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I. INTRODUCTION

Administrative agencies such as the Environmental Protection Agency (EPA) and the Occupational Health and Safety Administration (OSHA) are required under statutory mandate to protect people from the risk of toxic substances found in the work place and the environment. Risk assessment may be defined as the scientific or quasi-scientific basis these agencies use to determine health risk of an environmental chemical. The subsequent promulgation, after a chemical is found to be toxic, of new rules or modification of existing rules in order make the work place or the environment safe from this chemical may be termed risk management. However, there are significant knowledge gaps in this area, resulting in scientific controversies. From a legal standpoint, courts are often required to decide conflicts in the area of risk assessment, many of which are based on different interpretations of scientific data. This litigation involves analysis of highly technical and scientific information. For example, in the case

involving setting exposure standards for ethylene oxide, notice and comment resulted in 1600 pages of transcript and 300 exhibits. (1) Therefore, it is important to understand how scientific issues have influenced court decisions in the area of risk assessment.

The overall objective of this report is to provide the reader with an overview of important scientific fundamentals involved in risk assessment of toxic chemicals, especially carcinogens, and how the courts have relied on knowledge of science to decide environmental disputes between private entities and federal agencies. The specific objectives of this review are (1) to provide the reader with a brief summary of procedures used to determine whether a chemical is a carcinogen (2) to discuss important statutory scientific standards, (3) to critically examine judicial review of the scientific concepts and methods presented to the court by the agencies and parties challenging agency rules and (4) to suggest solutions to minimize non-beneficial health safety regulation. It should be noted that it is beyond the scope of this review to discuss the validity of scientific data presented to the Court; it is assumed that data presented were acquired using acceptable scientific methods and procedures.

II. FUNDAMENTALS OF RISK ASSESSMENT SCIENCE

A. Definition and Explanation of Terms

1. Pharmacokinetics: may be defined as the study of " the concentration in target organs and the interaction of a biologically active agent with putative sites of action (2)

2. Power: is a statistical concept, which quantifies the ability of a study to detect an excess risk that truly exists (3).

3. Cohort means group

4. Epidemiology: The study of the prevalence and spread of disease in a community (4)

5. Toxicology: The science of poisons - their source, chemical composition, action, tests and antidotes (5)

6. ppm: parts per million

7. One hit hypothesis: This theory states that even a single molecule of a hazardous chemical can interact with a molecule of DNA causing a mutation

which can then lead to cancer over the years. The linear (no-threshold) models (described later) subscribe to this theory.

8. Maximum tolerated dose (MTD) can be defined as "a dose as high as possible without shortening the animals' lives from non-carcinogenic toxic effects (6)

B. Human Epidemiological Studies (7)

Cancer risk from exposure to a hazardous chemical, found in the environment or the work place is determined from human data and/or animal studies. Results from these studies are analyzed to determine whether environmental or work-place exposure to an agent can cause an increase in the incidence of a health condition (e.g. cancer, birth defects etc).

The best way to obtain such data is from "well designed" epidemiological studies, which can be classified as (1) cohort studies or (2) case-control studies. The objectives of these studies are to obtain direct evidence of a possible link between exposure to a particular chemical and a given disease. As will be discussed, these studies have many weaknesses which led the Occupational Safety and Health Administration to comment '"Although the epidemiological method can provide evidence of a causal relationship between exposure and disease in the case of positive findings, it is by its very nature relatively crude and an insensitive. OSHA's policy when evaluating negative studies [is] to hold them to higher standards of methodological accuracy." (8). The following details will enable the reader to appreciate the basis of OSHA's concerns about such studies. The example of a work place pollutant, suspected of causing cancer, is used to briefly illustrate the two types of study designs.

1. Cohort studies: A group of workers from the contaminated work place (the "experimental" group) and a group of subjects who is not exposed to this chemical (the control group) are selected on specific criteria. No member of either group has been diagnosed with the disease in question. Investigators then follow the subjects in both groups to see how many in each group develop the disease over time. A finding that more subjects in the experimental group develop the disease (e.g. cancer) would support, but not prove, the hypothesis that the chemical in question is a risk factor. Understandably, higher the frequency of cancer in the experimental group, higher the risk from this chemical. Some critical issues, such as selection of the members of the experimental and control groups, statistical methods to calculate the number of

subjects in each group, the duration of the study and analysis and interpretation of the results do not find universal consensus among scientists.

