral information about indoor air quality.

The plaintiffs did not dispute the right of the EPA to establish a research program on the possible effects of ETS on indoor air quality. They did, however, contest its authority to engage in a carcinogen risk assessment and classification on the grounds that these are regulatory actions that go beyond the authority of the Radon Research Act. The court, however failed to accept this line of argument.

The court disagrees with Plaintiffs' argument that risk assessment constitutes a regulatory activity and is thus prohibited under the Radon Research Act. Both the NRC's [National Research Council] Redbook and EPA's Risk Assessment Guidelines identify regulatory activity as being comprised of two elements: risk assessment and risk management. Prohibition of certain conduct does not include prohibition of lesser included activities. (Osteen 1998: 15)

Moreover, the also court noted that the Radon Research Act also contains specific directives to the EPA that warrant its carcinogenic classifications.

First, Congress required EPA to characterize sources of indoor air pollution . . . Since they emit gasses and particulates, burning cigarettes are a source of indoor air pollution. By determining whether these emissions cause cancer in people exposed to burning cigarettes, EPA is characterizing a source of indoor air pollution. Second, Congress required the EPA to determine indoor pollutant effects on health . . . In determining whether health is affected by a pollutant, the researcher must identify whether a causal relationship exists between the pollutant and deteriorating health. Put simply, the researcher must determine how, if at all, a pollutant affects health. Once a researcher has identified how a pollutant harms human health, the risk is most often identified. This is especially true regarding carcinogens. The Radon Research Act's general language authorizing EPA to characterize sources of pollutants, research effects on health, and disseminate the findings encompasses classifying pollutants based on their effects. (Osteen 1998: 12-13)

Thus, the court found that the Radon Act, by providing authority for characterizations of indoor pollutants and their possible health consequences, provides authority for the EPA to engage in risk assessment. As the Court noted, "the Act requires more of the EPA than merely describing effects. Congress intended EPA to disseminate findings, or conclusions, based upon the information researched and

gathered. Utilizing descriptions of health effects to make findings is risk assessment” (Osteen 1998: 12). While the EPA is not provided with authority to engage in risk management, it is allowed to conduct risk assessments.

The Radon Research Act’s procedural requirements and the EPA

The Radon Research Act requires that the EPA create two advisory groups to assist it in its research and other statutory activities. One of these groups is to be “comprised of individuals representing the States, the scientific community, industry, and public interest organizations” (Osteen 1998: 5). The plaintiffs alleged that the EPA failed to comply with this requirement of the law. The EPA replied that it met its procedural obligations by consulting with its own Science Advisory Board.

Before considering the court’s analysis of this issue, it is important to be clear precisely what is at stake here. In one sense, this is a narrow legal argument about the conditions that satisfy a procedural requirement of the law. But, in another sense there is something far more important at stake. The procedural requirements for risk research and assessment are not incidental or peripheral to the research or the risk classifications that result from such a research procedure. Indeed, they are integral inasmuch as adherence to accepted scientific procedures and standards serves to preserve the integrity of the research findings.

In framing the legislation, the United States Congress understood that if science is to preserve its transparent and objective role in the public-policy process it must follow a procedure that was itself transparent and objective. In effect, because of the danger of the scientific procedure being subverted by a non-scientific agenda, the scientific procedure needs to occur in an arena in which all voices are heard and no position is excluded *a priori*. The Congress’ mechanism for ensuring the legitimacy of the scientific procedure was to have that procedure occur within, and be accountable to, a representative body that included all, not just some, of those likely to be effected by the research and any consequent regulation. As Judge Osteen, quoting the Court of Appeals for the District of Columbia, noted:

The most important aspect is the requirement of consultation with knowledgeable representatives of federal and state government, industry and labor. This goes far beyond the usual requirements of public notice and opportunity for comment set forth in the Administrative Procedure Act, and represents the Congressional answer to the fears expressed by industry and labor of the prospect of unchecked federal administrative discretion in the

field. These rather unique requirements of the Act are an important part of the ultimate legislative compromise, and must be given their due weight. (Osteen 1998: 32)

The representative advisory group serves, then, as an important check on the corruption of science in that, by bringing everyone to the table and then structuring its research program and determining its risk assessment on the record, the advisory group dramatically reduces the opportunities for manipulation of procedure, policy determination *a priori*, data misrepresentation and evidentiary selectivity. The failure to comply with the Radon Act's procedural requirements is not simply a legal quibble; failure to comply goes to the heart of the question of whether the EPA's research and risk assessment on ETS is an instance of corrupt science.

The EPA did not deny that it failed to create the required advisory group and Judge Osteen noted that the "EPA's procedural failure constitutes a violation of the law" (Osteen 1998: 37). The crucial question is, why would the agency act in a way so clearly in violation of the law? We would suggest that the EPA failed to create a representative advisory group because such a group, first, would have objected to and made transparent the improper research and risk-assessment procedure to which the EPA was committed; and second, would have prevented the ETS carcinogen classification. There are three pieces of evidence that support this conclusion.

First, the EPA obviously understood how the advisory group would operate and what was at stake in forming an advisory group. At a minimum, as Judge Osteen noted, an advisory group would have ensured that the research and risk-assessment procedures were on the record, preventing the gaps in the record that raise what the judge calls the "ugly possibility" of inappropriate methodology and selective evidence. By failing to create an advisory group, the EPA allowed itself to work in the dark—to work, both literally and figuratively, off the record.

But, by failing to create an advisory group, the EPA also ensured the exclusion from its research and risk-assessment procedures of critics of its ETS position, which in this case included many others in addition to the tobacco industry. In effect, there would be no contradictory voices, no disturbing dissent to the predetermined scientific procedure. The Tobacco Institute's attorney, John Rupp, complained to assistant administrator William Rosenberg about the procedural unfairness of ignoring the industry's role in the research and risk assessment procedure, noting that "at no time has there been an opportunity for a scientific discussion of fundamental issues regarding ETS" (Kluger 1996: 693). But, of course, the corruption of the procedure was deliberate: the EPA had no interest in having a "scientific discussion of

fundamental issues” with anyone who might provide compelling and credible evidence against its pre-determined position.

Second, the EPA, realizing that its manipulation of the research and risk-assessment procedure looked like an attempt to justify a pre-determined position, attempted to mislead the court about the steps it did take to ensure industry representation. The EPA told the court that it “formed an advisory group within the [Science Advisory Board] which included representatives of all the statutorily identified constituencies” (Osteen 1998: 21). This group, the Indoor Air Quality/Total Human Exposure Committee (IAQC), according to the EPA, contained three members (out of nine) who represented the tobacco industry. But the court concluded that this was not in fact true.

