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THE EVOLUTION OF FEDERAL PESTICIDE REGULATORY POLICY INVOLVING PUBLIC PARTICIPATION

By

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ABSTRACT

The Evolution of Federal Pesticide Regulatory Policy Involving Public Participation

by

Eileen R. Choffnes

An active area for public participation and environmental litigation over the last 12-14 years has been, and continues to be, the regulation of pesticides by the EPA. Public interest/advocacy organizations have played a significant part in the political decisions to regulate problem pesticides. Through the use of adversarial procedures they have shed light on the value judgments and political trade-offs inherent in these controversies.

On Thursday, August 7, 1980, the U.S. EPA published a rulemaking proposal which would have significantly amended the procedures used for conducting adjudicatory hearings under section 6 of the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA). Through the statements advanced in this proposed rulemaking the EPA sought to achieve an administrative resolution to what is fundamentally a polycentric controversy by infusing a managerial decisionmaking model into the realm of public policy formulation.

This study was undertaken to explore the institutional framework within which pesticide policy is made. During the course of this investigation it became clear that the proposed rulemaking discussed

herein was of questionable legality from the standpoint of the statutory language of FIFRA and the case precedents. It was hoped that this analysis would influence the EPA's regulatory interpretation of its affirmative duties under FIFRA.

On July 22, 1981, the EPA withdrew the rulemaking proposal which is the subject of this study. It is unclear whether the Agency will repropose this rule at some future data, but this study demonstrates some of the problems involved with public participation in pesticide decisionmaking.

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INTRODUCTION

> The rise of administrative bodies probably has been the most significant legal trend of the last century and perhaps more values today are affected by their decisions than by those of all the courts, review of administrative decisions apart. They also have begun to have important consequences on personal rights. . . They have become a veritable fourth branch of government, which has deranged our three-branch legal theories much as the concept of a fourth dimension unsettles our three-dimensional thinking.
>
> Courts have differed in assigning a place to these seemingly necessary bodies in our constitutional system. Administrative agencies have been called quasi-legislative, quasi-executive or quasi-judicial, as the occasion required, in order to validate their functions within the separation-of-powers scheme of the Constitution. The mere retreat to the qualifying "quasi" is implicit with confession that all recognized classifications have broken down, and "quasi" is a smooth cover which we draw over our confusion as we might use a counterpane to conceal a disordered bed.[1]

Since World War II, pesticide production and use has increased enormously. "The United States uses about 1 billion pounds of pesticides annually to control insects, diseases, rodents, weeds, bacteria and other pests that attack our food and fiber supplies and threaten our health and welfare."[2] The pesticide industry is thus very big business indeed "with current annual sales in the region of $4 billion."[3] While the use of pesticides has unquestionably contributed to the health, welfare and comfort of man's material needs, they are a mixed blessing.[4] They represent one of the most important classes of general environmental pollutants and their use results in massive, involuntary human and environmental exposure and contamination.

With the publication of the book Silent Spring[5], in 1962, Rachel Carson focussed public attention on the problems created by the

injudicious and indiscriminate use of pesticides. Carson summarized her position as follows:

> It is not my contention that [pesticides] must never be used. I do contend that we have put poisonous and biologically potent chemicals indiscriminately into the hands of persons largely or wholly ignorant of their potentials for harm. We have subjected enormous numbers of people to contact with these poisons, without their consent and often without their knowledge. If the Bill of Rights contains no guarantee that a citizen shall be secure against lethal poisons distributed either by private individuals or by public officials, it is surely only because our forefathers, despite their considerable wisdom and foresight, could conceive of no such problem.
>
> ...(F)urthermore,...we have allowed these chemicals to be used with little or no advance investigation of their effect on soil, water, wildlife, and man himself. Future generations are unlikely to condone our lack of prudent concern for the natural world that supports life.[6]

Former President Nixon, in late 1970[7], transferred principal authority for the regulation of pesticides from the United States Department of Agriculture (USDA) to the newly created Environmental Protection Agency (EPA). Earlier that year the USDA had been criticized, by Congress, in its traditional handling of pesticide regulation. Its activities in the area of pesticide safety were called "scandalously derelict"[8], in part because "farm groups, food producers, and the manufacturers of agricultural chemicals [were] strongly represented in USDA..."[9] By contrast the EPA, it was assumed, given its presidential mandate to "ensure the protection. . .and enhancement of the total environment"[10] would not be so biased towards the interests of those whose activities it sought to regulate.

An active area for public participation and environmental litigation over the last 12-14 years has been and continues to be the regulation of pesticides by the EPA. Perhaps the most controversial litigation was the first (successful) action by a public interest

organization to "ban" DDT. Starting in 1966, in Long Island, New York and terminating seven years later in the U.S. Court of Appeals for the District of Columbia, the DDT case, brought by the Environmental Defense Fund (EDF), established many important legal precedents including the standing of citizens groups to sue government agencies, as well as judicial review of government agency actions and inactions.

On Thursday, August 7, 1980,[11] the U.S. EPA published a proposed rulemaking in the Federal Register which would significantly amend the procedures currently used for conducting adjudicatory hearings under Section 6 of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). In the preamble to the proposed rulemaking, the EPA stated that, "(t)he objective of [this] proposal is to make the Agency's procedures for identifying and assessing potential problem pesticides, and making regulatory decisions. . .more open, responsive and efficient. The fundamental theme...is to create (a)...system in which...adjudicatory hearings are utilized...to probe and challenge decisions reached..."[12] While the Agency claimed that "these changes [were] designed to enhance public participation in the process"[13] in fact the ultimate effect of these proposed rules would have been to make public participation a hollow exercise in the formulation of pesticide regulatory policy "with the Agency internalizing most if not all of the critical value judgments involved."[14]

Through the proposals put forward in this proposed rulemaking the Agency sought to achieve an administrative resolution to what is really a polycentric controversy by infusing a managerial decision making model into the realm of public policy formulation.

The regulation of pesticides, like the regulation of other toxic/harmful substances, is fundamentally a trans-scientific process. In an ideal world one would like to have complete knowledge on the beneficial and non-beneficial outcomes derived from the use or non-use of a particular pesticidal agent before making a regulatory decision. Yet often the information available to a decisionmaker is, at best, incomplete or speculative. "Lacking scientific evidence the agencies are stuck with visceral estimates and political accomodations as the only basis for policy."[15] Thus, any policy decision requires a balancing between the scientific "facts," on the one hand, and the value biases of the decision-maker on the other.

Certain analytical techniques, among them cost-benefit analysis, risk-benefit analysis, and risk-risk analysis, have been employed by various regulatory agencies in an attempt to "logically" and "rationally" regulate potentially biohazardous agents. Cost-benefit analysis "refers to the systematic analysis and evaluation of alternative courses of action drawing upon the analytical tools and insights provided by economics and decision theory. It is a framework and a set of procedures to help organize the available information, display trade-offs, and point out uncertainties."[16] Risk-benefit analysis is a somewhat vague process which requires one to weigh the risks of a given activity against its social benefit(s). Full quantification and valuation under the risk-benefit framework is left to the expertise of the decision-maker.[17] The risk-risk framework "allows beneficial health effects to be considered along with adverse health effects."[18]

Although not formally required to under FIFRA, EPA has utilized a cost-benefit approach in its regulation of problem pesticides. While this approach provides a logical and systematic format for a decision-maker to follow in reaching a decision it has certain limitations:

> The most important and pervasive limitation on benefit-cost analysis is the role of values. Many of the factors that are likely to be most significant in a decision concerning toxic chemicals cannot be measured in common terms (such as dollars) that are agreeable to all concerned parties. Different individuals place different values on things... Thus, an analysis that assigns a quantitative value to one or more of these factors is necessarily subjective and, to some degree, arbitrary.[19]

Even larger problems are created by the distribution of benefits and costs over time. The value of future costs, in traditioal benefit-cost analysis, is reduced by the use of a discount rate. This discounting technique has often been used by regulators to minimize the intergenerational effects of chronic exposures to toxic substances.[20] For, where there exist,

> . . .complex problems involving large numbers of interested parties the concept of a single best solution is misleading. Quantitative techniques of decision making are of great value in solving many problems; however, they offer little prospect of serving as an impartial, irrefutable arbiter of the conflicts of interest involved in large policy problems.[21]

This study was undertaken to explore the institutional framework within which pesticide policy is made. During the course of this investigation it became clear that the proposed rulemaking dicussed herein was of questionable legality--both from the standpoint of the statutory authority of FIFRA and legal precedent. It was hoped that this analysis would influence the EPA's regulatory interpretation of its affirmative duties under FIFRA. On July 22, 1981, the EPA withdrew the

rulemaking proposal which is the subject of this study. It is unclear whether the Agency will repropose this rulemaking at some future date.

The discussion of pesticide regulation and the role of the public in the formulation of pesticide regulatory policy will consist of three parts. Part I will be concerned with the history of pest control and the evolution of pesticide laws in the United States. The second part relates to the regulatory framework used by EPA in implementing the Congressional mandates of the FIFRA. Finally, the role of public interest organizations in the formulation of pesticide regulatory policy will be narrowly discussed in terms of environmental litigation used to catalyze agency action and more broadly in terms of equity considerations and distributive justice.

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CHAPTER 1

History of Pest Control

> The evidence is abundant that with the single strategy of chemical control we not only have saturated the environment with deadly poisons that endanger a wide spectrum of living organisms, including man himself, but that we have begun to disrupt seriously the economic stability of the farming community, with disaster approaching if we follow our present course.[1]

The definition of a "pest" is wholly anthropocentric. In general a pest is any organism which reduces the availability, quality, or value of some human resource. The resource may be a plant or animal grown for food, fiber or pleasure. The resource may also be a person's health, well-being or peace of mind--any or all of which may be threatened from time to time by allergy-inducing or otherwise bothersome plants, disease-vectoring organisms, or biting, stinging and nuisance-type animals.[2] Therefore, any insect, weed, fungus, bacteria, rodent, virus, bird, etc., which competes with man's agricultural or domestic activities can be classified at any time as a pest. Once an organism has been identified as a "pest" every effort is employed to control or eradicate its depredation of man's resources. Often these control strategies bear a striking resemblance to "seek and destroy" missions.

For thousands of years, man could do nothing about pests but appeal to the power of magic and a variety of gods. Early humans, for the most part, had to live with and tolerate the ravages of insects and plant diseases. Through "trial and error" experiments, they gradually learned how to improve their lot.

Before 2500 B.C., the Sumerians had learned to use sulfur as an insecticide. The ancient Chinese used arsenic and mercury compounds, and the Romans used arsenic, to kill insects.[3] In the 19th century pesticide use expanded dramatically with the discovery of the insecticidal and fungicidal properties of Paris Green (copper aceto-arsenite) and with the finding that Bordeaux mixture (hydrated lime and copper sulfate) prevented powdery mildew infestation of grapes.

> Before 1900, the aim of pest control was to exterminate the pest by any means short of destroying the crop. For example, lead arsenate was used in large quantities for insect control, and it was common to observe fruits and vegetables for sale that were "powder white" with residues.[4]

During the 19th and 20th centuries a number of other inorganic compounds were tried as insecticides and fungicides but were not very effective. These included compounds of antimony, boron, copper, fluorine, manganese, mercury, selenium, sulfur, thallium and zinc. Chemical weed control became possible at the end of the 19th century when iron sulfate was found to kill broadleafed weeds but not cereal crops. It was more economical, however, to continue using hand weeding, tillage, and crop rotations with plants that could compete with weeds for the available sunlight, nutrients, and water, and clean cultivations to keep weed densities at manageable levels. Herbicides were not widely used until the second half of the 20th century.[5] Insecticide use, generally, was restricted to certain fruits and "high cash" crops until the mid-1960's.