These studies provide prospective toxicity data under more controlled conditions than case control studies (discussed next). However, there are many practical problems in conducting such a study. Important among them are: (1) these studies are expensive because (a) they require a large number of subjects and (b) given the long latency period for cancer (5-50 years) (9), the duration of the study often takes many years and (2) study dropout rate is high due to job relocation and other similar convenience issues. The long duration of such studies also raises ethical issues of exposing subjects to suspected carcinogens for extended periods in the workplace. Therefore, it is not surprising that epidemiological data is available only for a very small number of chemicals. (10)

2. Case control studies: Such studies, which are retrospective in nature, involve the identification of one group where members have been diagnosed with the disease in question ("cases") and another group in which no member has the disease ("controls"). Then, the number of workers from the hazardous work environment is identified in each group. As in a cohort study, a finding that more subjects in disease group worked in the hazardous environment, would support, again not prove, the argument that there is a connection the work place and the disease. Case control studies also offer the advantage that it provides direct estimates of relative risk. In addition, the required sample size is relatively small; such studies are less expensive to conduct and especially suited to study rare diseases (11). On the other hand, careful diagnosis is required to ensure a proper representative control group is selected. Sometimes, subjects may have been exposed to more than one risk factor as in rubber workers who are exposed to "vinyl chloride, polychlorinated biphenyls, chloroprene, selenium compounds, benzidine and its salts, aniline, carbon tetrachloride and benzene " which are suspected or federal carcinogens (12). It is therefore, not possible, at times to identify which is the causative agent and develops appropriate safety measures.

C. Animal studies:

1. Introduction:

Animal studies are the most frequently method used by the regulatory agencies to assess the risk of toxicity, particularly carcinogenicity, of a given chemical to human (13). This popularity stems from the advantages that animal studies offer. Firstly, there is strong scientific support for the fact that most substances

that are carcinogenic in one mammalian species also induces cancer in other species (14)

. Secondly, the pathology of development of tumors in a variety of species resembles that in humans (15). Therefore, animal data is a reasonable alternative to human epidemiological data (16). In general, animal studies are, as might be expected, also considerably less expensive than human epidemiological studies.

2. Dose-Response (High Dose) Studies

A well-designed animal study would involve three, sometimes four, groups of animals with about 50 animals per group (17). These groups are as follows; (1) control group (no exposure) (2) an experimental group fed the maximum tolerated dose, MTD (3) an experimental fed one-half the MTD and (4) sometimes, an extra group with one-fourth the MTD. These studies usually last about 1 to 2 years. Cancer incidence in the four groups is then analyzed statistically to conclude if the chemical is a carcinogen.

3. Determination of low-dose effects

The next step, the most controversial from a scientific stand point, is to predict the existence of the smallest dose, if any, below which the chemical is not carcinogenic. The determination of such "low-dose effects" of the chemical is based on extrapolation of results from the higher doses used in the study. The proper extrapolation technique (or mathematical model) to use is not well understood and is based on many assumptions. For example, if the mechanism of cancer induction in the control group is assumed to be same as in the treated animals, then the effects are additive and the dose response curve is linear (i.e. there is no threshold dose. In other words, the chemical causes cancer at any dose, however small. This is the basis for the Delaney Clause for food additives, which prohibits the use any such agent, which at any dose causes cancer in animals. In pertinent part, it says ". . . no additive shall be deemed to be safe if it is found to induce cancer when ingested by man or animal, or if it is found, after tests which are appropriate for the evaluation of the safety of food additives, to induce cancer in man or animal. . .." (18) . A compound is considered "linear" with respect to its carcinogenicity if it is believed to pose a hazard at any level of exposure, however low. In contrast, non-linearity refers

to a threshold dose, below which the compound poses no such danger and may be considered safe.

Due to scientific uncertainties associated with the selection of extrapolation techniques, the safe dose for a given chemical might vary from agency to agency. A dramatic example, depending on the model used, linear or non-linear, the magnitude of uncertainty may be a million fold. (19)

III JUDICIAL REVIEW

A. "Best Available Science"

Federal agencies are required to use the "best available science" when establishing exposure standards. For example, statutory standards are that when OSHA chooses to regulate a toxic chemical it must set

Standards which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life. Development of standards under this subsection shall be based upon research, demonstrations, experiments, and such other information as may be appropriate. In addition to the attainment of the highest degree of health and safety protection for the employee, other considerations shall be the latest available scientific data in the field, the feasibility of the standards, and experience gained under this and other health and safety laws. (20)