EPA claims that one of the listed members, Dr. Woods, represented industry. However, this is not possible since Dr. Woods left industry for employment with a university almost a year before the first draft of the ETS Risk Assessment was made available for review by IAQC . . . EPA further asserts that two other individuals represented industry. The ETS Risk Assessment IAQC listing does not contain the names of these individuals. The individuals are not listed in the IAQC ETS reviews’ transcripts, nor does EPA assert or direct the court’s attention to evidence that these individuals provided any participation in the ETS Risk Assessment.(Osteen 1998: 27–28)

The EPA’s contention that Dr. Woods represented the tobacco industry, even though he had taken up an academic appointment, is interesting in that it appears to represent a belief that one can never really leave the industry, that even whilst no longer in the industry’s employ one’s views will be the industry’s views. The same peculiar notion underlies the EPA’s further claim to the court that, because certain members of the IAQC were “associated with organizations that had received some industry funding pursuant to contract,” they could be considered industry representatives (Osteen 1998: 28). As the Court observed, this “does not convert these individuals into industry representatives” (Osteen 1998: 28). Moreover, even if the IAQC had functioned in the way the EPA claimed, it would have been, according to the court, a

poor proxy for industry representation. EPA sought parties near the “middle” of the spectrum when establishing SAB [Science Advisory Board] panels and allegedly avoided representation from either end of the spectrum. As a general rule, the tobacco industry occupies that end of the spectrum contesting the carcinogenicity

of ETS and EPA's motives. A committee aspiring to represent the middle of the ETS debate necessarily suppresses the tobacco industry's perspective. Further, industry's ability to submit comments to a "neutral" committee, which itself had access to EPA, is not equivalent to industry access to EPA. (Osteen 1998: 32)

But, of course, the EPA did not wish for the industry to have access for its corruption of the procedure was precisely designed to "necessarily suppress" the industry's perspective and participation in the research and assessment activities. Despite the EPA's claims, the record shows the IAQC did not and, indeed, could not function as the required advisory group since its representation did not include industry and its parties were chosen on the basis on their occupying positions near the middle of the spectrum.

The third and strongest piece of evidence supporting the claim that the EPA failed to create the required advisory group because such a group would have exposed and opposed its fraudulent research and assessment program is the fact that the agency came to the conclusion that ETS was a human carcinogen prior to beginning its research and risk-assessment procedure. Indeed, this is the clearest piece of evidence for the claim that the EPA's entire work with respect to ETS is an instance of corrupt science inasmuch as it reveals both that the EPA moved from policy and risk assessment (ETS causes cancer in humans) to research rather than vice versa, and that the Agency attempted to conceal this. As Judge Osteen noted:

Rather than reach a conclusion after collecting information, researching, and making findings, EPA categorized ETS as a "known cause of cancer" in 1989. EPA, *Indoor Air Facts No. 5 Environmental Tobacco Smoke*, ANR-445 (June 1989) (JA 9, 409–11). EPA's Administrator admitted that EPA "managed to confuse and anger all parties to the smoking ETS debate . . ." EPA Memorandum from William K. Reilly, Administrator, to Secretary Louis W. Sullivan, 2 (July 1991) (JA 6, 754). The Administrator also conceded, "[B]eginning the development of an Agency risk assessment after the commencement or work on the draft policy guide gave the appearance of . . . policy leading science . . .". (Osteen 1998: 88)

Having already reached a conclusion about ETS in the absence of the required research program, the EPA could simply not risk using the legally required advisory group. At the very least, such a group with members representing the tobacco industry would leave a public record of vigorous scientific debate about the risks of ETS to human popula-

tions, a debate that might well make both the public and the scientific community skeptical about the EPA's conclusions. At the most, a duly constituted advisory group might actually be so unconvinced by the agency's conclusions that it would overturn them. As the court observed, there is a clear "logic" to the EPA's pattern of conduct that was driven by the recognition that there was simply too much at stake to risk the possible interference in a pre-determined policy process of an advisory group.

In this case, EPA publicly committed to a conclusion before research had begun; excluded industry by violating the Act's procedural requirements; adjusted established procedure and scientific norms to validate the Agency's public conclusion, and aggressively utilized the Act's authority to disseminate findings to establish a de facto regulatory scheme intended to restrict Plaintiffs' products and influence public opinion. (Osteen 1998: 89–90)

It is simply not credible, then, that the EPA failed to note the significance of its action in failing to create the required advisory group.

The substantive issues

It is possible, of course, that the corruptions of the procedure that the court found, while serious, were nonetheless simply incidental and not material to the EPA's risk assessment. In effect, even though the EPA behaved badly in preventing the research process, the end product of that process—the risk assessment—was nonetheless legitimate. In order to resolve this issue the court needed to determine whether "consultation with the representative group would have likely produced a different result" (Osteen 1998: 38). And, in order to determine this, the court was required to examine the substance of the EPA's risk assessment. In effect, if the plaintiffs' claims about the quality of the EPA's risk assessment are true, namely that the assessment is arbitrary, capricious, and unreasoned, then it follows that the plaintiffs' legally required participation in the research and assessment procedure would have made a substantive difference. As Judge Osteen noted, the first issue

is whether EPA's consulting a representative committee, on which industry's concerns were represented during the research process, likely would have caused EPA to change the conduct or conclusions of its ETS assessment. The key to this determination is whether industry representatives could have presented a meritable criticism and advice. (Osteen 1998: 43)

What, then, of the industry's criticism of the EPA's ETS science? Was it meritable? The credibility of the EPA's risk assessment centres on three types of claims: first, about the biological plausibility of equating Mainstream Smoke (i.e. that inhaled by smokers) with ETS; second, about the epidemiological evidence regarding the health effects of ETS; and third, about the EPA's epidemiological methodology.

EPA's Biological Plausibility Thesis

The EPA's biological plausibility thesis is crucial to its risk assessment since it establishes an indispensable chain of argument. This runs as follows: first, the biological plausibility of equating Mainstream Smoke (MS) with ETS justifies the EPA's *a priori* hypothesis that ETS is a Group A carcinogen; second, this hypothesis justifies the EPA's use of one-tailed significance tests (see Appendix, page 105); and third, the use of one-tailed significance tests leads to the use of a confidence level of 90 percent as opposed to a confidence level of 95 percent. As the court noted, "these issues are more than periphery. If EPA's *a priori* hypothesis fails, EPA has no justification for manipulating the Agency's standard scientific methodology" (Osteen 1998: 65). Thus, if the biological plausibility argument is without merit, the entire risk assessment is seriously imperilled.

The plaintiffs raised three objections against the bioplausibility thesis, saying: "(1) [EPA] ignored Assessment findings about the differences between MS and ETS; (2) EPA ignored evidence rejecting any chemical similarity; and (3) EPA did not define the criteria used to reach conclusions about the similarity/dissimilarity/indeterminacy of MS and ETS" (Osteen 1998: 45). The plaintiffs' claims here were supported to a large extent both by the assessment and by prior EPA risk classifications. For instance, in chapter 4 of its report the EPA noted that "the rapid dilution of both SS [side-stream smoke, i.e. smoke coming from a smouldering cigarette] and exhaled MS into the environment and changing phase distributions of ETS components over time raise some questions about the carcinogenic potential of ETS under actual environmental exposure conditions" (Osteen 1998: 45, quoting USEPA 1992 at 4-29). Again, the assessment record notes that the primary author of chapters 5 and 6, Kenneth Brown, argues that "there are differences between active and passive smoking that may affect carcinogenic risk that are not fully understood." (Osteen 1998: 46-47, quoting Draft Report *Responses to Public Comments etc.*) Clearly the assessment's own authors appear to doubt the bioplausibility thesis. These doubts are also shared by others and the plaintiffs introduced evidence citing scientific literature that also rejected the bioplausibility hypothesis.

Finally, there is an absence of any defined criteria as to how the chemical similarity of MS and ETS was established. This gives rise to the suspicion that the EPA changed its position on the alleged similarity of MS and ETS depending on what sort of argument it was attempting to make. As Judge Osteen noted, “It is striking that MS and ETS were similar only where such a conclusion promoted finding ETS a carcinogen” (Osteen 1998: 61). Indeed, this suspicion is given considerable credence by the fact that in previous risk assessments the “EPA did not classify agents in Group A because they contain the same constituents as other Group A carcinogens” (Osteen 1998: 49).