Some of the early pesticides were recognized as poisons. France banned the practice of soaking food crop seeds in mercury and arsenic in 1786. Harmful effects other than acute poisoning were known. European women often used arsenic-impregnated pastes to whiten their skins in the

18th and 19th centuries even though arsenic was long recognized as a poison. The persistence of these chemicals also went unnoticed, though their accumulation sometimes caused crop damage. It is now known that the inorganic pesticides can remain in soils for up to 40 years, and many orchard soils contain large amounts of them today.

Synthetic organic chemical pesticides were developed by the U.S. government during World War II for use against insects that were vectors for diseases such as malaria and typhus. After the war they became widely available to farmers who saw in them an end to crop losses due to insects.[6]

The organochlorine compounds, especially DDT, were apparently "ideal." They were effective against a wide variety of insect pests at application rates that were extremely low compared to the amounts that had a known, immediate effect on humans and domestic livestock. Their persistence in the soil after use contributed to their popularity since pesticides belonging to this class did not have to be applied repeatedly during the growing season.

At first, residual traces of these chemicals found in foods and occasional fish kills after a body of water had been treated were generally accepted as mild side effects accompanying the desired insect control. Concern for the ecological effects of widespread pesticide use began to grow, however, after various studies demonstrated the almost universal presence of the organc lorine insecticides in the global environment. Increasing numbers of wildlife were dying, and the organochlorines were reaching high concentration in certain mammalian tissues. Measurable quantities could also be detected in the air, in stream beds, and even in the polar regions of the Earth.

Investigation of the high levels of DDT in animals not directly exposed to the insecticide led to an appreciation for the phenomenon of bioaccumulation. Like the chemically related PCBs, DDT is resistant to degradation. Having a high affinity for lipids it tends to collect in the fatty tissues of animals, where it can become much more concentrated than it is in the general environment. Predators feeding on animals in which this concentrating process has occurred--for instance, birds feeding on insects, worms, or fish--may be exposed to dangerous levels of the insecticide.

One of the more publicized effects of DDT has been the reduction of reproductive fitness in many species of birds resulting in declining bird populations. The organochlorine insecticides have also been detected in cow and human milk samples. A recent Environmental Protection Agency study (1978) found traces of organochlorine compounds in all milk samples tested. In addition, the ability of these insecticides to collect in animal tissues gives these compounds another mode of transportation, besides wind and water, to locations where they were not originally applied.

It was eventually confirmed in the late 1960's, that the organochlorine insecticides have the ability to induce cancer in laboratory animals and are, therefore, presumptive human carcinogens.[7] Stored in the fatty tissues of the liver, they can cause liver enlargement and interf re with the liver's ability to detoxify substances with which it comes in contact.[8]

The organochlorines also have declining efficacy against insects, since these organisms have a profound capability of developing physical and chemical mechanisms to protect themselves against almost any toxic

substance challenge. Hal Gordon, of the University of California (Berkeley)[9] observed, in 1961, that those species of insect larvae which fed on a variety of plants were tolerant to many insecticides. Such insects were found to possess a multi-purpose enzymatic degradation system called mixed function oxidases (MFO's). An individual insect may (under normal circumstances) have a low MFO level--when the insect comes in contact with an insecticide its MFO level rises. Thus, an insect with a high enough MFO level may be able to survive insecticide challenge and give rise to progeny which are more insecticide resistant than the parent generation. In this way, insects have developed a remarkably inducible system for neutralizing toxic compounds. The usual response of the grower, when confronted with insect resistance to a given insecticide, has been to apply greater and greater quantities of insecticide with little or no additional control achieved in the process (this situation is the classic example of the "Pesticide Treadmill"). While not eradicating the insect pest, the massive doses of insecticides have a profound effect upon beneficial organisms--often eliminating organisms responsible for plant pollination, soil aeration and waste degradation. The resultant reduction of beneficial organisms which prey upon insect pests (killed by insecticides) allows for pest population explosions and the emergence of new insect pests, requiring both more extensive and expensive control measures.[10]

Concerns about the oncogenic (cancer inducing) effects of the organochlorine insecticides led to the cancellations of most of their registered uses. DDT, aldrin/dieldrin, Kepone(R), mirex, chlordane/heptachlor and endrin can no longer be intensively used in the U.S. Their popularity, at the time regulatory actions were taken

against them, was declining anyway due to the resistance of numerous insect complexes to them.

To meet the challenge presented by insect resistance to and the persistance of organochlorines, the agri-chemical industry began to exploit other classes of insecticidal compounds--the organophosphates and the carbamates. These compounds are considered "soft" chemicals; they contain chemical groups which readily decompose under a variety of environmental conditions.

However, these compounds are acutely toxic to humans, attacking the nervous system of those exposed. Thousands of people are sickened and many die each year as a result of poisoning caused by contact with them.[11]

Some insects have also developed resistance to these insecticides as well, although resistance is not as wide-spread as it is to the organochlorines.

> (D)espite the dramatic increases in chemical pesticides during the past 30(+) years, average annual crop losses due to combined pest categories (i.e., insects, diseases, nematodes and weeds) have apparently not decreased; and there are indications that percentage crop losses attributed to insect damage have increased as much as two-fold during this period. There are a number of reasons for this.
>
> . . .(T)here are presently over 300 species of insects, mites and ticks that posses strains resistant to one or more chemical pesticides. . . (W)eed control literature is full of cases in which control is said to be "difficult" because of decreasing effectiveness of the herbicides, and many studies have shown the existence of weed strains relatively resistant to various herbicides.[12]

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CHAPTER 2

History of U.S. Pesticide Legislation

The federal regulation of pesticidal products began in 1910, when Congress passed the Federal Insecticide Act.[1] The purpose of this legislation was twofold: (1) to protect consumers from purchasing ineffective pesticide products, and (2) to protect consumers from purchasing pesticide products with deceptive/fradulent labelling claims.[2] Under the Act "[a] product was misbranded if its label was false and misleading or the package failed to contain the ingredients listed on the package label."[3] While the Insecticide Act of 1910 attempted to regulate the manufacture, sale and transshipment of pesticide products sold interstate it had no effect upon the intrastate sale of ineffective products. In as much as registration of pesticide products sold in interstate commerce was not required there was no mechanism available for the enforcement of infractions of the statutory mandates.

The manufacture of pesticides prior to World War II largely consisted of inorganic products such as calcium arsenate, lead arsenate, Paris Green, copper sulfate, fluorine compounds and ground sulfur, as well as, botanical extracts including pyrethrum dusts and extracts, rotenone dust and nicotine sulfate.[4] By the end of World War II there was a virtual "explosion" of activity synthesizing organic chemical compounds for general sale and use as pesticides. DDT was being heralded as a "miracle" insecticide that would be the ultimate solution to mankind's insect problems. As a result of the rapid expansion of the pesticide industry and the widespread distribution of these so-called

"economic poisons" Congress, in 1947, enacted the original Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).[5]

The FIFRA augmented the Insecticides Act's basic provisions. The fundamental goal of the legislation was the protection of the general public from personal and economic injury, taking into account not only the purchase of products subject to the dictates of the Act but all persons who might come into direct or indirect contact with pesticides or materials which could have been treated with them. The statute provided for the protection of users from unsafe products[6] and required that, before a pesticide product could be distributed and marketed in interstate commerce, it had to be registered with the United States Department of Agriculture (USDA). As employed in the Act, the term "pesticide" has often been substituted for the statute's use of the term "economic poison" which was defined as:

> (1) any substance or mixture of substances intended for preventing, destroying, repelling, or mitigating any insects, rodents, nematodes, fungi, weeds, and other forms of plant or animal life or viruses, except viruses on or in living man or other animals, which the Secretary shall declare to be a pest, and (2) any subtance or mixture of substances intended for use as a plant regulator, defoliant or desiccant.[7]

In passing the statute Congress intended to prevent or at least discourage misleading or false advertising claims, prevent inefficacious products from being marketed, and provide a legal remedy for the unsuspecting purchaser if such products were marketed.[8] With the statutory prohibition against ineffective products[9] Congress perpetuated, in FIFRA, the original orientation of the Insecticide Act.

The registration requirement was created as an aid in enforcement. "Registration [was] viewed as a means of notifying the Secretary of Agricuture of (1) the products being marketed; (2) their composition;

and, (3) the claims made for them."[10] FIFRA provided for seizures of pesticide products in situations where they were adulterated, misbranded, unregistered or insufficiently labeled, or when such devices were misbranded.[11] Manufacturers requesting registration were given an opportunity to modify their proposed product label if it failed to comply with any of the statutory requirements including: (1) the prominent display of poison warnings, (2) the inclusion of warning statements on the package label, and, (3) clear and concise instructions for proper product use.[12] Yet, even when such label changes were not made by the registration proponent, the USDA was obliged to register the product "under protest."[13] The Secretary of Agriculture was authorized to cancel a pesticide's registration and substitute in its place a protest registration "to protect the public"[14], yet the statute was noticeably silent on the precise definition of the standard and what criteria were to be applied by the Secretary in implementing and enforcing the Congressional intent of the standard. So far as the Act's product safety provisions were concerned all that was actually required was that the product be "safe" (i.e., low acute toxicity) when used according to the lable directions.[15]

Congress, in 1959, passed the Nematocide, Plant Regulator, Defoliant and Desiccant Amendment[16] to FIFRA. The 1959 amendments reflected the additional classes of pesticidal materials which had been developed and marketed since 1947. Congress felt that these materials should be subjected to the same registration requirements as those previously registered under the 1947 FIFRA.

In 1962, Congress modified the regulations to expand the definition of a "pest." Under the regulations materials used to repel birds,

reptiles, predatory animals, certain classes of fish, plant diseases, and weeds were brought under USDA/FIFRA control. As a result approximately 2,000 more products, produced by approximately 800 establishments, were subject to USDA inspection.[17]

FIFRA was once again amended by Congress in 1964.[18] Under the new amendments, the Secretary of Agriculture was given the authority to refuse to register a new product[19], thereby eliminating "protest" registrations. Moreover, the Secretary was given the authority to remove a pesticide product from interstate commerce if its safety and/or efficacy was of dubious validity.[20] A pesticide was defined as misbranded if, when used in accordance with the manufacturer's label directions, it was injurious to human or animal health. The amendments also authorized the Secretary to suspend a pesticide's registration if it was found to constitute an "imminent hazard to the public." Be that as it may, it was not until 5 years later that the Secretary of Agriculture published the first USDA regulations specifying what standards were to be applied by USDA in defining an "imminent hazard".[21]

Pesticide regulation, since 1947, had been plagued with difficulties almost from its inception. USDA, under FIFRA, was required by law to register pesticides sold in interstate commerce. However, it could not register a pesticide which left a residue in or on raw agricultural products unless the Food and Drug Administration (FDA) under the Federal Food, Drug, and Cosmetics Act (FFDCA) was able to establish a "safe" tolerance level. If the FDA determined that any residue was dangerous to human health it could refuse to grant a tolerance for that particular pesticide use. Without an established tolerance the USDA was prohibited from registering a pesticide for a particular use.