In a recently decided case (21), the Court held that EPA had not "considered the best available evidence" in setting the maximum contaminant level for chloroform under the Safe Drinking Water Act (SDWA). The SDWA directs EPA to set standards for certain contaminants found in drinking water. These two standards are (1) the maximum contaminant level goal (MCLG), which is defined as "the level at which no known or anticipated adverse effects on the health of persons occur and which allows for adequate margin of safety." (22); MCLG may be considered a desirable target concentration and (2) the maximum contaminant level ("MCL"), which is the enforceable standard based on practical considerations, which is "as close to the MCLG as is feasible" (23). Chloroform is a by-product of chlorination of water, which is the most widely method for ensuring drinking water safety by controlling microbial pathogens. In July 1994, based on rat studies which showed that chloroform is a probable

human carcinogen, EPA issued a proposed rule setting the MCLG of chloroform as zero, because the EPA was unable to suggest a threshold level below which there would be no risk of carcinogenicity. This is based on EPA's published default method of linear extrapolation of assessing risk under such circumstances. (24) The default method is based on the argument that, when there is insufficient data to support a threshold dose, then linearity may be assumed. Subsequent peer-reviewed research by a panel of scientific experts organized by the International Life Sciences Institute and under the auspices of the EPA, concluded in 1998 that chloroform was unlikely to pose any risk of carcinogenicity below a dose range of 300 ppb (parts per billion). (25). This lawsuit (26)was filed when EPA ignored this scientific evidence and published its final rule and again listed chloroform MCLG as zero. (27). The SWDA requires the EPA Administrator to use "the best available, peer-reviewed science and supporting studies conducted in accordance with sound and objective scientific practices." (28). The Court, based on this statutory mandate, ordered the EPA final rule to be vacated since it was " arbitrary, capricious and in excess of statutory authority" since " [b] est scientific evidence indicated that chloroform posed no risk of cancer below some threshold levelÂ…." (29)

Using the same "best available evidence" standard, the Supreme Court (30) set aside an OSHA rule lowering benzene exposure levels from 10 ppm to 1 ppm, on the basis that the agency had not shown that such lowering reduced risk. Benzene is a colorless, aromatic, volatile liquid, which is an important commercial commodity with an annual production of 11 billion tons in 1976 (31). "The entire population of the United Sates is exposed to small quantities of benzene, ranging from a few parts per billion to 0.5 ppm" (32). In April 1977, the National Institute of Occupational Safety and Health (NIOSH), the research arm of the OSHA, reported to the agency that exposure to benzene results in a five-fold increase in the incidence of leukemia in humans. Based on this report, the OSHA issued an emergency order effective May 21, 1977 lowering the exposure level to 1 ppm. (33). However, NIOSH made a critical error; the actual benzene concentration at the site of study, which had been initially reported to be between zero to 15 ppm, was later admitted by the investigators to be 100 ppm. A temporary restraining order by the Court of Appeals for the Fifth Circuit blocked this order from taking effect. Then, OSHA moved procedurally to make permanent the aborted 10-ppm standard by the notice and comment method (34). Interestingly, OSHA sought comments only about the feasibility of the 1ppm exposure standard. No comments were solicited as to health risk at exposures of 10 ppm or lower. Commenting on the

testimony of OSHA that it assumes the non-existence of a safe level in the absence of clear proof of such a level, the Court said " Given OSHA's cancer policy, it was in fact irrelevant whether there was any evidence at all of a leukemia risk at 10 ppm" (35). Impartial members of health science community would applaud the Court's decision on the grounds that proposals to lower exposure levels of carcinogens should be accompanied by evidence of benefits of such proposals, which usually have a negative economic impact.

B. Significant risk/Substantial evidence Standard

Arsenic, a by-product of metal smelting, was found to be carcinogenic in humans. (36) . Subsequently, OSHA decided to lower arsenic exposure in the work place from a maximum permissible level (PEL) of 500 microgram per cubic meter (ug/m3) to 10 ug/m3, which was challenged (37)on the ground that the agency's findings were not supported by substantial evidence as required by the Occupational Health and Safety Act (38). Under this Act, the agency is required to determine that (1) the current standard applicable to the toxic agent poses significant health risks and (2) such risk can be reduced by the proposed change (in this case, the lowering of arsenic exposure level(39). The Court in effect had to decide which of conflicting sets of data, offered by the 2 parties, it should accept. In reviewing conflicting data, the standard used by the Court usually favors the agency:

[W] here the agency presents scientifically respectable evidence, which the petitioner can continually dispute with rival, and we will assume equally respectable evidence, the court must not second guess the particular way the agency chooses to weigh the conflicting evidence or resolve the dispute. (40) Apart from the Chevron case (41)where agencies are allowed wide latitudes in statutory interpretation, the advantage the agencies have over parties that oppose agency decision is also noted in the holding that "OSHA is not required to support its finding . . . with anything approaching scientific certainty. (42)