In response to these arguments, the EPA claimed that the bioplausibility thesis is supported in three ways. First, since active smoking is a cause of lung cancer in humans, it is reasonable to assume that ETS is a cause of lung cancer in humans because ETS is chemically similar to MS. Second, there is evidence that non-smokers who are exposed to ETS absorb and metabolize significant amounts of it. Third, laboratory tests have shown that ETS causes cancer in animals and damages DNA. The EPA also rejected the assertion that it failed to provide criteria for determining the similarity of MS and ETS, arguing that it set out four criteria (Osteen 1998: 51–52, 54).

Judge Osteen found each of these arguments to be unconvincing due to the fact that “there is limited evidence in the record supporting EPA’s final basis for its bioplausibility hypothesis” (Osteen 1998: 57). In other words, whatever the *post hoc* explanations devised for purposes of litigation, the scientific record of the assessment process does not support the EPA’s claims of bioplausibility. Indeed, as the court notes, it is not simply that there is limited evidentiary basis in the record to support the EPA’s thesis—there is also substantial evidence in the assessment record that contradicts the EPA’s plausibility thesis.

For instance, the scientists on the IAQC’s final review panel themselves expressed significant reservations about the similarity of MS and ETS: “The data in Chapter 3 ‘do not . . . adequately support the conclusion that the two are chemically similar . . . [T]he data that are in there, speaking as a chemist, they simply don’t make the case . . . [T]he data . . . simply do not demonstrate that they are similar’” (Osteen 1998: 62).

What was most disturbing to the court was what might be called the convenience factor, the fact that the bioplausibility hypothesis was maintained in the assessment only when it served the purposes of the EPA in finding ETS a carcinogen and was abandoned in other places. The EPA attempted to justify these inconsistencies in the record on the basis of both quantitative and qualitative components of risk assessment but both these were completely rejected by the court: “Neither the Assessment [n]or the administrative record explains why physicochemical

inquiries require a bifurcated analysis instead of a combined analysis as per the Guidelines, or why MS and ETS are similar for purposes of hazard identification, but not for purposes of quantitative risk assessments” (Osteen 1998: 60). Moreover, the claims about ETS causing cancer in laboratory animals did not support the EPA’s hypothesis either, since the “studies detected no evidence of lung cancer . . . and the Assessment does not explain, nor does EPA direct the court to any evidence within the record explaining, how SS [side-stream smoke] condensate demonstrates similarities between MS and ETS” (Osteen 1998: 57–58).

It is not simply the convenience factor, however, that disturbed the court. There was also the issue of circularity. Sensing that the case against ETS could not be sustained on the basis of the bioplausibility thesis, the EPA sought to reinforce the thesis with epidemiological studies, claiming that the epidemiological evidence supported the bioplausibility thesis. This reasoning was patently circular in that the EPA’s “logic” turns on the independent integrity of the bioplausibility argument. As the EPA used the bioplausibility argument to relax the standards of statistical significance for the epidemiological studies, it could hardly then use the contrived significance of those studies to justify bioplausibility. In short, the bioplausibility thesis was being asked to do too much. It could not both justify a manipulation of the epidemiological data and derive its support from that same data.

The court is disturbed that EPA and Kenneth Brown buttress the bioplausibility theory with the epidemiology studies. EPA’s theory must be independently plausible. EPA relied upon similarities between MS and ETS to conclude that it is biologically plausible that ETS causes cancer. EPA terms this theory its “*a priori* hypothesis” in justifying Chapter 5’s methodology. Chapter 5’s methodology allowed EPA to demonstrate a statistically significant association between ETS exposure and lung cancer . . . Chapter 5’s analysis rests on the validity of the biological plausibility theory. It is circular for EPA to now argue that epidemiology studies support the Agency’s *a priori* theory. Without the theory, the studies would likely have done no such thing. (Osteen 1998: 58)

What emerges from the both the assessment record and the litigative record on bioplausibility is a second pattern of corrupt science. Consider the court’s conclusions:

The court is faced with the ugly possibility that EPA adopted a methodology for each chapter, without explanation, based on the outcome sought in that chapter. This possibility is most potent

where EPA rejected MS-ETS similarities to avoid a “cigarette-equivalents” analysis in determining carcinogenicity of ETS exposure. Use of cigarette-equivalents analysis may have lead [sic] to a conclusion that ETS is not a Group A carcinogen . . .

EPA’s assertion that “EPA did explain the numerous criteria it used in assessing similarity” . . . is without merit. EPA merely parrots the findings made in Chapter 3 of the ETS Risk Assessment. The record presents no evidence of EPA establishing similarity criteria before the Assessment . . .

The record does not support EPA’s arguments that EPA took MS-ETS differences into account and, despite them, concluded ETS is a known human carcinogen because non-smokers are exposed to and absorb carcinogens. EPA conceded that dilution, aging and exposure characteristics fundamentally distinguish ETS from mainstream smoke, and “raise . . . questions about the carcinogenic potential of ETS.” . . . The record does not explain how, after raising these questions, EPA could classify ETS a known human carcinogen based on similarities between SS and MS . . .

If confronted by a representative committee that voiced industry concerns, EPA would likely have had to resolve these issues in the record. It is not clear whether EPA could have or can do so. These issues are more than periphery. If EPA’s *a priori* hypothesis fails, EPA has no justification for manipulating the Agency’s standard scientific methodology. (Osteen 1998: 60–65)

What is most striking about the court’s language is the repeated use of phrases like “the record presents no evidence,” the “record does not explain how,” and the “EPA’s assertion is without merit,” all of which point to the unreasoned, unscientific character of the EPA’s bioplausibility hypothesis. Now it might be argued that the court’s language and analysis point to nothing more disturbing than incompetent science, that there is nothing here that rises to the level of corrupt science. While the process of the assessment might be corrupt, the substance of the assessment’s science is merely incompetent, not corrupt.

Though in some senses appealing, this interpretation of the EPA’s science is untenable for three reasons. First, the convenience factor is a clear mark of corrupt science. Rather than taking a consistent position about the alleged MS-ETS similarities, the EPA crafted positions depending on the required outcomes of particular chapter in its report. Rather than basing its MS-ETS equivalency on some chemical basis, the EPA founded it instead on a pre-conceived policy outcome: namely, that ETS causes cancer in humans. Despite the contrived explanations offered to the court, the only way in which the contradictory claims about MS-ETS

similarities make any sense is within the pre-determined position of finding ETS a carcinogen. And this is without question the defining characteristic of corrupt science—mandated conclusion driving scientific explanation. In effect, Judge Osteen had discovered that the bioplausibility hypothesis was a pseudo-scientific front protecting a scientifically unjustified position, hence his reference to the “ugly possibility.”

Second, the circularity of the bioplausibility and epidemiological arguments and EPA’s tenacious defence of their interconnection is unlikely to be the product of mere incompetence. The logical unacceptability of such reasoning is obvious to anyone operating at the EPA’s level of policy and scientific sophistication. The EPA is clearly aware of the fact that it can only sustain its carcinogenicity finding through the bioplausibility thesis; it is clearly aware of the fact that its bioplausibility thesis provides the rationale for manipulating the statistical outcomes of the epidemiological evidence; and it is clearly aware of the fact that it claims the bioplausibility thesis is in turn supported by the manipulated epidemiological evidence. If the argumentative circle were less tight the circularity might be less apparent. But to characterize such openly illogical and manipulative practices as the product of inadvertence or incompetence is to ascribe to the EPA a level of inconceivable methodological schizophrenia.