This "balkanization" of regulatory agency authority over pesticide registration decisions was, not surprizingly, detrimental to the efficient formulation of pesticide policy. Often the missions of USDA and FDA were in direct conflict with one another. Historically, USDA's mission had "been to promote food production and, therefore, [it] heavily promoted the use of pesticides. . .(l)ong-term health concerns were often subordinate to [its] primary goal. FDA, on the other hand, was charged with safeguarding the public health."[22]

In order to more effectively and efficiently manage pesticide regulation President Nixon, in 1970, proposed a reorganization of all federal governmental agencies having authority over pesticides. The United States Environmental Protection Agency (EPA) was created in December, 1970.[23] Among other things, EPA was given the exclusive responsiblity of administering and enforcing FIFRA. As the "Kennedy Report" staff pointed out, though,

> (a)ssigning all pesticide regulation to one agency helped (to) alleviate bureaucratic overlap and administrative inefficiency; but. . .did not cure the basic conflict. . . (M)any of the personnel and much of the philosophy from USDA were simply transferred to the EPA pesticide program in 1970.[24]

After extensive hearings and under intense lobbying from public interest organizations, trade associations and the pesticide industry Congress greatly expanded EPA's pesticide regulatory task with the enactment of the Federal Environmental Pesticide Control Act (FEPCA) in 1972.[25]

> In addition to requiring the processing of new pesticide registration applications and pesticide residue tolerance petitions, FEPCA required that all pesticide products previously registered over the past 30 years--including some 35,000 federally by USDA and by EPA since 1970, and 15,000 by states--[be] reviewed and subject to a reregistration process, and classified for either general or restricted

> use, or both. This task was directed by Congress to be accomplished by October 21, 1976. The intent of Congress...was to subject those pesticides approved under earlier, less stringent safety standards to application of much improved modern standards. Under FEPCA, Congress placed on EPA the responsibility to determine whether any of these pesticides "will perform [their] intended function without unreasonable adverse effects"[26] on human health and on the environment. If, after an examination of the risks and benefits, the Administrator...determines that the pesticide causes "unreasonable adverse effects," he may restrict, suspend, or cancel the use of the pesticide.[27]

FEPCA defined "unreasonable adverse effects on the environment" to mean "any unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide."[28] As required by this definition the balancing of risks and benefits is of particular importance with respect to four principal types of decisions which the Administrator must make: registration, classification, cancellation, and suspension.

> APPROVAL OF REGISTRATION - The Administrator shall register a pesticide if he determines that, when considered with any restrictions imposed under subsection (d) [classification]--
>
> (A) its composition is such as to warrant the proposed claims for it;
>
> (B) its labeling and other material required to be submitted comply with requirements of this Act;
>
> (C) it will perform its intended function without unreasonable adverse effects on the environment; and
>
> (D) when used in accordance with widespread and commonly recognized practice it will not generally cause unreasonable adverse effects on the environment.[29]
>
> CANCELLATION and CHANGE IN CLASSIFICATION - If it appears to the Administrator that a pesticide or its labeling or other material required to be submitted does not comply with the provisions of this subchapter, or when used in accordance with widespread and commonly recognized practice, generally causes unreasonable adverse effects on the environment, the Administrator may issue a notice of his intent either--

> (1) to cancel registration or change its classification...or,
>
> (2) to hold a hearing to determine whether or not its registration should be cancelled or its classification changed.[30]
>
> ORDER--If the Administrator determines that action is necessary to prevent an imminent hazard during the time required for cancellation or change in classification proceedings he may, by order, suspend the registration of the pesticide immediately.[31]

The statute then goes on to define an "imminent hazard" as "a situation which exists when the continued use of a pesticide during the time required for cancellation proceeding [sic] would be likely to result in unreasonable effects on the environment or will involve unreasonable hazard to the survival of a species declared endangered by the Secretary of the Interior under Public Law 91-135 (the Endangered Species Act)."[32]

Thus the Administrator was given the authority to prohibit the further sale and use of a pesticide product if he determined that the product caused "unreasonable adverse effects" on the environment. Upon the issuance of a notice of intent to cancel[33] a registration the registrant could request a formal adjudicatory hearing on the merits. Alternatively, the Administrator could issue a notice of his intent to hold a hearing in order "to determine whether or not [the pesticide's] registration should be cancelled."[34] It is quite clear from the legislative history of the Act that the purpose behind the inclusion of this alternative hearing procedure was to allow the Administrator to "initiate formal review without placing a stigma on the product when h. is not convinced that the registration should be cancelled."[35]

During either type of formal hearing the pesticide may continue to be produced and marketed. Some of these hearings may take years to complete. For example,

> Eighty-six registrants requested a hearing as a result of a notice of cancellation issued on March 18, 1971, for the major pesticides aldrin and dieldrin. Shell, sole manufacturer of these pesticides, played the primary role in contesting the cancellation notice. The hearing continued for twelve months and resulted in a transcript of more than ten thousand pages in addition to many thousands of pages of exhibits.[36]

Should the Administrator discover, after he has issued a notice of intent to cancel,[37] that the continued use of the suspect pesticide will present an "imminent hazard to the public" during the time required to complete the cancellation hearings he can issue a notice of intent to suspend the pesticide's registration. A suspension notice, like a temporary injunction, has the practical effect of removing suspect pesticidal products from the marketplace pending the resolution of the cancellation hearings process. Under this procedure, as with the notice of intent to cancel, registrants are entitled to a formal adjudicatory hearing on the narrow question of an imminent hazard. By law this must be an expedited hearing.[38]

FEPCA's definition of "imminent hazard" has been interpreted by the courts to mean a "substantial likelihood that serious harm will be experienced during the year or two required in any realistic projection of the administrative [cancellation] process."[39] Moreover, "the function of the suspension decision is to make a preliminary assessment of evidence and probabilities, not an ultimate resolution of conflicting issues."[40]

In both types of proceedings, (cancellation and suspension), the statute and case law place the "burden of establishing the safety of a product requisite for compliance with the labeling requirements...squarely at all times on the applicant and registrant."[41] For the Administrator to survive a court challenge to his decision

regarding cancellations and suspensions, "(i)t [is] enough...that the administrative record [contain] respectable scientific authority supporting the Administrator."[42]

Rebuttable Presumption Against Registration/Reregistration

On July 3, 1975, the EPA published in the Federal Register[43] regulations covering the registration, reregistration, and classification of pesticides mandated by FEPCA. (It must be kept in mind that FEPCA was passed in 1972, and Congress mandated that by October, 1976, approximately 50,000 pesticides which had been previously registered by USDA on efficacy considerations were to be reevaluated on the basis of human and environmental health hazards.) The regulations specified the types of data which registrants were required to supply to EPA in order for them to retain or receive a product registration[44]. They also established criteria for classifying a pesticide for general or restricted use, and criteria for denying or cancelling registrations on the basis of acute toxicity. As a practical matter the 1975 regulations were, in reality, a codification of the toxicological principles established during the DDT, aldrin/dieldrin, and heptachlor/chlordane cancellation/suspension hearings.

The regulations defined acute toxicity as "the property of a substance. . .to cause adverse effects in an organism through a single short-term exposure."[45] A pesticide was chronically toxic if it,

> (A) Induces oncogenic effects in experimental mammalian species or in man as a result of oral, inhalation or dermal exposure; or induces mutagenic effects by multitest evidence.
>
> (B) Produces any other chronic or delayed toxic effect in test animals at any dosage up to a level, as determined by the Administrator, which is substantially higher than that

> to which humans can reasonably be anticipated to be exposed, taking into account ample margins of safety; or,
>
> (C) Can reasonably be anticipated to result in significant local, regional, or national population reductions in nontarget organisms, or fatality to members of endangered species.[46]

"Mutagenic" refers to the capacity of a pesticidal compound to induce genetic changes in the offspring of exposed parents.[47]

The Administrator, in the preamble to these regulations, stated his rationale for using animal test data as a predictor for the potential of a compound to cause adverse health effects in man.

> (T)he use of animal test data to evaluate human cancer risks has been widely accepted by the scientific community and by public policymaking agencies... (B)ecause of [the] inherent limitations of animal testing 'a substance that will induce cancer in experimental animals at any dose level, no matter how high or low, should be treated with great caution'...(N)egative results are of limited value and although a no-effect level may theoretically exist, it is frequently impossible to establish with sufficient confidence to justify sanctioning widespread exposure.
>
> Moreover, the term "oncogenic" is used in the regulations because the Administrator (has) determined that the distinction between "benign" and "malignant" tumors is not meaningful in determining the hazard of cancer to man on the basis of tests conducted on a laboratory species, given the "increasing evidence that many tumors can develop into cancers." "...(F)or the purpose of carcinogenicity testing, they should be considered synonymous."[48]

A rebuttable presumption against registration (RPAR) arises when, after the Agency evaluates information submitted to it by the proponent of registration, it finds that any one of three criteria, indicative of unreasonable adverse effects, is met or exceeded: acute toxicity; chronic toxicity; or lack of emergency treatments in man to ameliorate the toxic effects resulting from a single exposure. These criteria represent "indicators of potential hazard";[49] the pesticide will be presumed to pose an unreasonable risk once any one of the three criteria

is met or exceeded. Once triggered, an applicant is notified of the presumption against registration and is given an opportunity to submit rebuttal evidence.[50]

At all times the ultimate burden of persuasion regarding the safety of a pesticide lies with the proponent of registration. With respect to an RPAR, the burden of going forward means, "the necessity of producing evidence. . .of a particular fact in issue."[51] The evidence "must be such that a reasonable man could draw from it the inference of the existence of a particular fact to be proved."[52] To successfully rebut a presumption against registration, the proponent must ultimately prove that its product does not pose unreasonable adverse effects on the environment[53] or that, in spite of the risks presented by the pesticide, the benefits of continued use justify its continued registration.[54]

One writer has observed that,

> (t)he decision whether a presumption against registration has been successfully rebutted is a difficult one. By raising the presumption the Agency indicates its belief that there is a high probability of significant danger, whereas the manufacturer's attempted rebuttal is an indication of its belief that the hazard is less significant than the initial tests suggest. The decision is thus likely to require a complex evaluation based upon competing and perhaps equally tenable, explanations of the data.[53]

If the proponent of registration produces information of sufficient weight and validity to rebut the presumption against his product the Administrator may overturn his original decision. Were this to happen, the presumption would be said to be rebutted. If, however, a substantial question of safety were still found to exist, the 1975 regulations required the initiation of formal adjudicatory proceedings under FEPCA.

In the fall of 1975, the Administrator of EPA (Train) recommended procedural modifications to the way the Agency was implementing FIFRA. The central theme of his memorandum "was to de-emphasize the role of FIFRA adjudicatory hearings in decisionmaking about problem pesticides, and institute a system in which the Agency would make pesticide decisions prior to hearings. . . ."[56]

> With regard to cancellation and suspension decisions, I believe that misconceptions by the public are attributable, in part, to our reliance on the adversary hearing process to ensure that all pertinent facts are brought out. While these procedures have been effective, they have inhibited full participation by the Office of Pesticide Programs in the decision process [many of whom came from USDA] and have restricted effective public involvement in this aspect of the program. I have determined that the Agency should carry out a more open evaluation of risks and benefits in advance of decisions to issue notices of intent to cancel or suspend.[57]

Train wrote the memorandum in an apparent response to the proposed 1975 FIFRA amendments contained in H.R. 8841 (later to become P.L. 94-140).

As enacted, the 1975 FIFRA amendments imposed significant constraints upon EPA's pesticide regulatory authority. Before the Administrator could issue a public notice of his intent to cancel a registration, intent to hold a hearing to determine whether a registration should be cancelled, or change a classification, he had to first, at least 60 days prior to submitting the notice to the registrant, submit a copy of the notice to the Secretary of Agriculture and a newly created Scientific Advisory Panel[58] (SAP) for comment.[59] Both the Secretary of Agriculture and the SAP were given a 30 day comment period to respond to EPA's proposed notice.[60] After this 30 day period, the Administrator could publish the notice plus any comments received from the Secretary and the SAP, including EPA's reply, in the Federal Register.[61] Furthermore, all regulations, both proposed and

final, were to be provided to the Secretary of Agriculture, the House Committee on Agriculture, the Senate Committee on Agriculture and Forestry and the SAP for comment.[62] Finally, the Agency was required to take into account, in any proposed action, the impact on production and prices of agricultural commodities, retail food prices and the agricultural economy.[63]

Several provisions of the Federal Pesticide Act of 1978,[64] enacted September 30, 1978, pertain to the RPAR process and the Agency's Rules of Practice for cancellation hearings. A new section entitled "Interim Administrative Review", imposed new requirements on the Agency concerning the quality of information which must be available to the Administrator before an RPAR may be issued.