The first question for the Court in this case (43) was to answer: Is the health risk to workers at an arsenic exposure level of 500 ug/m3 supported by substantial evidence? A related second issue was: Will the lowering of exposure to 10 ug/m3 "significantly reduce" that risk The OSHA using mostly epidemiological data from copper smelters(44)calculated a range of 148-767 excess deaths per 1000 employees at an exposure level of 500 ug/m3; the corresponding range was 2.2-29 excess deaths at the new statutory standard of 10 ug/m3 (45). The Supreme Court has stated that a "reasonable person might well consider . . .[even one excess death per 1000] significant . . .." (46) The

petitioner unsuccessfully argued that data used by OSHA were flawed and, therefore could not be considered "substantial evidence"; they presented conflicting evidence, which found no excess risk of death at the pre-existing exposure concentration of 500 ug/m3. The Court went so far as to not only find OSHA's data more reliable (i.e., provided 'substantial evidence") but also stated "the [petitioner's] data was "critically flawed". (47)

Ethylene Oxide, a highly reactive gas, is a chemical widely used in manufacturing, and to smaller extent in sterilization of hospital equipment. OSHA, after prodding by the Court, subsequent to public interest litigation, issued a final rule relating to long-term permissible exposure limits (PEL) and short term exposure limits (STEL) of 1 ppm and 10 ppm, respectively for ethylene oxide (EtO). (48). The agency however had reserved judgment on the STEL for EtO, because the Office of Management and Budget (OMB) had objected to the publication of these rules; specifically, OMB's had concerns, inter alia, related to STEL limit issued for ETO which, OMB felt was "unsupported by any reasonable risk assessment or inference from available scientific data" (49). Following reopening the record for further public comment, OSHA declined, in view of conflicting opinions presented at the notice and comment stage, not to issue a final ruling on STEL for EtO. This resulted in several challenges (50); of pertinence are those from a public interest group, Public Citizen Health Research Group ("Public Citizen") and an industrial group, the Association of Ethylene Oxide Users ("AEOU"). Of pertinence to this discussion is AEOU's contention that epidemiological evidence submitted by OSHA was "rendered totally valueless by their methodological flaws" (51). Under the ruling of Benzene case (52), OSHA had to show first that the pre-existing PEL of 50 ppm posed "significant risk". In support of this requirement, OSHA submitted results from human epidemiological and animal (rat and monkey) studies, which showed EtO exposure is liked to cancer. Though the human studies had certain flaws admitted by OSHA, the Court, taking into consideration the cumulative evidence of the results from human and animal studies submitted by OSHA concluded that the "substantial test was met" because a "reasonable person could draw from this evidence the conclusion that exposure to EtO presents a risk of cancer." (53) It also emphasized that this "Court's role is not review the evidence de novo to arrive at our own estimate of the risks;" (54).

As the second required step, OSHA provided data on quantification of risk using a mathematical model which assumes that there is no safe threshold exposure for EtO which was challenged by AEOU on the highly specific

scientific grounds that (1) OSHA improperly ignored evidence that shows that EtO does have a safe threshold (2) there are errors in the extrapolation of breathing rates from rats to humans. Arguments by AEOU in support of issue (1) involve comments of one participant during the notice and comment that speculated that that EtO has a safe threshold dose given the fact that EtO can be detoxified (i.e., metabolized) in the body. The Court overruled this objection by stating that this participant had reached "a tepid conclusion that is reasonable to believe that a safe level of ethylene oxide exits" (55). With respect to the rat to human conversion of inhalation rates, the basis of the AEOU challenge was rather weak since it was based on differences in conversion factors used by different agencies. The Court dismissed this challenge on the grounds that OSHA's results were not "seriously flawed" (56)

Public Citizen unsuccessfully challenged OSHA's decision to omit STEL in the final rule (57) for EtO on basis that a key scientific (pharmacological) fact used by the agency had not been shown to be true. OSHA's decided to exclude STEL for EtO on the assumption that EtO lacked "dose-rate effects" (58). This means that the effects of EtO are dependent on the dose and not the duration over which workers are exposed to this dose (i.e. dose-rate). Public Citizen argued that analyses done by the agency suggested that EtO did have dose-rate effects. In ruling against Public Citizen, the court said, "These statements [made by Public Citizen] do not amount to scientific certainty binding on the agency" (59). While this statement would suggest that a stronger show of scientific evidence would help the petitioner, the Court, in the same breath added "We reiterate that the very nature of scientific on the frontiers of scientific knowledge will rarely allow a court to compel an agency to adopt a particular hypothesis." (60)

However, on the issue whether STEL was needed at all, the Court disagreed with OSHA. The Court however did not accept the agency's argument that STEL was not needed for EtO since PEL would require that employers keep levels at 1 ppm. The Court correctly argued that since PEL was based on an eight-hour average, short term exposures can be higher than 10 ppm yet meet the PEL limit of 1 ppm. The Court remanded this issue to OSHA for further study the interrelationship between PEL and STEL.