Third, the consistent willingness of the EPA during the trial to misrepresent its positions, its evidence, and its reasoning on bioplausibility to the court, together with its apparent unconcern with tortured and clearly untenable explanations, distinguishes its actions from the simply incompetent. Even the persistent language of the court (“the record does not explain how,” “the EPA’s assertion is without merit”) indicates a polite disdain for the agency’s contorted defense of its insupportable assertions. To take but one example: despite the agency’s claims that criteria for MS-ETS had been established prior to the assessment, there is no evidence of such a criteria having existed. Indeed, as the court noted, no such criteria were presented at the IAQC final review panel, where the panel’s neutral scientists raised fundamental and unanswered questions about the chemical similarity of MS and ETS. Thus, even allowing for the significant gaps in the assessment record, the record that does exist is at odds with the EPA’s central hypothesis.

A similar instance of significant non-confirming evidence being completely ignored was the review of the EPA’s own Risk Criteria Office, which recommended against the approach taken in the assessment (Osteen 1998: 64). As the court finally concluded, it is apparent that the arguments submitted during litigation do not represent the argumentative process or position developed during the assessment. Rather

they are fundamentally misrepresentations designed to make the unreasonable look reasonable, “*post hoc* rationalizations devised during litigation” (Osteen 1998: 73).

What the evidence shows, therefore, is that the EPA report was the result of corrupt science. Not only were the EPA’s procedures corrupt, its arguments about, and evidence—perhaps, more appropriately, lack of evidence—for, bioplausibility, display a pattern of corruption.

EPA’s epidemiological evidence

The second issue on which the validity of the EPA’s ETS assessment turns is the extent of the epidemiological evidence that the EPA examined. By the time that the EPA risk assessment appeared, there were 58 studies that examined the risks of lung cancer in ETS-exposed populations. Of these, 33 looked at the risk of lung cancer to non-smoking females married to male smokers and, of these 33, the EPA based its assessment on 31 studies that were available at the time that it conducted its second IAQC review. One of the 31 studies was not ready in its complete form so that the agency used interim results only. In order to draw conclusions from all of the studies, the EPA submitted them to meta-analysis.

Given that the EPA based its analysis on only 31 of the available 58 studies, it is worth noting which studies were excluded and why. The studies excluded fell into three groups: 12 studies examined the cancer risks of females exposed to ETS in the workplace; 13 looked at cancer risks of females exposed to ETS during childhood; and two looked at cancer risks of females married to smokers. The EPA is remarkably silent as to why these 27 studies were excluded, the assessment noting only that more were included than excluded.

Now the EPA’s IAQC noted that one of the conditions necessary for meta-analysis is a “precise definition of criteria used to include (or exclude) studies” (Osteen 1998: 67). But, it is clear from the assessment record that the EPA undertook the meta-analysis in the absence of any articulated criteria as to which studies to include. As the IAQC observed, “[s]pecific criteria for including studies was not provided. The importance of this was reinforced at the Committee meeting when a reanalysis was presented on a different set of studies than those in the report. This resulted in a change in the overall risk estimate. Decisions as to study inclusion should be made *prior* to analysis, based on clearly stated criteria” (Osteen 1998: 67, quoting USEPA 1991: 32–33).

The importance of having criteria is thus twofold. On the one hand, it is necessary for the validity of the meta-analysis because it forces a clear examination of the differences and similarities in the data that are being combined. On the other hand, it provides an important

procedural element of transparency by certifying that the database is not biased towards some pre-determined outcome. This last requirement is particularly important in this case, as the EPA had already committed itself to a conclusion about ETS before it began its risk assessment. If the agency cared at all about scientific and policy integrity, it would have taken exceptional care in this phase of its assessment to conform to the procedural requirements outlined as necessary by its own IAQC.

But, it did not. As the plaintiffs noted, this failure to create criteria and the consequent unexplained exclusion of important epidemiological data provides strong evidence of arbitrary, unreasoned decision-making: "Plaintiffs contest that EPA excluded studies and data on workplace and childhood exposure to ETS, as well as the 'two largest and most recent' US spousal smoking studies, because inclusion would have undermined EPA's claim of a causal association between ETS exposure and lung cancer" (Osteen 1998: 68). In an attempt to deny the claim of arbitrariness designed to insure a pre-determined outcome to the assessment, the EPA offered the court five *post hoc* reasons for excluding these 27 studies and including the remaining 31.

First, the data in the childhood and workplace studies were said to be "less extensive and therefore less reliable" (Osteen 1998: 68). The court noted that the EPA's evidence for this claim was unconvincing both on the question of extent and reliability (Osteen 1998: 68–69). Second, the EPA argued that the workplace studies were excluded because of potential "confounders," i.e. methodological errors or problems that undermine a meta-analysis). Again, the court found no support in the record for this claim. Third, the EPA explained that workplace studies were excluded because most did not classify subjects by the amount of their exposure. Here as well, the court noted that this reasoning was not part of the assessment record. Fourth, the EPA claimed that the childhood studies were excluded because they were founded on distant and perhaps unreliable memories and represented a more limited exposure than spousal exposure (Osteen 1998: 70). But, as the court noted, there is nothing in the record to support the claim that "childhood exposure data should be ignored" (Osteen 1998: 70). Again, the record does not reveal that the EPA used reliability of memory of total lifetime exposure as a selection criterion. Indeed, if memory reliability were to be used as a criterion, many of the studies would have to be excluded, as all rely to some extent on recollection.

Fifth, regarding the spousal studies completed after the comment period had passed and the EPA already had a considerable database, the agency justified its use of preliminary data from only one of the three studies (the Fontham study) on the grounds it was the largest Ameri-

can ETS study and used methodology superior to any other study (Osteen 1998: 71). These claims however, were again not supported by the record. As the EPA failed to create criteria by which to select studies, it could hardly claim that the Fontham study's methodology was superior. For, without criteria in which the weight given to methodology is clearly articulated, such a claim appears to be nothing more than another explanation designed purely for the purposes of litigation. Indeed, as Judge Osteen noted, given that there was no record of the methodology employed in the other two studies, it would be impossible even to compare methodologies (Osteen 1998: 71).

This returns us to the critical influence of the EPA's violations of procedure on its substantive conclusions. With the open, representative procedure required by law and a full deliberative record, the EPA would have found it virtually impossible to be arbitrary and, indeed, even to appear arbitrary. Disputes about criteria and study selection against that criteria would be answerable at least in principle. As Judge Osteen noted:

EPA's study selection is disturbing. First, there is evidence in the record supporting the accusation that the EPA "cherry picked" its data. Without criteria for pooling studies into a meta-analysis, the court cannot determine whether the exclusion of studies likely to disprove EPA's *a priori* hypothesis was coincidence or intentional . . .

In making a study choice, consultation with an advisory committee voicing these concerns would have resulted, at a minimum, in a record that explained EPA's selective use of available information. From such a record, a reviewing court could then determine whether EPA "cherry picked" its data, and whether EPA exceeded its statutory authority. (Osteen 1998: 72-73)

Of course, even allowing the EPA the benefit of the doubt about its motives does not clear it of other failures. By excluding nearly half of the available studies, the EPA failed to follow its own risk-assessment guidelines and contravened the Radon Research Act. The Act states that the EPA should gather data and information on all aspects of indoor air quality, while the agency chose selectively to ignore significant amounts of data: "At the outset, the court concluded risk assessments incidental to collecting information and making findings. EPA steps outside the court's analysis when information collection becomes incidental to conducting a risk assessment" (Osteen 1998: 72-73).