> . . .the Adminstrator may not initiate a public interim administrative review process to develop a risk-benefit evaluation of the ingredients of a pesticide or any of its uses prior to initiating a formal action to cancel, suspend or deny registration of such pesticide, . . .unless such interim administrative process is based on a validated test or other significant evidence raising prudent concerns of unreasonable adverse risk to man or to the environment.[65]

> The Conference Report defined "validated test" as. . .a test conducted and evaluated in a manner consistent with accepted scientific procedures, and that the term "other significant evidence" be defined as evidence that relates to the uses of a pesticide and their adverse risk to man or to the environment. It is the intent of the conferees that "other significant evidence" of adverse risk means factually significant information and is not to include evidence based only on misuse of the pesticide.[66]

It is also noteworthy that the Conference Report without amending the statute, addressed modifications in the RPAR process:

> To avoid the time-consuming RPAR process if [in the Administrator's judgement] humans or their environment are not at risk or the potential for exposure is minimal, the Administrtor is directed to forego the RPAR process and proceed with the reregistration of the pesticide product.[67]

The guidance of the conferee's, if followed faithfully by the Agency, will have far-reaching consequences for pesticides which are presumptive carcinogenic and mutagenic agents. Instead of triggering an RPAR, the Agency will be compelled to take exposure into account. The implicit assumption, should this come to pass, is that there exists some putative threshold exposure level for carcinogenic/mutagenic agents or that there may be some arbitrary level in the increased rate of cancer or mutations, (due to exposure to these toxic compounds), that is "socially acceptable" to the general puplic.

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CHAPTER 3

PUBLIC PARTICIPATION IN TRANS-SCIENTIFIC PESTICIDE DECISIONMAKING AT EPA

Growing concerns over the pollution of the global environment with chlorinated hydrocarbon insecticides led Rachel Carson, in 1962, to focus public attention upon their potential for harm.

> ...(T)he central problem of our age has therefore become the contamination of man's total environment with such substances of incredible potential for harm--substances that accumulate in the tissues of plants and animals and even penetrate the germ cells...to alter the very material of heredity upon which the shape of the future depends.[1]

In the years following the publication of Silent Spring[2] the public became increasingly critical of the indescriminate use of these compounds by public agencies and private farmers. As Ms. Carson noted, "(i)t is the public that is being asked to assume the risks that the insect controllers calculate. The public must decide whether it wishes to continue on the present road..."[3]

Since Silent Spring, through actions of public interest organizations like the Environmental Defense Fund (EDF), non-agricultural interests have played an important role in the formulation of pesticide policy within the Environmental Protection Agency (EPA). Often, that role has been one of initiating agency action when a registered pesticide posed an unreasonable adverse effect on man or the environment. At other times public interest organizations have been active participants in pesticide cancellation and suspension proceedings under FIFRA in support of the Agency's position. The

cancellations of DDT, aldrin/dieldrin, and chlordane/heptachlor by EPA were due in large measure to the involvement of public interest environmental organizations in these proceedings.

Almost all of the registered uses of DDT (1,1,1-trichlorophenylethane) were cancelled on 30 June 1972,[4] by then EPA Administrator William Ruckelshaus. Earlier the Court of Appeals for the District of Columbia held that cancellation proceedings should be commenced whenever a registration of a pesticide raised a "substantial question of safety"[5]

Under the pre-1972 FIFRA a pesticide was misbranded,

> (i)f the labeling...does not contain directions for use which are necessary and...adequate for the protection of the public...[6],
>
> (i)f the label does not contain a warning or caution statement...adequate to prevent injury to living man and other vertebrate animals, vegetation and invertebrate animals...[7],
>
> (i)f in the case of an insecticide, nematocide, fungicide or herbicide when used as directed...shall be injurious to living man or other vertebrate animals or vegetation, except weeds, to which it is applied, or to the person applying such economic poison...[8]

The 1972 amendments to FIFRA provided that a registration should be cancelled or denied if it appeared that the pesticide when used as directed or according to common practice, "generally caused unreasonable adverse effects on the environment..."[9] The statute defined "unreasonable adverse effects on the environment" to mean "any unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide."[10]

TOXICOLOGICAL TESTING

Whether the regulatory standard to be applied, in a use cancellation/suspension decision, was the "substantial question of "safety" test or the "unreasonable adverse effects" test, the Administrator was compelled by the statute to make "both a factual determination on the evidence before him and an application of a legal standard."[11] The factual determination was based upon an evaluation of the results of toxicological testing of the pesticide in question. In the case of DDT, aldrin/dieldrin and chlordane/heptachlor the administrative decision to cancel or suspend their registrations was made on the basis of the results of carcinogenicity tests on one or more rodent species and the extrapolation of these data to infer presumptive human health effects.

It is virtually impossible to claim that a compound is "safe", that is, that it presents absolutely no risk to organisms that come into contact with it. Therefore, it is necessary to evaluate the degree of hazard presented by a given compound over some period of time.

Acute Toxicity

There are numerous ways to assess the risk posed by any particular chemical. Acute toxic effects may occur if a single, short term exposure to a given compound is sufficient to cause adverse effects (usually morbidity or mortality) in a test organism. The results of acute toxicity tests are usually expressed as LD50s or the lethal dose of a substance that will kill 50% of the test organisms. As would be

anticipated the onset of symptoms for an acutely toxic substance are severe and rapid.[12]

Chronic Toxicity

Often a substance exerts its toxic effect(s) only after prolonged or chronic exposures. Of particular concern, for regulatory purposes, is whether exposure to a compound at some time in an organism's life will result in cancer at some later point in time. There are two principle methods employed in determining whether a substance may be a carcinogen: (1) epidemiological studies involving people exposed to the substance, and, (2) long-term tests on laboratory animals.

Epidemiological Studies

Epidemiological evaluations are of limited utility in identifying carcinogens. For one thing, they are relatively insensitive.

> For something to be defined as a risk in an epidemiologic study,...usually a large difference in rates of disease or a large relative risk [have] to be found.[13]
>
> ...For example, the overall incidence of lung cancer in the general population in 1970 was about forty cases for each 100,000 people. . .as the probability of an individual contracting lung cancer in that year was thus about one in 2,500, an extremely large number of people would have [had] to [have been] observed to ensure that there would be enough cases of the disease to study.[14]

In addition, epidemiological studies are very costly and often difficult if not impossible to conduct due to the usual lag period between the initial exposure to a carcinogenic agent and the manifestation of the disease. The "latency period", (that is, the time between exposure to the carcinogenic agent and the appearance of a cancer), is usually thought to be anywhere from 15 to 40 years. Furthermore, the human

population is often ubiquitously exposed to numerous carcinogenic agents. It is often quite a formidable, if not impossible, task to identify groups of people with sharply contrasting exposures to a particular chemical agent.

> Because of these problems...and because testing for carcinogenicity of chemical substances is beyond the ethical bounds placed by society on human experimentation, most data for identifying potential cancer risks...come(s) from animal studies.[15]

Species-to-Species Correlation

Fundamental to cancer research is the presumption that what is found to induce cancer in one species of animal must also be assumed to induce cancer in others.[16] Whether they occur in humans or experimental animals chemically-induced tumors are, in general, more similar than dissimilar to one another. This principle, known as the "species-to-species extrapolation principle, is grounded in the fact that [the] fundamental life processes in mammalian and other animals are basically the same as those in humans."[17] Thus, unless there is compelling evidence to the contrary, a substance which is found to induce cancer in a sensitive animal surrogate species, in a properly designed and executed study, must be considered a presumptive human carcinogen.[18]

Theory and Practice of Carcinogenesis Bioassays

Rodents are generally used as the surrogate species of choice in carcinogenesis assays. There are several reasons for this. Mice and rats have been used for over fifty years, and hamsters over thirty years, in evaluating the carcinogenic potential of various environmental

agents. Over this period of time it has been shown that, "most chemicals that cause cancer in humans...cause cancer in rodents, although not necessarily in the same organ or in all species and strains tested."[19]

> The rat and mouse have been shown to be susceptible to the carcinogenic action of a large variety of compounds and, indeed, most of our knowledge of chemical carcinogenesis is based on the use of these species. The hamster and guinea pig are relatively resistant to the carcinogenic action of several compounds which produce tumors readily in the rat and mouse. On the other hand, no known carcinogens for the hamster and guinea pig are inactive in the rat and mouse.[20]

Perhaps more importantly, all chemicals known to cause cancer in man, with the possible exception of arsenic, also do so in experimental animals.[21]

The carcinogen bioassay[22] is a chronic toxicity study which usually subjects the test animal species to a lifetime exposure to the substance being evaluated. When using mice or rats as the test species, the duration of exposure is usually 18-24 months. Typically the number of animals used is limited--often not more than 600 animals of both sexes and species.[23]

Different groups of animals are exposed to different levels of the test agent up to and including the maximally tolerated dose (MTD).[24] Theoretically, the MTD is the largest quantity of test agent administered during a chronic study that will not result in excess morbidity or mortality in these animals. "In practice, the MTD is considered to be the highest dose that causes no more than a 10 percent loss in weight compared to control animals."[25]

Fifty animals of each species and sex are normally tested at each of two dose levels including a control (no compound) level. Animals are examined periodically for signs of disease or overt toxicity. Often

animals are sacrificed at predetermined times, and their tissues examined to evaluate the time to tumor date observed after continuous exposure to the carcinogenic agent. At the termination of the study, all of the animals remaining are sacrificed and their tissues preserved for pathologic evaluations.

In order to compensate for the limited population numbers used in these studies and, thus, their limited sensitivities the administration of large dosages of the compound is usually required.

> The need for large dosages of chemicals in some experiments is a reflection of the facts (1) that some carcinogens are much less potent than others and (2) that animal experiments,...must make use of finite animal resources. To illustrate this point, let us suppose that humans and rats are equally sensitive to some chemical carcinogen which causes one case of cancer in every 10,000 persons (or rats) to which it is given. If 220,000,000 Americans were exposed to this chemical, 22,000 cases of cancer would occur. On the other hand, if fed to the typical 50 rats used in an experiment, the chances that even one rat would get cancer is one-half of one percent; 10,000 rats would have to be fed the chemical (at human dosages) to observe even one cancer.[26]

The likelihood of obtaining a carcinogenic response is increased by raising the dosage levels of carcinogenic agents. Furthermore, should one obtain negative results in a study employing high doses one can have greater confidence that the results obtained are a true reflection of the carcinogenic potential of the compound in question.

There is a common mythology that high doses of any chemical can cause cancer in appropriately designed carcinogenesis bioassays. This is simply not supported by the facts.

> High doses of noncarcinogens may produce a variety of toxic effects but will not produce cancer. The intrinsic carcinogenicity of a chemical does not depend on dose level although the proportion of animals developing cancers and the earliest time that tumors are detected are usually related to dosage.[27]

Or in another light,

> The bottom line on carcinogenisis testing is this. You can drown an animal in a pool of some substance, suffocate an animal under heap of it, or beat an animal to death with a sock full of it, but if it isn't carcinogenic, you can't give an animal cancer with it.[28]

Actually, only about 20% of those chemicals suspected of causing cancer, because of their similarity to known carcinogens, have been demonstrated to produce cancer in carcinogenesis bioassays.[29]

There are several inherent problems with extrapolating the results of animal studies to man. First, one must assume that rodents and humans are equally sensitive to the same toxic agent. Yet, for any given substance, humans may be either more sensitive, equally sensitive, or less sensitive than rodents to the carcinogenic or other toxic effects of the chemical agent being investigated. By extension, the use of this assumption can lead to over or understatements of risk depending upon the type of test used and the animal species being studied.