C. Model Selection: Threshold v. No-threshold

Petitioners argue that OSHA choice of a no threshold (i.e., linear) model to assess the risk of arsenic exposure to workers in smelters was not based on scientific reasoning but by administrative fiat (61). In addition, they proposed a non-linear or threshold model to quantify carcinogenic risk of arsenic (62). The

Court dismissed the arguments on the basis that the expert for OSHA had analyzed the epidemiological data by both linear and non-linear models and found that a linear model better described the data. (63). In the absence of a details of data provided by each party, independent analysis of the scientific merits of the Court's holding is difficult. It appears from many decisions of the Court that it behooves parties wishing to challenge administrative agencies to fully understand the scientific methods and standards used by them as part of the decision making process to filing suit.

D. Opinion of scientists is not scientific evidence

In January 1991, EPA issued the final rule for MCGL and MCL for thirty-eight organic and inorganic chemicals (64). This case involves 4 of these chemicals, 1,2 dibromo-3-chloropropane ("DBCP"), ethylene dibromide ("EDB") tetracholroethylene ("perc') and polychlorinated biphenyls ('PCBs") for each of which EPA set MCLG of zero.

The challengers' (65) first argument, a general one, which applies to all four chemicals, is that EPA, arbitrarily and capriciously failed to consider two available pieces of scientific evidence in proposing the final rule (66). This evidence consisted of two items: (1) a short (three- page) letter to the editor by two scientists (67), which proposed that "low doses of carcinogens appear to be . . .less hazardous than is generally thought" (68) and (2) a declaration by another scientist who "pointed out the difficulties inherent in drawing conclusions about humans from studies conducted in animals" (69). The Court held that EPA gave this "new evidence" adequate attention, since neither of these documents contained any statistical analysis of available data nor pointed out weaknesses of methods and data generally relied upon by the scientific community. In other words, the Court concluded that the petitioner's "new data" were mere opinions of a few scientists and did not constitute scientific data. This author would agree that the court's decision was well reasoned and would also find much support in the scientific community.

E. Adequate evidence is needed

Petitioners argued that EPA had not adequately explained reasons for its action in setting the MCLG and MCL for the pesticide 1,2 dibromo-3-chloropropane ("DBCP") at zero and 0.0002 mg/l, respectively. (70) In addition, the petitioners claimed that EPA had improperly rejected data submitted by them. Here, the petitioners had submitted two epidemiological studies during the

comment phase which provided epidemiological data obtained from workers exposed to DBCP, primarily through inhalation in the workplace; the petitioners also stated that "do [] not show any statistically significant increase in either overall cancer rates of any specific cancer type." (71). The EPA in turn argued that they rejected the petitioner's epidemiological date based on its (EPA's) assessment that the two-year follow up time was inadequate and the study lacked adequate statistical power (one factor which reduces statistical such power is the use of an inadequate number of subjects in the study). Without access to all the data, it is not possible to critically evaluate EPA's decision from a scientific point of view. However, a two-year follow up is indeed very short, given the latency period (interval between exposure to development of cancer) can be as long as 40 years (72). The Court correctly concluded that the EPA had satisfactorily explained the reasons for rejecting petitioner's data and met the standard of "satisfactory explanation" required in rule making (73).

In the same case (74), the Court sustained EPA's decision to reject petitioners epidemiological data on ethylene dibromide, a pesticide (banned for this use by the EPA in 1983) and a gasoline additive (under EPA regulation), based on EPA's assessment that (1) the study used a small population size (there were 156 subjects in this study) (2) exposure rates were poorly characterized and (3) mortality studies in workers are inconclusive with respect to cancer risk.

Another criticism the EPA had for the two studies submitted by the petitioner involved differences in the route of administration (75). Since these epidemiological studies were obtained based on human exposure via inhalation, EPA argued, "toxicity may depend on dosing route . . .." (76). The EPA's argument, though accepted by the Court, was speculative because it appears that data on body burdens (amount of chemical entering the body) were not available. Firstly, the carcinogenicity of a chemical is independent of the route by which it enters the body. Of more importance is the amount of chemical that enters the body, and not the route per se. On a linear model, any exposure can cause cancer (though the probability decreases as the dose decreases); while in a non-linear model, cancer is caused only when exposure exceeds the threshold dose. In either case, pharmacokinetic assessment of body burdens (e.g., blood concentrations) of a chemical is needed to properly compare studies using two different routes (in this example, inhalation via oral).