In the absence of both a procedure ensuring objectivity and fairness, and criteria for methodological soundness, it is difficult not to conclude that the EPA's insistence on including the Fontham study was

based less on reasoned decision-making than on its desire to support its *a priori* conclusion about ETS. Without the Fontham study, the epidemiological evidence would not have produced the desired conclusion. Further, including all three of the large American studies along with the workplace and childhood exposure studies would have made EPA's carcinogen classification of ETS impossible. Hence, the court's observation that, for the EPA, information collection became incidental to conducting a risk assessment. (Osteen 1998: 72–73).

Is this assessment of the EPA's methods and motivation too harsh? Consider the following scenario. You announce a scientific conclusion prior to examining the scientific evidence supporting that conclusion. Upon examining the scientific evidence, you find that the bulk of the evidence (in this case, epidemiological studies) does not support your conclusion. You now have two options: one, to withdraw or modify your conclusion; two, to adjust the evidence to support your conclusion. You decide to maintain your conclusion. This means that the embarrassing counter-evidence must be dealt with. To do this you create, off the record, two classes of evidence, evidence that is helpful to your conclusion and evidence that is unhelpful to your conclusion. Evidence that is unhelpful to your conclusion is not used; evidence that is helpful becomes the foundation of your case. When asked later to explain why certain pieces of evidence were examined and became the basis of your conclusion, you put forward a series of explanations, although there is no evidence that any of these actually drove your original selection.

We would argue that what emerges from the court record is just this scenario—a predetermined conclusion driving a selective procedure of evidence-gathering, in which the key to selection is not scientific integrity but support for the EPA's pre-determined conclusion. It is this scientifically corrupt procedure in which, as Judge Osteen noted, evidence collection and examination become incidental to truth-finding.

The EPA's Epidemiological Methodology

The third and final issue upon which the validity of the EPA's ETS risk assessment hinges is the agency's epidemiological methodology. The plaintiffs raised seven specific methodological issues, charging that the EPA deviated from accepted scientific procedure and its own risk assessment guidelines in a manner designed to ensure a pre-ordained outcome (Osteen 1998: 73–74). Despite the significant problems already identified with the assessment, however, Judge Osteen thought it unnecessary to delve further into what he calls the EPA's epidemiological web. But, there were two methodological issues so serious and in which the EPA's conduct was so unjustified that the court considered they merited further examination.

The first of these issues was the question of confidence intervals. The plaintiffs alleged that the EPA, without explanation, switched from using standard 95-percent confidence intervals to 90-percent confidence intervals in order to enhance the likelihood that its meta-analysis would appear statistically significant. This shift assisted the EPA in obtaining statistically significant results that could be used to support a Group A classification (Osteen 1998: 74).

With a 95-percent confidence interval, there is a probability of only 5 percent that the result of a test is a product of chance. Generally, researchers are unwilling to accept higher probabilities of error. In its 1990 draft of the risk assessment, the EPA used a 95-percent interval but in subsequent drafts they switched to a 90-percent confidence interval. This change was criticized by Geoffrey Kabat, who served on the IAQC and also contributed to the risk assessment: "The use of 90 percent confidence intervals, instead of the conventionally used 95 percent confidence intervals, is to be discouraged. It looks like [a]n attempt to achieve statistical significance for a result which otherwise would not achieve significance" (Osteen 1998: 75, quoting Kabat 1992 at 6 [July 28, 1992] [JA 12, 185]).

Why, then, in the face of such internal criticism, would the EPA change its confidence intervals? In its risk assessment, the EPA argued that this usage was justified by the *a priori* hypothesis that a positive association existed between exposure to ETS and lung cancer (Osteen 1998: 75). But, as noted earlier, this explanation fails because it is circular. In a second attempt, the EPA explained to the court that use of the 95-percent confidence interval with the one-tailed test would have produced an apparent discrepancy: statistically significant study results using the standard *p*-value of .05 might nevertheless have a 95-percent confidence interval that included a relative risk of one (Osteen 1998: 75–76) In short, these studies would have failed to confirm that ETS was a significant health risk. As Judge Osteen observed:

The record and EPA's explanations to the Court make it clear that using standard methodology, EPA could not produce statistically significant results with its selected studies. Analysis conducted with a .05 significance level and a 95% confidence level included relative risks of 1. Accordingly, these results did not confirm EPA's controversial *a priori* hypothesis. In order confirm its hypothesis, EPA maintained its standard significance level but lowered the confidence interval to 90%. This allowed EPA to confirm its hypothesis by finding a relative risk of 1.19, albeit a very weak association. (Osteen 1998: 77)

What drove the EPA to change its confidence intervals and its epidemiological methodology is thus the same thing that drove it to select certain epidemiological studies in preference to other studies: the determination, regardless of the costs to scientific integrity and its statutory responsibilities, to justify its pre-determined position that ETS was a human carcinogen. Indeed, the record shows that even after carefully selecting its studies, the EPA still could not make its ETS case without abandoning normal scientific procedures. Again, the court noted that the record does not provide any reason for the EPA's abandonment of the 95-percent confidence interval (Osteen 1998: 78), despite the agency's clear responsibility to explain changes in methodology used during the conduct of a risk assessment. But, the EPA can no more explain why it changed confidence intervals than it can explain anything else about its procedure and findings. To do so would be to admit to scientific corruption.

Further reason to believe that the EPA's science was corrupt is to be found in the court's comments about the second problem with the EPA's epidemiological methodology. As a result of its statistical analysis using a 90-percent confidence interval, the EPA concluded that the relative risk (RR) of ETS was 1.19, and it was this finding that provided a large measure of its justification for the Group A classification (Osteen 1998: 76). Yet, as the plaintiffs noted, the EPA failed to provide any reason why such a weak RR justified a Group A classification. Every other Group A carcinogen had been required to exhibit a much higher relative risk (Osteen 1998: 76) and a recent candidate for Group A status with an RR range of between 2.6 and 3.0 had not been classified as a Group A carcinogen. Further, Dr. Kabat of the IAQC had noted: "An association is generally considered weak if the odds ratio is under 3.0 and particularly when it is under 2.0, as is the case in the relationship of ETS and lung cancer" (Osteen 1998: 76-77).

Clearly then, there is no precedent for Group A classification on the basis of such a weak RR. But, why should the consistency, adherence to normal procedure, and evidence-based decision-making be thought important at this final stage? Consider what had preceded this final step in the assessment procedure: certain epidemiological studies had been deemed relevant and others irrelevant on the basis of no clear criteria; the relevant epidemiological studies had been analyzed at a 90-percent confidence level rather than the usual 95-percent confidence level; and this, in turn, had produced a RR of 1.19, which in no other circumstance would be judged sufficient to justify Group A classification.

Two things about this process of scientific corruption were particularly troubling to the court. First, and most obviously, with such a weak RR, the problems with study selection and methodology meant

that the EPA could not show a statistically significant association between ETS and lung cancer in non-smokers (Osteen 1998: 78). In other words, the risk assessment was invalid.

Second, while the Radon Act authorizes the EPA to collect information, conduct research, and disseminate findings, the EPA's epidemiological basis for its risk assessment actually represented a suppression, if not a misrepresentation, of information. The EPA did not disclose certain facts either in the record or in the assessment: its inability to demonstrate a statistically significant relationship under normal methodology; its rationale for adopting a one-tailed test; and the shaky foundations for its RR rating for ETS. Instead of disclosing information, the agency withheld significant portions of its findings and reasoning in striving to confirm its *a priori* hypothesis (Osteen 1998: 79).