> For example, the lowest dose of thalidomide inducing birth defects in pregnant women is 0.5 mg/kg/day; the corresponding values for the mouse, rat, and dog are 30, 50, and 100 mg/kg/day, respectively. Thus, humans are 60 times more sensitive than are mice to thalidomide, 100 times more sensitive than rats, and 200 times more sensitive than are dogs.[30] ...[C]ertain aromatic amines, such as 2-naphthalamine, are potent bladder carcinogens for man, monkeys, and dogs, but not for rats, mice and other rodents, in which tumors at other sites are produced.[31]

If one is to use the results of animal tests as valid indicators of potential human harm then one must also assume that there is a direct correlation between the dose of a compound and the appearance of tumors at that dosage level. This one-to-one correlation is conventionally referred to as the linear dose-response extrapolation. Practically speaking it is almost impossible to verify this theory through direct experimentation. Our collective inability to determine the presence or

absence of a "threshold" for carcinogens centers upon our lack of "hard data" on the lower ends of the dose-response curve. However, in order to err on the side of protection of public health it has become standard practice "to assume that the extrapolation is linear."[32] (See figure 1, next page.) The inevitable conclusion one comes to is that on the basis of experimental animal data, there is "no known method [to] predict a safe human (exposure) level for carcinogens,...if...such safe levels for humans exist at all."[33]

FIFRA calls for the cancellation of a pesticide's registration if the use of that compound results in adverse effects on non-target organisms. The environmental effects of not only DDT, but also aldrin/dieldrin and chlordane/heptachlor were thoroughly documented. Yet, in the cancellation and cancellation/suspension hearings to follow, the regulatory decision to curtail or eliminate the use of these products from the marketplace were made not on the basis of their environmental fate and effects but rather on the basis of their presumptive human carcinogenicity. It was on this body of information that the EPA cancelled the use registrations for the insecticides DDT, aldrin/dieldrin and chlordane/heptachlor.

DDT

First synthesized in 1874, DDT (dichlorodiphenyltrichloroethane) was not recognized as having insecticidal properties until 1939.[34] DDT was originally classified by the U.S. Army during World War II as a "top secret." It was used militarily both in the Asian and European campaigns, in dust form, for the control of typhus.

> Most Americans became acquainted with pesticides in the newsreels of World War II. Lice-ridden soldiers were filmed

Figure 1

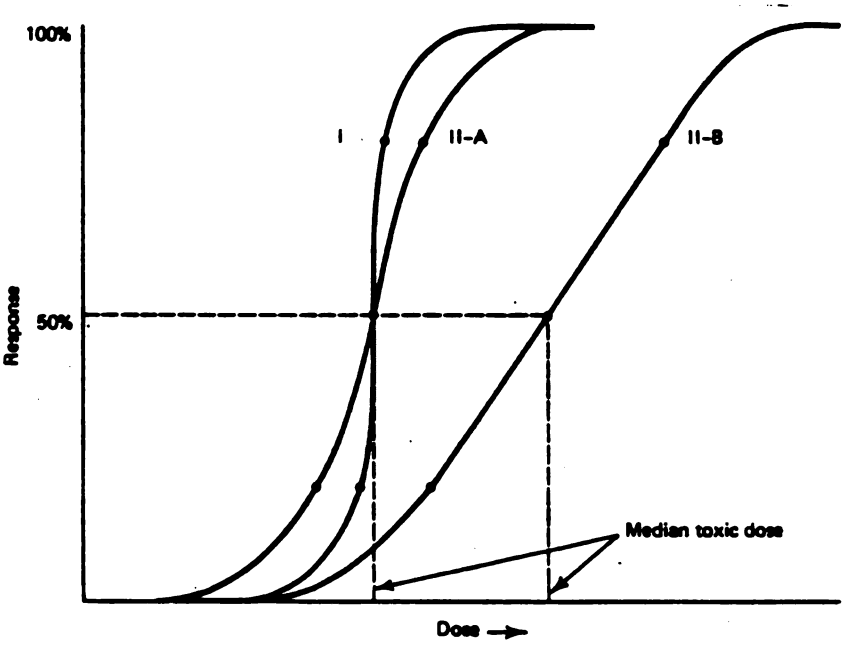

Diagram of dose-response relations. Curve I shows a sharp dose-response curve; curve II–A, a slightly displaced curve with an identical midpoint. Curve II–B is an effect with a broader response. (133)

> lining up in the streets of Naples to be fumigated with DDT. They would disappear in a cloud of dust and emerge smiling and obviously healthy.[35]

In the wake of World War II DDT was heralded as a way to eradicate insect-borne diseases, such as malaria and yellow fever, and end forever the plight of the farmer being at the mercy of crop-eating insects.[36] The Nobel Prize for 1948, was awarded to Müller, the Swiss chemist who discovered DDT's broad insecticidal properties.[37]

A familiar sight on summer evenings during the post-war years was the fogging machine moving up and down urban and suburban streets as children played in and out of the dense clouds of DDT mist.[38] Since the children displayed no obvious ill effects from their DDT exposures the public was lulled into believing that DDT was harmless.

Rachel Carson's book Silent Spring raised concern over the widespread and often indescriminate use of pesticides. In the aftermath of this publication many committees were established to evaluate the critic's allegations that DDT--which had been used for over two decades without any (apparent) ill-effects--was a highly dangerous substance which, (1) had collected in the food chain, (2) killed beneficial insects, (3) upset the ecological balance, and, (4) posed a hazard to aquatic, avian and human life.

The United States Food and Drug Administration, on April 4, 1969, announced the seizure of 28,000 pounds of coho salmon from Lake Michigan.[39] The levels of DDT residues found in their tissues exceeded the FDA's established tolerance limit. Sometime later that year, in August, 1969, a three-day symposium was convened in Corvallis, Oregon to discuss "the biological impact of pesticides in the environment."[40] The

conferees reported that DDT in high concentrations caused physiological damage to the nervous system, liver, and kidneys of test animals.[41]

As early as 1947, a study by Fitzhugh and Nelson,[42] reported that rats fed DDT displayed an increased incidence of liver tumors compared to control animals. (It should be noted that rats are exceedingly resistant to the development of liver tumors.) In 1969, Innes, et al.,[43] demonstrated that DDT, as well as other insecticides, caused the development of tumors in mice. The "Report of the Secretary's Commission on Pesticides and Their Relationship to Environmental Health"[44] (hereinafter, the Mrak Report), stated that,

> (t)he evidence for the carcinogenicity of DDT in experimental animals is impressive and the Panel [the Technical Panel on the Carcinogenicity of Pesticides] takes no exception to the conclusions as to DDT recorded in the JNCI [Innes Study] report of the National Cancer Institute study. This study has demonstrated that DDT increased the incidence of cancer in mice under the experimental conditions employed.[45]

Indeed, the Mrak Report further stated that the results of the carcinogenicity studies they evaluated were significant at the 0.01 level.[46] They recommended that the "exposure of human beings to [this] pesticide...be minimized and that use of [this] pesticide be restricted to those purposes for which...advantages to human health...outweigh the potential hazard of carcinogenicity."[47]

EDF, in conjunction with the Western Michigan Environmental Action Council and the National Audubon Society, filed a petition with the USDA in October, 1969, requesting the issuance of cancellation notices for all economic poisons containing DDT and suspension of the registrations of these pesticides pending the completion of the cancellation proceeding.[48] On 20 November, 1969, in response to the

cancellation/suspension petition, the Agriculture Department issued a notice of intent to cancel the registrations of DDT for minor uses including (1) use on shade trees; (2) tobacco; (3) in and around the home, and, (4) in aquatic environments, marshes, wetlands, and adjacent areas. In the Federal Register notice that followed, (25 November 1969), the Department stated that it "[was] considering cancellation of any other uses of DDT unless it [could] be shown that certain uses [were] essential in the protection of human health or welfare..."[49]

EDF objected to the Department's handling of the DDT issue. They alleged (1) that the November cancellations were unenforceable, (DDT could still be purchased by anyone and used for a disallowed purpose), (2) that the cancellation notice should have included all registered uses of DDT, and, (3) that DDT posed a great enough threat of harm to man or the environment to warrant the Secretary to suspend the registrations of all DDT uses.[50]

In order to obtain judicial relief for their grievances against the Department's actions (and inactions), EDF filed a petition for review in the U.S. Court of Appeals for the District of Columbia. The court held in EDF v. Hardin[51] that the case was to be remanded back to the Department of Agriculture with instructions to the Secretary to either issue the remaining cancellation notices or explain his reason for deferring his decision to issue the same.

The Department's response to the court's orders was in the form of an eight page statement of findings.[52] The Secretary found that (1) DDT in large doses had produced cancer in test animals but its effects in small doses on man were unknown; (2) that DDT was toxic to certain birds, bees, and fish; and that (3) DDT had important beneficial uses

associated with crop protection and disease vector control. (At the time, total domestic sales (1970) were 11,966,196 pounds--98 percent of which was used on cotton[53].) Based upon these, and other, findings the Secretary concluded "(t)hat the use of DDT should continue to be reduced in an orderly, practicable manner which will not deprive mankind of uses which are essential to the public health and welfare. To this end there should be a continuation of the comprehensive study of essentiality of particular uses and evaluations of potential substitutes."[54] In effect, while the Secretary admitted that the continued use of DDT did present a hazard to man and the environment, he did not feel that it posed an imminent hazard which would have called for immediate suspension.

EDF again petitioned the Court of Appeals (D.C. Circuit) to review the Secretary's findings and inactions. (During the pendancy of the court's deliberations, the Department of Agriculture was divested of its pesticide regulatory authority. Authority for regulation and registration of pesticides was transferred to the newly created Environmental Protection Agency (EPA).) The court found that the Secretary's refusal to suspend the remaining uses of DDT or to initiate the formal administrative hearings to review DDT's registrations was grounded upon an improper interpretation of FIFRA.

> (T)he FIFRA requires the Secretary to issue notices and thereby initiate the administrative process whenever there is a substantial question about the safety of a registered pesticide...(W)hen...he reaches the conclusion that there is a substantial question about the safety of a registered item he is obliged to initiate the statutory procedure that results in...a public hearing. ...(O)ne important function of that procedure is to afford the registrant an opportunity to challenge the initial decision of the Secretary. But the hearing...serves other functions... Public hearings bring the public into the decision-making process, and create a record that facilitates judicial review. If hearings are held only after the Secretary is convinced beyond a doubt that cancellation is necessary, then they will be held too

> seldom and too late in the process to serve any of those functions effectively.[55]

The decision of EDF v. Ruckelshaus[56] required the EPA to initiate the administrative hearing process by issuing notices cancelling all registered uses of products containing DDT. The court further instructed the EPA to determine whether the available information on DDT in Agency files constituted an "imminent hazard" warranting a suspension order. EPA issued cancellation notices, pursuant to court order, on all remaining DDT uses within 1 week of the Court of Appeals' decision (15 January 1971).[57]

On March 18, 1971, the Agency published its reasons for denying suspension of products containing DDT.[58] EPA elaborated its general criteria for suspension as follows:

> (T)his Agency will find that an imminent hazard to the public exists when the evidence is sufficient to show that the continued registration of an economic poison poses a significant threat of danger to health, or otherwise creates a hazardous situation to the public that should be corrected immediately to prevent serious injury, and which cannot be permitted to continue during the pendency of administrative proceedings. An 'imminent hazard' may be declared at any point in a chain of events which may ultimately result in harm to the public. It is not necessary that the final anticipated injury actually have occurred prior to a determination that an 'imminent hazard' exists... (S)ignificant injury or potential injury to plants or animals alone could justify a finding of imminent hazard.[59]

In reaffirming his decision not to suspend the registrations for DDT products, Ruckelshaus justified his position as follows. "The present scientific evidence indicates that there would be no significant hazard if only carefully limited amounts of DDT were released into the environment by virtue of restriction of DDT to the most critical uses." And for that reason the, "(p)recipitous removal of DDT from interstate commerce would force widespread resort to highly toxic alternatives in

pest control... The widespread poisonings, both fatal and non-fatal, which may reasonably be projected, present an intolerable short-term health hazard."[60] It is instructive to note the apparent conflict between the criteria for suspension ("significant or potential injury to plants or animals") and the justification for the continued use of DDT during the pendency of the hearing process ("highly toxic alternatives...may present an intolerable short term [human] health hazard"). I have inserted the word "human" in the latter quotation because it was upon the issue of chronic health risk to humans that the insecticides DDT, aldrin/dieldrin and chlordane/heptachlor were ultimately resolved in favor of cancellation. One must also be somewhat sceptical of the Administrator's reasons not to cancel all uses of DDT. The only significant use remaining at this time was for cotton pest control for which there were numerous substitute chemicals available. It was ironic that the Agency was fighting for the retention of this use given the fact that the cotton insect complex was totally resistant to DDT and had been since the mid-1950s.