In July 1997, EPA issued final rules for primary and secondary standards for ozone (77). Primary standard refers to a concentration level "requisite to protect public health" with an adequate margin of safety and a secondary standard a level "requisite to protect the public welfare. (78) Challengers to these

standards complained that the EPA had ignored data presented by them on health benefits of troposphere ozone, such as protection shield against cataracts and skin cancers caused by the sun's ultraviolet rays (79). The criteria to be used by the EPA for each pollutant under the Clean Air Act (80) are to "reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of such pollutant in the ambient air, in varying effects. In accepting the petitioner's complaint, the Court remanded the ruling for further consideration by EPA on the beneficial effects of troposphere ozone.

F. Animal data is acceptable to decide human carcinogenicity

The petitioner's argued that EPA had relied (presumably inappropriately) on data from animal studies where they had used "extremely high doses", (81). This is one of the major scientific criticisms of animal studies where high doses are often used to test for carcinogenicity. It is therefore argued in some circles that toxicity of chemicals are overestimated and such reports needlessly frighten the public, resulting in legislative action (or reaction) resulting in a "vicious cycle" (82), as one author, now a Supreme Court justice characterized this sequence of events. Further, the petitioners contend that tumors "tended to develop at the site of contact (83), suggesting that the chemical is safe when ingested (i.e., not a systemic toxin). EPA countered that they had used two rodent species (rats and mice) and three exposure routes (oral, inhalation and dermal) to determine that DBCP, a known carcinogen produced tumors at both local and distant sites. The court ruled that the EPA had "sufficiently justified its reliance on animal studies" (84). Based on the available of EPA's data, it appears that the petitioners had a weak case from a scientific point of view. Perhaps it was for tactical reasons that such a suit was filed, since it is difficult how the petitioners' scientific experts could have missed EPA's animal data.

G. Validity of the method used for chemical analysis

In one case (85), the petitioners argued that the chemical method used for analysis of PCBs in water samples was adopted without the required notice and comment (86). EPA had admitted that the method used at the proposed rule stage had been verified in only one laboratory (87). The method (Method 508 A) involves the use of the well-established analytical technique called gas chromatography (88), which is widely used by scientists to measure compounds such as PCBs at low (e.g. parts per million) concentrations. From a scientific standpoint, the petitioner's argument is weak, because when a relatively simple method is properly validated, even if only in one laboratory, other laboratories should be able to confirm the original work. In fact, the

SDWA only requires that the EPA " use data collected by accepted methods or best available methods (if the reliability of the method and the nature of the decision justifies use of the data). (89) Before promulgating the final rule, the EPA confirmed the reliability of Method 508 A by showing that results obtained with this method was consistent with those obtained by several private laboratories (Water Studies 23-25). The Court, using these supporting scientific data and other past regulatory holdings (90), agreed that EPA adequately validated Method 508 A and denied the petitioners request that the case be remanded so that they may submit criticisms of the Water Studies 23-254. From a scientist's perspective, the holding is sound because the petitioners had a chance to comment on the possible weaknesses of method 508 A and could have suggested improvements during the notice and comment phase.

IV. SOLUTIONS FOR EFFECTIVE RISK ASSESSMENT

Many feel that we live in an over regulated society. Further, it also widely believed that the cost to benefit ratio is very high. For example, it has been estimated that regulation of benzene and coke by-product recovery plants cost over $200 million but only saves 3 to 4 lives; in another case regulatory costs has been estimated to about $180 million to save one statistical life (91). Morally and ethically it is understandably offensive to put a price tag on human life. Perhaps, such cost estimates may be found conscionable if it is noted that this money can be used for helping other lives, such as funding programs to reduce hunger, or to improve nourishment for children in the US or in the world at large.