Two of the most characteristic features of corrupt science are its misrepresentation of reality and its misrepresentation of its procedure. Rather than acknowledging alternative evidence or problems with its evidence that would cast doubt on its conclusions, and rather than admitting the complexity of the issue under review and the limits of the evidence, corrupt science presents what is at best a carefully chosen partial truth as the whole truth necessary for public policy. In effect, public policy is manipulated into reaching certain conclusions on the basis of data that have been fabricated, falsified, misrepresented, or massaged to appear in a guise fundamentally at odds with reality. Corrupt science misrepresents not only reality but also its own procedures in arriving at its conclusions. Instead of acknowledging the selectivity of its procedure and the official insistence to demonstrate a predetermined conclusion, corrupt science invests both its procedure and its conclusions with a mantle of indubitability.

This is precisely what the court found in this case. The EPA failed to disclose its procedures and its failure to make its case under normal scientific procedures. It also failed to disclose its reasoning for changing its normal procedures, both methodological and with respect to the RR level required for Group A status. Most importantly, it failed to reveal how dependent its findings were on these departures from the norm. As a result, what the EPA presented as fact would be accepted by the casual observer as being scientifically supported when in actuality the truth was fundamentally different.

Conclusion

Judge Osteen began his analysis of the EPA's risk assessment by asking whether a different procedure would have produced different results; in effect, he asked whether the EPA's science was open to question. In his conclusion, he pulled together his findings both about the procedure

and the substance of the EPA's risk assessment, to determine whether the risk assessment demonstrated reasoned decision making. He entered judgment in favour of the plaintiffs and vacated the EPA's ETS risk assessment. The Judge's major findings were:

- (1) In 1988, EPA initiated drafting policy-based recommendations about controlling ETS exposure because EPA believed ETS is a Group A carcinogen. (Osteen 1998: 87)
- (2) Rather than reach a conclusion after collecting information, researching, and making findings, EPA categorized ETS as a "known cause of cancer" in 1989. (Osteen 1998: 88)
- (3) EPA determined it was biologically plausible that ETS causes lung cancer. In doing so, EPA recognized problems with its theory, namely dissimilarities between MS and ETS. In other areas of the Assessment, EPA relied on these dissimilarities in justifying its methodology. (Osteen 1998: 80)
- (4) EPA did not explain much of [sic] the criteria and assertions upon which EPA's theory relies. (Osteen 1998: 80)
- (5) EPA claimed selected epidemiologic studies would affirm its plausibility theory. The studies EPA selected did not include a significant number of studies and data which demonstrated no association between ETS and cancer. (Osteen 1998: 80)
- (6) EPA did not explain its criteria for study selection, thus leaving itself open to allegations of "cherry picking." (Osteen 1998: 80-81)
- (7) Using its normal methodology and its selected studies, EPA did not demonstrate a statistically significant association between ETS and lung cancer. (Osteen 1998: 81)
- (8) This should have caused EPA to reevaluate the inference options used in establishing its plausibility theory. (Osteen 1998: 81)
- (9) EPA then claimed the bioplausibility theory, renominated the *a priori* hypothesis, justified a more lenient methodology. (Osteen 1998: 88)
- (10) EPA claimed, but did not explain how, its theory justified changing the Agency's methodology. (Osteen 1998: 81)
- (11) With a new methodology, EPA demonstrated from the selected studies a very low risk for lung cancer based on ETS exposure. Based on its original theory and the weak evidence of association, EPA concluded the evidence showed a causal relationship between cancer and ETS. (Osteen 1998: 88-89)

- (12) In conducting the ETS risk assessment, EPA disregarded information and made findings on selective information; did not disseminate significant epidemiologic information; deviated from its Risk Assessment Guidelines; failed to disclose important findings and reasoning; and left significant questions without answers. EPA's conduct left substantial holes in the administrative record. While doing so, EPA produced limited evidence, then claimed the weight of the agency's research evidence demonstrated ETS causes cancer. (Osteen 1998: 90)
- (13) So long as information collection on all relevant aspects of indoor air quality, research, and dissemination are the lodestars, the general language of the Radon Research Act authorizes risk assessments. (Osteen 1998: 89)
- (14) Gathering all relevant information, researching, and disseminating findings were subordinate to EPA's demonstrating ETS as a Group A carcinogen. (Osteen 1998: 90)
- (15) In the Radon Research Act, Congress granted the EPA limited research authority along with an obligation to seek advice from a representative committee during such research. Congress intended industry representatives to be at the table and their voices heard during the research procedure. EPA's authority under the act is contingent upon the agency hearing and responding to the represented constituents' concerns. The record evidence is overwhelming that IAQC was not the representative body required under the Act. Had EPA reconciled industry objections voiced from a representative body during the research procedure, the ETS risk assessment would be very possibly not have been conducted in the same manner nor reached the same conclusions. (Osteen 1998: 91)

Corrupt science re-visited

Does the pattern of conduct described here consistently constitute corrupted science? We would argue that it does. Indeed, we would suggest that the EPA's ETS risk assessment is a case study in the corruption of science. Recall that corrupt science involves four characteristics: (1) movement from policy to science rather than science to policy; (2) misrepresentation of reality through misrepresentation of evidence; (3) misrepresentation of procedures; and (4) attempts to suppress dissent through attacks on the character of the dissenter and the motivation for dissent rather than on its logic and the evidence. The court's findings are decisive in each of these areas.

(1) Movement from policy to science

The record clearly shows that the EPA began with a conclusion about ETS (quotations 1, 2, 14 above) rather than with a question. As the court noted, the EPA's collection and assessment of evidence was merely incidental window-dressing to the procedure of conducting a risk assessment. (Osteen 1998: 72–73) Everything that the EPA did was designed to bring about the desired conclusion.

(2) Misrepresentation of reality

First, the EPA proposed a bioplausibility hypothesis that was unsupported, then sought to bolster the thesis with epidemiological evidence while simultaneously claiming that the same evidence supported bioplausibility (quotations 2, 3, 8, 9 above). Further, the EPA attempted to mislead the court about the evidence for the bioplausibility thesis and its inconsistent use of the theory.

Second, rather than present the entire evidentiary record, the EPA arbitrarily excluded certain epidemiological studies that demonstrated no association between ETS and cancer (quotations 5, 6, 7, above). The EPA provided no credible reasons for its exclusion of certain studies and inclusion of other studies.

Third, having found that even its selected studies failed to demonstrate an association between ETS and lung cancer, the EPA re-analyzed its studies using a 90-percent confidence interval rather than a 95-percent interval. The agency provided no explanation for its change in methodology. This allowed the agency to demonstrate a statistically significant risk of lung cancer in non-smokers exposed to ETS (quotations 7, 9, 10, 11, above).

Fourth, the EPA used the resulting RR of 1.19 as the major basis for its Group A classification of ETS although every other Group A carcinogen had required a higher RR and its own IAQC member, Dr. Kabat, indicated that a RR of 1.19 indicated a weak association. The EPA failed to provide the court with convincing reasons for this inconsistency (quotations 10, 11, 12, above).

Fifth, the EPA failed to disclose its inability to demonstrate statistical significance under normal scientific procedures and the fact that its weak RRs were obtained only after changing methodology. Indeed, the agency withheld significant portions of its findings and reasoning (Osteen 1998: 79; quotations 12, 13, above).

(3) Misrepresentation of processes

First, the EPA failed to conform to the procedural requirements of the Radon Act, requirements that were designed to create an objective and transparent procedure of risk assessment in which all sides had the op-

portunity, on the record, to examine the evidence (quotation 15, above). Further, the EPA attempted to maintain that it had discharged its procedural responsibilities for openness and objectivity through the IAQC, although the IAQC contained no industry representation and that its concerns on several points were ignored by the agency.