The consolidated cancellation proceedings on DDT began on August 17, 1971. The hearing lasted seven months with 125 witnesses, 370 exhibits, and 9,300 pages of testimony.[61] Intervenors to the administrative hearing, on the side of the Agency, included the Environmental Defense Fund (EDF), the Western Michigan Environmental Action Council and the National Audubon Society. The case against cancellation was presented by Eli Lilly and Company, H.P. Cannon and Sons, the National Agricultural Chemicals Association and the Secretary of Agriculture (Earl Butz).

The EPA and EDF position was that DDT was a nonspecific chemical which indiscriminately killed target and non-target organisms alike. Furthermore, once dispersed, DDT was an uncontrollable, durable chemical that (1) persisted in the aquatic and terrestrial environments; (2) collected in the food chain and was passed up to higher forms of aquatic and terrestrial life; (3) that under certain conditions DDT or its metabolites persisted for many years in soils; (4) that it could move from the site of application via runoff and/or volatilization, and, (5) proof of this could be found by its presence in areas geographically remote from direct application sites and by it or its residues being present in the tissues of pelagic (open-ocean) animals.[62]

Industry's position was that "whatever harm to the environment might be attributed to DDT (was the result of) misuse and overdosing that occurred in years past."[63] They did not challenge the assertion that continued use of DDT on cotton and other crops in the South and Southeastern U.S. would result in its appearance in the food chain and in the marine environment. But they vehemently denied any allegations which implied that DDT uses posed a threat to man or the environment.[64]

On the issue of carcinogenicity, EPA and EDF, presented evidence demonstrating DDT's carcinogenic effects in laboratory animal studies. The Bionetics Study sponsored by the National Cancer Institute fed 120 compounds to two strains, both sexes, of mice. DDT was one of a total of 11 compounds to produce an elevated incidence of tumors. DDT induced hepatomas in males and lymphomas in females at dietary feeding levels of 140 parts per million (ppm).[65]

Research conducted under the auspices of the International Agency for Research on Cancer (IARC) of the World Health Organization were

performed in Lyon (France) and Milan (Italy). The Lyon and Milan studies were multi-generational in design and ultilized 6000 mice from both inbred and outbred strains. The Lyon study exposed male and female mice to 2, 10, 50 and 250 ppm of DDT in the diet. DDT was found to induce hepatomas in male mice at all dosages studied; only females at the 250 ppm level displayed comparable results. The hepatomas were found to have metastasized to the lungs or kidneys in 5 animals. The Milan study demonstrated comparable results; increased hepatomas were observed in male and female mice fed either 250 ppm or 100 ppm of DDT.

Witnesses for EPA/EDF attesting to the positive correlation between tumorigens (substances causing benign tumors) and carcinogens (substances causing malignant tumors) included three members of the Mrak Report's panel on carcinogencity: Dr. Umberto Saffiotti, Dr. Marvin Schneiderman and Dr. Samuel Epstein. Their testimony formed the basis for EPA/EDF's definitions of carcinogencity--the seven cancer "principles." These principles asserted that,

> -A carcinogen is any agent which increases tumor induction in man or animals.
>
> -Well-established criteria exist for distinguishing between benign and malignant tumors; however, even the induction of benign tumors is sufficient to characterize a chemical as a carcinogen.
>
> -The majority of human cancers are caused by avoidable exposure to carcinogens.
>
> -Carcinogenesis is characterized by its irreversibility and long latency period following initial exposure to a carcinogenic agent.
>
> -While chemicals can be carcinogenic agents only a small percentage actually are.
>
> -A carcinogenic agent may be identified through analysis of tumor induction results with laboratory animals exposed to the agent, or on a post hoc basis by properly conducted epidemological studies.

> -Any substance which produces tumors in animals must be considered a carcinogenic hazard to man if the results were achieved according to the established parameter of a valid carcinogenesis test.[66]

Industry counterarguments primarily addressed the high levels of DDT which the test animals were exposed to in order to induce cancer; the extrapolatability of animal results to man; and, the lack of human evidence that DDT was a demonstrated human health hazard. The first point was addressed by the chief of the unit on carcinogenesis, of the IARC, Dr. Lorenzo Tomatis. Tomatis was supervisor of the Lyon study used by EPA as evidence of DDT's carcinogenecity. He stated that, "(t)here is no evidence that DDT has the same effect in man that it has in our experimental animals insofar as the induction of hepatomas is concerned. So far there is absolutely no evidence of that."[67]

Dr. Leon Golberg's testimony sought to understate the relevance of animal data by drawing attention to the high levels of DDT needed to elicit a carcinogenic effect in mice. He felt that "while the object of the toxicologist is to seek out target organ effects elicited by high doses, his purpose...is to provide guidance...Grossly aberrant pathways and rates of metabolism that may exist at exaggerated doses make it imperative not to assume that effects at these doses are necessarily characteristic of the changes occurring at lower levels of exposure."[68]

Industry rested its rebuttal of the health hazard data ultimately upon the lack of human epidemiological data for DDT. In essence their position was that the failure to find an effect was absolute proof that there, in fact, was no effect. This position was supported by the testimony of Dr. Jesse L. Steinfeld (Surgeon General of the U.S.) and Dr. John Higginson, Director of IARC. Higginson claimed

that there was, "no evidence at present that during the recent part of the last 25 years there has been a significant trend or modification in trend in cancer patterns that would suggest a reasonable association with the use of DDT in our present environment."[69] The Surgeon General's testimony augmented that of Dr. Higginson when he stated that "(i)f there were some very blatant disease caused by DDT it seems to me we should have found it by now."[70] The testimony of these two witnesses ignored the fact that statistically relevant epidemiological studies were virtually impossible to perform since there were no adequate control populations, i.e., persons who were not exposed to DDT. Also, unless DDT caused a very rare form of cancer or other disease, it would in all probability not be detected by an epidemiological study.

No evidence produced by the proponents of registration established that DDT conferred anything but marginal benefits to cotton farmers. And, as many substitute insecticides were available, the continued registrations of DDT were clearly not essential.

In his opinion and order, William Ruckelshaus concluded that the

> Agency and EDF have established that DDT is toxic to non-target insects and animals, persistent, mobile and transferrable and that it builds up in the food chain. No label directions for use can completely prevent these hazards. In short,...they have established the risk of the unknown. That risk is compounded where, as is the case with DDT, man and animals tend to accumulate and store the chemical...(T)he risk to human health from using DDT cannot be discounted...The possibility that DDT is a carcinogen is at present remote and unquantifiable; but if it is not a siren to panic, it is a semaphore which suggests that an identifiable public benefit is required to justify continued use of DDT. Where one chemical tests tumorigenic in a laboratory and one does not, and both accomplish the same task, the latter is to be preferred...There is no persuasive evidence of record to show that the aggregate volume of use of DDT for all uses in question...will not result in continuing dispersal and buildup in the environment.[71]

ALDRIN/DIELDRIN

"I mean there is no fooling around, the major issue is cancer."[72]

Aldrin and dieldrin, (A/D), are closely related cyclodiene insecticides. (Aldrin is readily converted to dieldrin by an oxidation process both in the body and in the general environment.) They were introduced into the United States as broad spectrum, nonsystemic, persistent insecticides in 1948, by the Julius Hyman and Company. Two years later, Shell Chemical Company, a subsidiary of Shell Oil, became the exclusive distributor of A/D. In May, 1952, Julius Hyman and Company was incorporated into Shell Chemical. From 1952 to 1977 A/D was manufactured in the United States exclusively by Shell.

Until the mid-1950s, A/D was sold primarily for use on cotton against the boll weevil. Its popularity for cotton insect control began to decline as the insect pests of cotton became resistant to A/D. (A/D sales declined from a high, in 1966, of 22 million pounds to about 12 million pounds in 1972[73].) After 1955, A/D were aggressively marketed and sold for use in corn against the corn soil insect complex (cutworms, wireworms, and rootworms). As of 1972, corn soil usage accounted for 80 percent of total A/D sales; termite control, constituting 15 percent of the market, was the second largest usage category. In the eight state Corn Belt, A/D were used principally for crop insurance purposes rather than for treatment of actual infestations. Still, this market accounted for sales of 7.6 million pounds of aldrin in 1974; sales of dieldrin for the same year amounted to 600,000 pounds.

Both aldrin and dieldrin are acutely toxic to humans in contrast to DDT's extremely low acute toxicity. In the case of aldrin, poisoning can occur via any of three exposure routes: ingestion, inhalation and/or skin absorption. Severe symptoms may result from the ingestion or dermal absorption of from one to three grams--especially in the presence of liver disease. Renal damage, ataxia, tremors, convulsions, followed by CNS (central nervous system) depression, respiratory failure and death can occur from acute exposures. Over a prolonged period of time, chronic exposures may result in liver or hepatic damage.[74]

Like DDT, A/D are highly persistent, lipophilic compounds. Four years after a single application of dieldrin, 50 percent can still be recovered from a treated field. Grain and forage crops grown on A/D treated soils become contaminated with dieldrin residues. When contaminated grains are fed to livestock the residues of dieldrin appear in meat and dairy products. There is conclusive evidence that residues of A/D are present in the adipose tissue of nearly every member of the U.S. population.[75] The consumption of contaminated foodstuffs was ultimately the major, if not the primary, source of human exposure.

On 3 December 1970, one day after the EPA formally came into existence, the Environmental Defense Fund petitioned the EPA for the cancellation and immediate suspension of all registered uses of A/D[76], on the basis of the severe environmental damage and carcinogenicity of these compounds. One year earlier, the Mrak Commission found both aldrin and dieldrin to be carcinogenic compounds when tested in the mouse.[77] They recommended that,

> the exposure of human beings to these (compounds) be minimized and that use of these pesticides be restricted to those purposes for which there are judged to be advantages to human health which outweigh the potential hazard of carcinogenicity.[78]

In response, the Administrator of EPA, William Ruckelshaus, on 18 March 1971, issued notices of cancellation of A/D based upon a finding of a "substantial question of safety."[79] The Agency's decision was premised upon the legal distinction between the substantial question of safety test, upon which all cancellation decisions are based, and the "imminent hazard" test, which forms the legal standard for the issuance of suspension notices.