In searching for solution, one has to first understand dynamics of regulatory law making. Such understanding will help identify areas to focus on in this search. The main players that drive the regulatory engine are the Congress, the courts and regulatory agencies. Based on legitimate or perceived public health concerns, scientific studies are conducted by the regulatory agencies, as their mandates require them to do, to explore the validity of these concerns. If found necessary, the agencies promulgate new rules or modify them. Challenges to these rules, if they cannot be resolved at the agency level, end up in our courts, which have to resolve these technically very complex disputes. Based on the case law discussed (see supra), it has clear that agency decisions are given much deference by the courts. It is only on rare occasions that an agency loses in court. Perhaps, this is the reason it has been stated that answers to the problem of over regulation is unlikely to be found in our court system (92). However, establishment of special court, as with patent litigation, that deals exclusively with cases connected with environmental and workplace health hazards is one solution. Such a court must have its team of independent health

scientists and economists to provide it with a critical analysis of the scientific issues and a cost/benefit analysis of the regulation in dispute. The advantages of such a court are: (1) it would be more willing to challenge agency decisions on scientific/ technical and economic grounds; this should help the quality of rule making and (2) over time, such a court can be expected to develop insights into the complicated nature of risk assessment; such knowledge should be a driving force towards a more uniform interagency policy of regulating hazardous substances, either in the environment or workplace.

It has been opined that changes in the regulatory agencies might be helpful (93). The present structure has been stated to suffer from "[t] unnel vision, a classic administrative disease, [which] arises when an agency so organizes or subdivides its tasks that each employee's individual conscientious performance effectively carries a single-minded pursuit of a single goal too the point where it brings about more harm than good". (94) As a consequence, one former EPA administrator noted is that while most (about 95%) of the toxic material could be removed from waste sites in a few months, years are spent trying to clean up the remainder (95). The author recounts one of his own experience where the "cost of cleaning up the last bit [] cost about $9.3 million" (96). In effect, this money was spent " to protect non-existent dirt-eating children" (97). Changes, proposed to prevent such outcomes are establishment of a centralized agency with civil servants with wider expertise in such areas as "health and environmental agencies, Congress and OMB" (98). This agency will have five features (1) a mission of building a coherent risk-regulating system (a) interagency jurisdiction, (3) political insulation (4) prestige and (5) authority.(99) The Science Advisory Board of the EPA has been given as a model to develop this centralized agency. The need for more science in the courtroom was also emphasized in the silicone breast implant settlement (100). A critic of such a "super agency", might object that it is politically unacceptable and undemocratic, elitist, ineffective, impractical (101).

In conclusion, discussions are urgently needed in the legal community on the use and misuse of science in risk assessment . It is hoped that such discussions would lead to more efficient risk assessment regulation and enforcement, which have significant health and economic implications for society.

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1. 49 Fed. Reg. at 25737 (quoting from Public Citizen Health Research Group v. Tyson 796 F. 2d at 1483)

2. National Research Council. Pharmacokinetics. p. xi (quoting Carl F. Cranor, Regulating Toxic Substances (1993), at 118)

3. 48 Fed. Reg. at 1875

4. Stedman's Medical Dictionary, 23rd Edition, page 470-71

5. Id at 1461

6. U.S. Congress, Office of Technology Assessment, Identifying and Regulating Carcinogens (Washington, D.C.: GPO, 1987, p. 39 (quoting Carl F. Cranor, Regulating Toxic Substances (1993) at p.17)

7. See generally Carl F. Cranor, Regulating Toxic Substances (1993)

8. Industrial Union Department, AFL-CIO v. Amer. Petroleum Institute 448 U.S. 607, 635

9. Carl F. Cranor, Regulating Toxic Substances (1993), at p. 30

10. Researchers wanting to compare human data with animal data could find such data for 23 chemicals. Bruce C. Allen, Kenneth S. Crump and Annette M. Shipp, " Correlations Between Carcinogenic Potency of Chemicals in Animals and Humans," Risk Analysis December 1988, pp. 531-44 (Carl F. Cranor, Regulating Toxic Substances (1993), at p. 16

11. Carl F. Cranor, Regulating Toxic Substances (1993) at p. 30

12. Id.

13. Id. p.12-15.

14. U.S. Interagency Staff Group on Carcinogens, "Chemical Carcinogens: A Review of the Science and Its associated Principles," Environmental Health Perspectives 67, 1986: 274 (quoting Carl F. Cranor, Regulating Toxic Substances (1993), at 16).

15. Carl F. Cranor, Regulating Toxic Substances (1993), at 16

16. Id

17. Id. at 17

18. 21 USC sec. 348 (c) (3)(a).]