Second, the EPA's risk assessment failed to disclose the nature of its scientific procedure, namely, that it moved from conclusion to evidence rather than from evidence to conclusion, and that everything was subordinate to demonstrating that ETS was a Group A carcinogen. (Os-teen 1998: 90; quotations 1, 2, 14, above)

Third, the EPA's risk-assessment procedure failed to provide a record of the rationale for its decisions to ignore the criticisms and reservations of its own Risk Criteria Office and IAQC members, its decisions about its data selection, its decisions about epidemiological methodology, and its decision to assign Group A status to ETS in the presence of a weak association. In the absence of such a record, it is impossible to conclude whether the EPA acted rationally (quotations 12, 15, above).

Fourth, the EPA failed to reveal the circular argumentative procedure involved with the bioplausibility thesis, the arbitrary procedure of its data selection, and the methodological departures from standard scientific practice (quotations 7, 10, 11, 12, above).

(4) *Suppressing dissent*

First, the EPA viewed the tobacco industry's scientific positions as untenable not on the basis of evidence or logic but simply because they were advanced by the industry. As industry attorney Rupp's letter indicated, at no time was there an opportunity for a scientific discussion of the fundamental issues regarding ETS (Kluger 1996: 10). The EPA's attitude to the legitimacy of the industry's science is neatly captured in the reply of an assistant EPA administrator, William Rosenberg, to Rupp: "Frankly, the tobacco industry's argument would be more creditable if it were not so similar to the tobacco industry's position on direct smoking" (Kluger 1996: 693).

Second, the EPA's certainty about ETS, in advance of the evidence, created such a belief in the unfalsifiability of its *a priori* hypothesis that it apparently encouraged a climate in which even members of its own advisory panel like Dr. Geoffrey Kabat, who disagreed with its procedures and conclusions, were challenged not on the basis of their scientific arguments but on their alleged and in fact non-existent connection to the tobacco industry. As Jacob Sullum writes, "In this context anyone who questioned the case against ETS risked being portrayed as a tool of the cigarettes—even if, like Kabat, he had never received a dime

from them” (Sullum 1998: 172). Inasmuch as the *a priori* hypothesis was a revealed dogma, dissent could have no legitimate foundation. Character, not coherence and consistency, was the criteria against which disagreements were measured.

Corrupt science?

Thus, the case for corrupt science is compelling. Each of the characteristics of corrupt science is present in multiple instances, wound together within a consistent pattern. It would be difficult, indeed, to provide an alternative account that provides so coherent an explanation for so many of the uncontested facts of the case.

The Controversy around the IARC study

The Osteen decision was not the only 1998 setback to the ETS junk-science/junk-policy machine. A second blow to the claim that second-hand smoke was a risk for lung cancer for non-smokers came from the unlikely source of the World Health Organization’s International Agency for Research on Cancer (IARC).

The study by the IARC on the relationship between ETS and lung cancer in non-smokers is the largest study ever conducted outside of the United States. The study, spanning ten years and involving 12 cities in seven European countries, involved 650 cases and 1542 controls, and was designed to provide a definitive answer to the question of the risks of lung cancer for non-smokers from second-hand smoke. The study is, thus, extremely important in answering our questions as to whether second-hand smoke is a risk to the health of non-smokers.

In its 1998/1997 biennial report published in March 1998, the IARC published a summary of its ETS study (IARC 1998). The study found no statistically significant increase in the risk of lung cancer for non-smokers exposed to ETS in four settings: workplace, home, home and workplace together, and childhood. Of particular interest from a regulatory standpoint was IARC’s finding that ETS exposure in indoor public settings such as restaurants did not result in a statistically significant risk of lung cancer for non-smokers.

In itself, there is nothing extraordinary about these results; indeed, they are similar to the majority of other ETS studies. What is, however, extraordinary is the response from the international public-health community and anti-smoking movement that followed a story about the IARC study in the United Kingdom’s *Sunday Telegraph* on March 8, 1998.

In an article headlined *Passive Smoking Doesn’t Cause Cancer—Official*, the *Telegraph* reported:

The world's leading health organization has withheld from publication a study which shows that not only might there be no link between smoking and lung cancer but that it could even have a protective effect.

The findings are certain to be an embarrassment to the WHO, which has spent years and vast sums on anti-smoking and anti-tobacco campaigns. The study is one of the largest ever to look at the link between passive smoking—or environmental tobacco smoke (ETS)—and lung cancer, and had been eagerly awaited by medical experts and campaigning groups.

Yet the scientists have found that there was no statistical evidence that passive smoking caused lung cancer.

Responding to the *Telegraph's* story, which was reported in Canada in the *Ottawa Citizen*, the British group, Action on Smoking and Health (ASH), complained to the Press Complaints Commission that the *Sunday Telegraph's* claims were “false and misleading,” a complaint that was subsequently rejected by the Commission. The United Kingdom's Chief Medical Officer, Sir Kenneth Calman, claimed that IARC's results had been “misreported” and the WHO characterized the *Telegraph* article as “false and misleading,” asserting that “passive smoking does cause lung cancer, do not let them fool you.”

Reaction from the Canadian anti-smoking movement was equally strong. David Sweanor, the Senior Legal Counsel for the Non-Smokers' Rights Association (NSRA), wrote in a memorandum to the Toronto NSRA office:

A Citizen reporter called me at home, twice about this “story” yesterday. He said they had this as an exclusive from the *Telegraph* . . .

I explained that they were being “had.” That Neil Collishaw at WHO was not aware of any such study, that it had too small a sample size to be statistically significant, that there were major authoritative reports that looked at all of the data, that this had “tobacco industry PR machine” written all over it . . .

This is tabloid journalism. It misinforms the public . . .

IARC may or may not have done such research. If they did, the methodology is such that it was bad science and would not have passed peer review. Of 650 lung cancer patients, likely about 600 were there due to direct smoking.

I spoke with Neil this morning. He does not know anything about this “WHO commissioned” research. (Sweanor 1998)

Several things about Mr. Sweanor's comments are worth noting. First is the apparent ignorance, not only of the anti-smoking movement, which claims comprehensive knowledge about tobacco science, but also of a senior Canadian official at WHO, Neil Collishaw of a major, 10-year scientific study about ETS. It is also curious that Mr. Sweanor appears to know quite a bit about what is apparently wrong with a non-existent piece of research.

Second is the apparent ignorance about how epidemiology works and what statistical significance means. For one thing, the "small-size" argument is incorrect as any risk assessment can be made statistically significant by increasing the sample size. IARC set the sample size itself so it can hardly *post facto* complain about the size not producing statistical significance. Again, the 650 cases were, by the very nature of the study's design, carefully chosen to be non-smokers, not smokers as Sweanor claims. Indeed, if Sweanor believes that 92 percent of all cases in ETS studies are misclassified—that is, that they are really smokers rather than non-smokers—as his claim that 600 of the 650 cases were smokers suggests, then the case against ETS completely collapses since all studies would be invalidated by misclassification bias. In effect, they would be studies about smokers and not about non-smokers. Contrary to Sweanor's claim, the IARC's study is not bad science; it is rather customary epidemiology.

Third is the outright inaccuracy of Sweanor's claims. The *Citizen* was not being "had": the study was done by IARC and not by the tobacco industry; it was not publicized by the tobacco industry but by WHO; the newspaper did not misinform the public but accurately reported IARC's own findings; and, finally, the IARC's study did pass peer review, being published in the *Journal of the National Cancer Institute* (Boffetta 1998).