The inherent dangers of A/D considered by the Agency to present substantial questions as to the safety of these products

> are similar to those encountered with DDT...they result from the persistence of dieldrin [since aldrin residues quickly break down into dieldrin] in the environment and its potential toxicity at low levels. Some studies indicate that dieldrin alone, or in possibly synergistic combination with DDT, has an equivalent potential for adverse effect on non-target predatory wildlife resulting from its low level toxicity intensified by its mobility and concentration up certain food chains. The scientific data also indicate that dieldrin, again like DDT, has affinity for storage in the fatty tissue in a number of animals, including humans. There are also similar carcinogenic data developed in the laboratory from high dosage rates of dieldrin administered to test animals.
>
> Dieldrin and aldrin apparently have a lower threshold of toxicity to warm-blooded animals than does DDT. In fact, instances of non-lethal human poisoning have occurred in those occupationally exposed to heavy concentrations of dieldrin...Recovery...was slow but apparently complete. These potential hazards deserve a full public airing in the administrative forum provided by the cancellation proceeding.[80]

While admitting that dieldrin raised safety questions analogous to those raised by DDT and that "there [were] also similar carcinogenic data developed in the laboratory from...dieldrin administered to test animals"[81], Ruckelshaus refused to issue suspension notices for all registered uses of A/D.

It should be remembered from the DDT decision that suspension was indicated whenever an imminent hazard was believed to exist.

> [T]his Agency will find that an imminent hazard...exists when...evidence is sufficient to show that continued registration of an economic poison poses a significant threat of danger to health, or otherwise creates a hazardous situation to the public...which cannot be permitted to continue during the pendency of administrative proceedings...It is not necessary that the final anticipated injury actually have occurred prior to a determination that an 'imminent hazard' exists...(S)ignificant injury or potential injury to plants or animals alone could justify a finding of imminent hazard.[82]

EDF responded to the Administrator's decision not to suspend A/D by appealing the Agency's refusal to the Court of Appeals for the District of Columbia Circuit for review. (At the same time the Shell Chemical Company exercised its statutory right to convene an administrative hearing and also have the National Academy of Sciences select a scientific advisory committee to review and evaluate the evidence against A/D.)[83]

In March and May of 1972, one year after the Administrator's initial decision on A/D, the scientific advisory committee and the court of appeals (respectively) issued their written opinions. The committee affirmed the EPA's decision that suspension was unwarranted. Furthermore, it felt that notices of intent to cancel A/D uses on corn soil, as a pre-planting seed dressing and for termite control were unnecessary (these were the major uses of A/D - constituting approximately 95 percent of domestic sales.)[84] Two months later, (5 May 1972), the court of appeals remanded the case back to EPA for a more in-depth discussion of the risks and benefits inherent in the continued use of A/D. The court was especially troubled by the Administrator's "one-sentence discussion" of A/D's carcinogenic risk--without

identifying any offsetting benefits which would demonstrate to the court's satisfaction that the substantial question of safety identified by the Agency did not also constitute an imminent hazard.

> By definition, a substantial question of safety exists when notices of cancellation issue. If there is no offsetting claim of any benefit to the public, then the EPA has the burden of showing that the substantial safety question does not pose an "imminent hazard" to the public.[85]

Especially when "the matter involved is as sensitive and fright-laden as cancer."[86]

The following month, Ruckelshaus reaffirmed his cancellation decision with one caveat; that he was contemplating the suspension of certain uses of A/D.[87] (There is reason to believe that Shell was anxious to avoid suspension at all costs. It reached an agreement with the user/registrants (persons who purchased A/D directly from Shell) to discontinue the majority of the suspicious uses including dust formulations, aerial application and mothproofing[88].) Concluding that the evidence on the carcinogenicity of dieldrin was too uncertain, the Administrator, in his final decision on A/D (December, 1972), decided that suspension of dieldrin or its uses was unwarranted.[83,88]

The cancellation hearing on the risks and benefits of A/D did not begin until August 7, 1973--nearly two and one-half years after the original notices of cancellation were issued. One year into the hearings, on August 2, 1974,[89] the new Administrator of EPA Russell Train, issued a notice of intent to suspend A/D.[90] Shell Chemical had refused an earlier request of the Agency to delay the manufacture of 10 million pounds of A/D scheduled to begin on September 1, 1974. (Ten million pounds of active ingredient would translate into 50 million pounds of formulated product which would have to be disposed of

somehow). Train was concerned not only about the environmental risks inherent in disposing of this quantity of chemicals, if he decided in favor of cancellation when the hearings were scheduled to end in four to five months, but also the carcinogenicity of these insecticides. In his statement of his intention to suspend the registrations and production of A/D, Train listed five factors which contributed to his finding that the continued production and use of these chemicals constituted an "imminent hazard" to public health:

> (1) That even at low dietary levels of exposure (0.1 ppm) dieldrin caused statistically significant increases of tumors in both mice and rats.
>
> (2) Measurable amounts of dieldrin were present in 96 percent of all meat, fish, and poultry sampled; 83 percent of all dairy products sampled; and, 88 percent of all garden fruit.
>
> (3) 99.5 percent of all Americans tested had measurable amounts of dieldrin in their adipose tissues, the average being 0.29 ppm.
>
> (4) Because of their greater consumption of dairy products, children are especially at higher risk of dieldrin consumption per pound of body weight than any other population group in this country, and
>
> (5) The average American daily dietary intake of dieldrin subjects the population to an unacceptably high cancer risk.[91]

The suspension hearing, begun on 7 August 1974,[92] was concerned exclusively with whether A/D posed a cancer hazard to human beings, and whether it provided any tangible benefits which would outweigh its risks. The major areas of contention between the Agency/environmental intervenors and the proponents of continued registration, Shell Chemical and the USDA, centered upon (1) the validity of the experimental animal carcinogenicity data, and (2) the relevance of such data to a presumed carcinogenic effect in humans.

The position of EPA/EDF was formulated and summarized as the "nine cancer prnciples", an elaboration and affirmation of the "seven cancer principles" established during the DDT cancellation hearing. The nine cancer principles were as follows:

(1) A carcinogen is any agent which increases tumor induction in man or animals.

(2) Well-established criteria exist for distinguishing between benign and malignant tumors; however, even the induction of benign tumors is sufficient to characterize a chemical as a carcinogen.

(3) The majority of human cancers are caused by avoidable exposure to carcinogens.

(4) While chemicals can be carcinogenic agents, only a small percentage actually are.

(5) Carcinogenesis is characterized by its irreversibility and long latency period following the initial exposure to the carcinogenic agent.

(6) There is great variation in individual susceptibility to carcinogens.

(7) The concept of a "threshold" exposure level for a carcinogenic agent has no practical significance because there is no valid method for establishing such a level.

(8) A carcinogenic agent may be identified through analysis of tumor induction results with laboratory animals exposed to the agent, or on a post hoc basis by properly conducted epidemiological studies.

(9) Any substance which produces tumors in animals must be considered a carcinogenic hazard to man if the results were achieved according to the established parameters of a valid carcinogenesis test.[93]

In five separate feeding studies, involving three genetically different strains of mice, A/D were clearly established as carcinogenic agents (the mice were of inbred, outbred and hybrid strains). The IARC concluded that when mice and rats were exposed to dieldrin by the oral route:

> The hepatocarcinogenicity of Dieldrin in the mouse was demonstrated and confirmed in several experiments, and some of the liver tumors were found to metastasize. A dose-response effect has been demonstrated in both sexes with an increased incidence in females at the lowest dose tested, 0.1 ppm in the diet.[94]

These conclusions were corroborated by Shell's own test results which demonstrated that the increase in the incidence of tumors was dose-related (all three strains of mice in seven tests had a high increase in hepatocarcinogenicity).[95] The incidence of liver tumors was diminished at levels above 10 ppm due to dieldrin's acutely toxic effects upon the test animals. At 0.1 ppm, the lowest level evaluated, there was an increase in both benign and malignant tumors.[96] Those tumors that did become malignant had an increased tendency to metastasize to other parts of the body, especially the lungs.[97]

In both male and female mice A/D shortened the latency period for tumor development.[98] A/D increased the development of tumors after mice had been exposed to it for as little as two weeks; more pronounced effects were apparent after an exposure of one month's duration.[99]

Simultaneous exposure to A/D and DDT apparently has a synergistic effect upon tumor development. Mice were shown to have an increased incidence of tumors when fed 50 ppm DDT. When mice received a diet containing 50 ppm DDT plus 5 ppm A/D their incidence of tumors increased dramatically; the incidence of malignant tumor formation in males increased by a factor of four and females by a factor of eight when compared with mice only exposed to DDT.

> It is certainly clear from these observations that Dieldrin and DDT have additive effects when it comes to carcinogenicity. Further, the evidence indicates that Dieldrin is primarily responsible for this...effect. Using th 50 ppm group as the controls the carcinogenic effect of the combined feeding of Dieldrin and DDT is very highly significant by statistical analysis.[100]

The data available from tests on rats, while less extensive than that on mice, was no less conslusive. Two studies, Tunstall I and FDA 1964, confirmed the carcinogenicity of A/D in rats. A markedly increased incidence of liver and other tumors was observed in exposed animals. These findings take on an added significance since the strains used in the feeding studies had a low spontaneous rate of hepatocarcinoma.[101]

The ultimate question in the A/D suspension hearing was whether A/D could be considered a human carcinogen. "Because man's response to carcinogens is similar to that of rodents, the finding that a substance is carcinogenic in experimental animals indicates that it poses a similar risk to man."[102] In support of the EPA/EDF position, Dr. Walter Heston testified that:

> Knowing this, and knowing the general biological similarity of mice and other mammalian species, including man, we can reasonably expect that in a population of human beings exposed to Aldrin-Dieldrin, cancer of some kind will occur in some individuals, and these individuals will not have been afflicted in the absence of these compounds...The human population is so much more genetically diverse than any laboratory animal, that if a chemical has been shown to be carcinogenic by a significant induction in any laboratory strain of mammal, we can reasonably expect that at least certain human beings would also respond to the chemical by developing some kind of neoplasm.[103]

Dr. Don Stevenson, Director of Shell's Tunstall Laboratory made the strongest rebuttal argument for the registration proponent (Shell Chemical). He and other Shell witnesses adamantly insisted that the data demonstrating the carcinogenicity of A/D in laboratory animals was insufficient to support cancellation/suspension. Dr. Stevenson urged a progressively escalating standard of proof for the (human) carcinogenicity of A/D. Dr. Stevenson's criteria for carcinogenicity included:

(1) Induction of carcinogenicity must be statistically significant at all dose levels.

(2) A uniformly positive dose-response relationship must be found at all doses, even if there is competing toxicity and high mortality at high doses.

(3) A causal association between A/D treatment and carcinogenic effects cannot be sustained unless the mechanism of action of the carcinogen can be demonstrated.

(4) Conclusions on carcinogenic effects of A/D cannot be accepted until the possibility of unknown "augmenting factors" has been excluded.

(5) A carcinogenic effect must be consistent and reproducible in a series of different tests before it can be accepted.

(6) The induction of liver tumors in mice is no indication of carcinogenic effects, even if they are unequivocally malignant.

(7) Tumor production in mice, even in various different organs and even when replicated, cannot be accepted as evidence of carcinogenicity.

(8) Even the finding of carcinogenic effects in two or more animal species is unacceptable proof in the absence of evidence in humans.[104]

Dr. Stevenson's position was supported by the testimony of Massachusetts Institute of Technology cancer specialist Dr. Paul Newberne.