19. From Cranor "An overview of risk Assessment," in Proceedings: Pesticides and other Toxics: Assessing Their Risks, ed. J. White (Riverside: University of California, College of Natural and Agricultural Sciences, 1990, page 83 (quoting Carl F. Cranor, Regulating Toxic Substances (1993), Appendix A)

20. 29 U.S.C sec. 655 (b) (5)(1982)

21. Chlorine Chemistry Council v. Environmental Protection Agency, 2000 U.S. App. Lexis 5825 (D.C. Cir, 2000)

22. 42 U.S.C. § 300g-1(b)(4) A

23. Id. § 300g-1(b)(4)(B)

24. 61 Fed. Reg. 17960, 17968/3

25. 63 Fed. Reg. 15674 (1998)

26. Chlorine Chemistry Council v. Environmental Protection Agency, 2000 U.S. App. Lexis 5825

27. 63 Fed. Reg. at 68398/3

28. 42 U.S.C. sec. 300g-1(b)(3)(A)

29. Chlorine Chemistry Council v. Environmental Protection Agency

30. Industrial Union Department, AFL-CIO v. American Petroleum Institute, 448 U.S. 607

31. Id at 615

32. Id

33. 42 Fed. Reg. 22516 (1977)

34. Id at 27452

35. Industrial Union Department, AFL-CIO v. American Petroleum Institute, 448 U.S. 607 at 625

36. 40 Fed. Reg. 3392.

37. ASARCO v. OSHA 746 F.2d 483,

38. 29 U.S.C. sec 655(f)

39. Industrial Union Department, AFL-CIO v. Amer. Petroleum Institute, 448 U.S. 607 at 625

40. ASARCO v. OSHA 746 F.2d 483 at 491 (quoting United Steelworkers of America v. Marshall, 647 F.2d 1189 (DC Cir, 1980)

41. Chevron, Inc. v. Natural Resources Defense Council 467, U.S. 837

42. Industrial Union Department, AFL-CIO v. Amer. Petroleum Institute, 448 U.S. 607 at 656

43. ASARCO v. OSHA 746 F.2d 483

44. 48 Fed. Reg. 1864, part III

45. 48 Fed. Reg. at 1866

46. Industrial Union Dept. v. Amer. Petroleum Inst. 448 U.S. 607, 655

47. ASARCO v. OSHA 746 F. 2d at 491

48. 49 Fed. Reg. 25737

49. Public Citizen Health Research Group v. Tyson 796 F. 2d 1479, 1483 (D.C., Cir., 1986)

50. Id.

51. Id at 1487

52. Industrial Union Department, AFL-CIO v. Amer. Petroleum Institute, 448 U.S. 607 at 625

53. Public Citizen Health Research Group v. Tyson 796 F. 2d 1489

54. Id

55. Id at 1499

56. Id at 1502

57. 49 Fed. Reg. 25734, (1984)

58. Public Citizen Health Research Group at 1505.

59. Public Citizen Health Research Group v. Tyson 796 F. 2d at 1505

60. Id

61. ASARCO v. OSHA 746 F.2d at 492

62. Id

63. Id. at 493

64. 56 Fed. Reg. 3, 526 (1991)

65. Intl. Fabricare Inst. v. US EPA 972 F. 2d 384 (D.C. Cir., 1992)

66. Id at 391

67. Bruce N. Ames and Lois Swirsky Gold, Pesticide, Risk, and Applesauce 244 Science 755 (May 19, 1989) (quoting Intl. Fabricare v. US EPA at p. 391)

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72. L. Tomatis, "Environmental Cancer Risk Factors: A Review," Acta Oncologica 27 (1988): 465-472 (quoting Carl F. Cranor, Regulating Toxic Substances (1993), at page 16)

73. Motor Vehicle Mfrs. Ass'n of the United States, Inc. v. State Farm Mut. Auto Co., 463 U.S. 29

74. Intl. Fabricare v. US EPA at 394

75. Intl. Fabricare v. US EPA

76. Id at 393

77. 62 Fed. Reg. 38856

78. Id at sec. 7409 (b)

79. American Trucking v. United States Environmental Protection Agency, 175 F. 3d 1027

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81. Intl. Fabricare v. US EPA Id at 393

82. S. Breyer, Breaking the Vicious Cycle, 1993.

83. Intl. Fabricare v. US EPA at 39?

84. Intl. Fabricare v. US EPA Id at 393

85. Intl. Fabricare Inst. v. US EPA at 398-399

86. Final Rule, 56 Fed Reg. at 3551

87. Proposed Rule Fed. Reg. at 22,099-100

88. Id.

89. 42 USC sec 300g-1 (3)(A)

90. Community Nutrition Inst. V. Block, 749 F. 2d 50, 57-58 (D.C. Cir., 1984)

91. Stephen Breyer, Breaking the Vicious Cycle (1989)

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100. Marcia Angell, Science on Trial (1997)

101. Stephen Breyer, Breaking the Vicious Cycle (1989) at 72-70.