What Mr. Sweanor's claim of "bad science" and what claims of "false and misleading" reporting by ASH and WHO really mean is that the IARC's study is terribly inconvenient science because it exposes, in the most dramatic fashion, the manufactured nature of the case for the risk of lung cancer from ETS. Not only does the IARC's study destroy the claim that there is a "scientific consensus" that ETS causes lung cancer in non-smokers but it also puts an end to the anti-smoking movement's false claim that the only studies showing ETS not to be a risk are those funded by the tobacco industry.

With so much at stake, it is imperative for the anti-smoking league that the IARC's study be discredited at all costs through the techniques of junk science, even though the study is the product of one of the health community's premier research organizations. What we see at

work are the usual techniques: misrepresentation of date, misrepresentation of process, and questioning of character rather than research quality. Thus, IARC itself in reporting its research claims that its relative risk for non-smokers exposed to ETS is 1.16, without explaining that since the confidence interval is 0.93 to 1.44, the results should be interpreted as meaning either an increase or a decrease in risk and, more importantly, that the results are not statistically significant, that is, it is impossible to distinguish them from chance. In effect, by failing to comment on the confidence interval and the issues of statistical significance, IARC conveys the fundamentally misleading impression that its study found an increased risk of 16 percent of lung cancer in non-smoking subjects exposed to ETS. Hence WHO can claim that passive smoking “does cause lung cancer, do not let them fool you.” But, clearly the only people being fooled are those who do not understand epidemiology and who take IARC’s claims at face value. The only scientific conclusion to be drawn from the IARC’s study is that it shows no association between ETS exposure and lung cancer in non-smokers. Claims by the IARC and WHO otherwise are simply junk science: scientific findings baldly misrepresented for policy purposes.

Moreover, Sweanor’s claim that IARC’s study procedure is “bad science” because it is flawed by poor study design and sample size appears to be another instance of inaccuracy. The IARC’s study is the second largest ETS study ever done; it ran over ten years and both the sample size and the study procedure were carefully designed by IARC. It is only because the results are inconvenient that the procedure is retroactively described as flawed.

Finally, not only Sweanor’s comments about the *Citizen* being “had” and that this story “had ‘tobacco industry PR machine’ written all over it” but also the world-wide effort by the health community and the anti-smoking movement either to link the IARC’s study or its publicity to the tobacco industry are themselves junk-science techniques. Rather than looking at the quality of the evidence, one focuses instead on the connections and character of the scientist in an attempt to discredit and suppress dissent. What makes this strategy so bizarre in this instance is that the anti-smoking movement was forced to discredit the source of its own scientific claims.

Conclusion

To return to where we started, both of these case studies show—in quite different respects—not only why sizeable majorities of most democratic societies believe that second-hand smoke causes cancer in non-smokers but how these beliefs have originated. In the first instance, the

techniques of junk science—misrepresentation of scientific reality, misrepresentation of scientific procedure and suppression of dissent—were used to establish an untruth. In the second instance, these same techniques of junk science were used to protect that untruth. Both instances raise fundamental and deeply troubling issues for a public health community and anti-smoking movement apparently undisturbed by their willingness to engage in producing junk science for the sake of winning their war against tobacco.

Appendix One-tailed and two-tailed tests

A null hypothesis is a precisely stated assertion associated with a statistical test; results of that test are intended to determine whether the null hypothesis should be accepted (regarded as true) or rejected (regarded as untrue).

Because we are more comfortable accepting demonstrations that statements are false than otherwise, statisticians usually arrange their experiments so that the null hypothesis is contrary to the underlying thesis. Thus, rejection of the null hypothesis corresponds to confirmation of the thesis.

Suppose that like the EPA we want to demonstrate that exposure to ETS increases the risk of lung cancer. Since we cannot examine everyone exposed to ETS, we design a statistical experiment to determine whether our thesis seems to be true. Our null hypothesis is: Exposure to ETS does not increase the risk of lung cancer. Next, we select random samples of individuals exposed to ETS and random samples of individuals not exposed to ETS. If equality holds between the two samples—that is, if the rates of lung cancer are not different—we have failed to demonstrate our thesis. If, on the other hand, individuals exposed to ETS have significantly higher rates of lung cancer, we can reject the null hypothesis.

In posing a null hypothesis for statistical testing one always states an alternative hypothesis that is to be accepted if the null hypothesis is rejected. The alternative hypothesis must encompass the entire range of alternative to the null hypothesis. In this case, the correct alternative hypothesis is that the risk of lung cancer in populations exposed to ETS and populations not exposed ETS are *different*; that is, populations exposed to ETS might have *increased* risks of lung cancer or they might have *reduced* risks of lung cancer.

This is an example of a two-tailed analysis since exposure to ETS can either increase or decrease the risk of lung cancer. In using a one-tailed test, the EPA failed to state the correct alternative hypothesis to its null hypothesis. The EPA, in effect, *assumed* that ETS exposure could only increase the risk (one tail) of lung cancer. Since a substantial number of studies have shown a *decreased* risk with ETS exposure . . . two-tailed tests are required. (Luik 1993/1994: 54)

References

- Boffetta, P., et al. (1998). Multicenter Case-Control Study of Exposure to Environmental Tobacco Smoke and Lung Cancer in Europe. *Journal of the National Cancer Institute* 90, 19: 1440–50.
- Gori, Gio B., and John C. Luik (1999). *Passive Smoke: The EPA's B[E]trayal of Science and Policy*. Vancouver, BC: The Fraser Institute.
- DeRosa, C. (1990). Memorandum to William H. Farland, Director, Office of Health and Environmental Assessment (April 27).
- Kabat, G. (1992). *Comments on EPA's Draft Report: "Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders."* S[ci]ence] A[d]visory] B[oard] 9.15 at 6 July.
- Kluger, R. (1996). *Ashes to Ashes: America's Hundred-Year Cigarette War, the Public Health, and the Unabashed Triumph of Philip Morris*. New York: A. Knopf.
- Luik, John C. (1993/1994). Pandora's Box: The Dangers of Politically Corrupted Science for Democratic Public Policy. *Bostonia* (Winter): 54.
- Osteen, W.L. (1998). *Order and Judgment in: Flue-cured Tobacco Cooperative Stabilization Corporation et al. V. United States Environmental Protection Agency and Carol Browner, Administrator, Environmental Protection Agency*. U.S. District Court, Middle District of North Carolina, Winston-Salem Division (July, 17). Reprinted after page 102 in Gori and Luik 1999.
- Sullum, J. (1998). *For Your Own Good*. New York: Free Press.
- Sweaner, David (1998). Memo to Non-Smokers' Rights Association (NSRA). Digital document published, with permission, by Randel Marlin at news-group ncf.sigs.propaganda+media.
- Teich, A.H., and M.S. Frankel (1992). *Good Science and Responsible Scientists. Meeting the Challenge of Fraud and Misconduct in Science*. Washington, DC: American Association for the Advancement of Science.
- United States Environmental Protection Agency [USEPA] (1991). An S[ci]ence] A[d]visory] B[oard] Report: Review of Draft Environmental Tobacco Smoke Health Effects Document.
- (1992). *Respiratory Health Effects of Passive Smoking. Lung Cancer and Other Disorders* (December 1992). Washington, DC: Office of Research and Development.
- International Agency for Research on Cancer (IARC) (1998). *Biennial Report, 1996–1997* (March). Lyon: World Health Organisation.