> From a scientific standpoint,...the fact that chemicals, carcinogenic in other species, produce hepatic nodular lesions in the mouse, does not warrant the labelling of a compound of unknown activity as a carcinogen simply because it causes nodules in the mouse liver.[105] [Upon reevaluation of tissue slides alleged by Shell to display "hyperplastic nodules", a team of independent pathologists found an excess incidence of liver cancer in all cases.]
>
> ...(T)here is some evidence that a number of factors may be involved in the induction of mouse liver lesions. Until more is known about their role, it would be unsound to base conclusions on carcinogenic activity on the sole induction of mouse liver tumors, even assuming they are all cancers. It is my feeling that mice as a species...should not be used for safety testing.[106]

It is perhaps ironic that the Shell Chemical Company and its witnesses, while critical of the "appropriateness" of mouse studies after A/D were demonstrated to be carcinogenic in the mouse, predominately relied upon mouse studies in support of the safety of continued use of A/D.[107]

Dr. Samuel Epstein challenged Shell's assertion that one must know the mechanism(s) of causality before one can establish that a compound is a carcinogenic agent.

> The emphasis by Shell witnesses that knowledge of mechanisms must be defined before any agent can be considered carcinogenic [in man], even though this agent has been demonstrated to induce carcinogenic effects in valid experimental systems, can only be regarded as misleading in the extreme. In fact, in spite of a very considerable amount of research, the basic mechanisms of action of any single carcinogen have not yet been elucidated. This requirement of Shell would define away the entire field of chemical carcinogenesis.[108]

In his opinion on the suspension of A/D EPA Administrator Train rejected Shell's arguments that one must know the mechanisms by which a compound exerts its carcinogenic effects and must have corroborative human epidemiology data before one could state that A/D were carcinogens.

> Our knowledge of cancer mechanisms is still imperfect and it may take many years before we understand the mechanisms with certainty. Furthermore, epidemiological studies are difficult or impossible to conduct on the effects of aldrin-dieldrin...It is the carcinogenic effect of Aldrin-Dieldrin, not the mechanism, that concerns us here.[109]

Train was also presented with similarly inconclusive evidence in his evaluation of the benefits of continued A/D use. At issue was whether the "banning" of A/D would lead to yield reductions in corn. He concluded that "the macroeconomic impact of the proposed suspension order would be almost negligible",[110] since: (1) most of the A/D used

was used prophylactically, and, (2) substitute compounds were readily available to replace the loss of A/D to corn farmers.

In October, 1974, EPA ordered a comprehensive ban on almost all registered uses of A/D. Under Train's decision existing stocks of A/D, those manufactured prior to 2 August 1974, could still be sold and used.

EDF appealed the Administrator's decision on the narrow issue of the continued sale and use of existing stocks of A/D. (In reality, the EDF wanted the Court of Appeals to affirm the validity of the "nine cancer principles" and thereby establish, by legal precedent, a regulatory tool which could be used by EPA to aggressively pursue carcinogenic pesticides.)[111] The Court of Appeals for the D.C. Circuit wholly affirmed the EPA decision on A/D finding that the "suspension order represent(ed) a rational exercise of the broad discretion conferred on the Administrator by the (FIFRA)".[112] The decision of the court also reaffirmed the legal standard for the finding of an "imminent hazard" which would initiate suspension proceedings against a registered pesticide.

CHLORDANE/HEPTACHLOR

Like aldrin/dieldrin, chlordane/heptachlor (C/H) are two closely related cyclodiene insecticides. They undergo metabolic and environmental transformation to persistent, stable and lipophilic epoxide derivatives: chlordane is transformed into oxychlordane and heptachlor becomes heptachlor epoxide. These stable derivatives were implicated as posing an imminent hazard to public health during the cancellation/suspension hearings for C/H.

Since the late 1940s, C/H had been exclusively manufactured and sold by Velsicol Chemical Corporation. C/H had historically been used in agriculture for the protection of corn against attack by soil insects. Non-agricultural uses for C/H included treatment of homes for termite infestation and as general insecticides for use around the home, lawn, and garden. The agricultural uses of these insecticides was declining long before their suspension in 1975, due to increasing insect resistance and the introduction of less environmentally persistent alternative insecticides. The suspension of A/D, temporarily increased the agricultural use of C/H, reversing the declining market demand for these insecticides.

In mid-1974, the Environmental Defense Fund (EDF) petitioned the Administrator of the EPA, Russell Train to cancel and suspend all registered uses for C/H.[113] It based its petition upon the same grounds as that for A/D, namely ubiquitous environmental contamination and the finding that C/H had been found to be carcinogens by the Mrak Commission.[114] EDF felt that the continued environmental and human exposures to these compounds not only raised a substantial question of safety, calling for the issuance of cancellation notices, but also posed an imminent hazard to public health calling for their immediate suspension.

On 18 November 1974, (almost one month after the termination of the A/D suspension hearings), Russell Train issued a notice of his intent to cancel[115] all but two registered uses of C/H (The two non-cancelled uses were: subsurface ground injection for termite control, and dipping roots and tops of non-food plants). The cancellation was based upon a finding that "the pesticides appear(ed) to pose substantial questions of

safety amounting to an unreasonable risk to man and the environment.[116] The substantial question of safety was based upon six classes of evidence:

(1) Information derived from laboratory experiments suggested that low levels of heptachlor and heptachlor epoxide (10 ppm and 0.5 ppm, respectively) significantly increased the incidence of liver tumors (including carcinomas) in rats and mice.

(2) Residues of C/H had been found in the tissues of fish, birds and wildlife.

(3) Residues of C/H had been found in meat, fish and dairy products.

(4) Residues of C/H had been found in a 1970-72 human monitoring study in over 90 percent of hospitalized patient's adipose tissue.

(5) Residues of C/H had been found in the tissues of stillborn fetuses, and in cord blood, and,

(6) Residues of C/H had been found in milk samples of nursing mothers.[117]

The "nine cancer principles" developed during the A/D suspension hearings, and later affirmed in EDF v. EPA, were presented in the Agency's first pretrial brief of 1 April 1975, as "the most advanced research findings and policy of both national and international cancer experts and agencies" in support of the proposed cancellation.[118] In its reply brief Velsicol objected to the inclusion of principles number 2 and 7 which were concerned with the essential similarly between benign and malignant tumors following administration of a carcinogenic agent and the scientific inability to set "thresholds" for carcinogens[119], respectively. They moved to have the validity of these principles referred to a committee of the National Academy of Sciences for review. As one would expect, EPA/EDF objected to Velsicol's motion. The grounds for their objection rested upon a finding that benign and malignant

tumors have synonymous scientific and regulatory implications in carcinogenicity testing. Velsicol's motion to strike principles 2 and 7 was flatly denied by Judge Perlman (the same Administrative Law Judge who handled the A/D hearings).[120]

On 29 July 1975, Train issued a notice of intent to suspend all registered uses of C/H except those previously exempted in the cancellation notice.[121] The Administrator made his determination of an "imminent hazard" to public health based upon: (1) new corroborative evidence on the carcinogenicity of C/H, and, (2) the anticipated 12-18 month duration of the cancellation hearing which would result in an additional release of 38 million pounds of C/H into the general environment. In his notice of intent to suspend Train adopted the cancer principles developed during the cancellation hearings on A/D and C/H as "the basis for evaluation" of cancer risks, insuring their incorporation during the suspension proceedings. The Administrator's finding of facts were as follows:

> The use of animal test data to evaluate human cancer risks has been widely accepted by the scientific community and by public policy-making agencies. Such data are particularly appropriate because the relatively short life-span of test animals allows for testing for the entire latency period for tumor development and because of our relatively well-developed understanding of the pathological development of tumors in mice and rats. When compared to the millions of people who may be exposed to a pesticide, the number of animals used in tests to evaluate oncogenicity is extremely small. The variability of human response to carcinogens is generally greater than that of the test animals. Epidemiological cancer data are desirable, but because of the long latency period of tumor induction in humans, because of frequently encountered widespread contamination which makes it impossible to establish an uncontaminated control group and because of the obvious ethical and legal problems associated with conducting cancer research on humans, reliable epidemiological data are rarely available. Accordingly a positive oncogenic effect in test animals is sufficient to characterize a pesticde as posing a cancer risk to man. By the same reasoning, negative results from

> oncogenic effect in test animals is sufficient to characterize a pesticde as posing a cancer risk to man. By the same reasoning, negative results from oncogenic animal tests have only limited significance. The number and sensitivity of the test animals as compared to the general human population are the principal reasons for this limited utility. Because of these inherent limitations of animal testing a pesticide that induces tumors in experimental animals at any dose level must be considered to be a carcinogen. As noted above, negative results are of limited value since they do not rule out the possibility that the chemical will induce tumors in test animals if, for example, the number of exposed animals or the length of exposure were increased. Although a no-effect level may theoretically exist for carcinogens, as yet there is no scientific basis for establishing such a level. Thus, human exposure to a carcinogen at levels below those which induced positive effects must be considered to present a cancer risk. Finally, although the distinction between "benign" and "malignant" tumors is of ppimary importance to the individual, it is not a meaningful distinction in determining the cancer hazard to man on the basis of tests conducted on laboratory animals. Given the increasing evidence that many "benign" tumors can develop into cancers, for purposes of determining whether a pesticide poses a cancer hazard to man on the basis of laboratory experiments, the terms "benign" and "malignant" should be considered synonymous.[122]

The suspension hearing on C/H began on 12 August 1975, with ALJ Perlman presiding.[123] The regulatory battle to ban C/H, like the regulatory battle to ban A/D, centered upon the carcinogenicity of C/H. Unlike A/D, Velsicol disputed the finding that what its pathologists diagnosed as "hyperplastic" nodules were in fact neoplastic nodules and "frank" carcinomas of the liver.[124]

With the exception of two studies, a 1965 FDA mouse study which served as the basis for the Mrak Commission to conclude that heptachlor and heptachlor expoxide were carcinogenic compounds and a 1975 study performed by NCI, the main body of information on C/H, nine studies, were unpublished.[125] It was on the basis of this unpublished data base that C/H were claimed by the manufacturer/registrant to be noncarcinogenic and safe. The studies were generated under contract to Velsicol by two commercial testing laboratories: the Kettering

Laboratories of the University of Cincinnati, Ohio and the International Research Development Corporation (IRDC) of Mattawan, Michigan. Both laboratories found the compounds to be noncarcinogenic. It is especially illuminating to note that while the Kettering study on rats did find a dose-related excess of "hepatomas" in the treated animals they concluded that, "(t)he data obtained from these animals (were) not sufficient to implicate Heptachlor epoxide as the agent responsible for the development of these hepatomas."[126]

Velsicol's position throughout the C/H suspension proceeding was that the diagnosis of cancer was a subjective matter among pathologists. In order to dispel the controversy surrounding the uncertain diagnosis of the liver pathology slides alleged by Velsicol to contain hyperplastic nodules, EPA assembled a team of five independent pathologists, headed by Dr. Melvin Reuber (an expert in the field of rodent liver pathology) to re-examine the slides. In every case Reuber and his team found a high incidence of unequivocal liver cancers. (There were no discrepancies in the diagnoses of industry pathologists and the independent pathologists with respect to untreated control animals and positive control animals exposed to acetylaminofluorine.)

In his opinion Judge Perlman on 12 December 1975, concluded that

> heptachlor and its metabolite appear to be a carcinogen in the mouse on the basis of the FDA and the IRDC studies and may be a carcinogen in the CFN rat on the basis of the Kettering study and that chlordane appears to be a carcinogen in the mouse on the basis of the IRDC study...[127]

Judge Perlman used the terms "apear to be" and "may be" because of the absence of metastasis or meaningful invasion of the liver tumors to other sites in the body. As he concluded, "we are hesitantly unwilling at this time to find that heptachlor and chlordane are conclusively carcinogens in laboratory animals."[128]

Perlman's decision was rejected by Train on 24 December 1975, by the issuance of an order suspending the registrations of C/H. Train emphasized that in failing to find an "imminent hazard" in the continued use of C/H Judge Perlman misapplied both the pertinent statutory provisions of FIFRA and the legal precedents defining the legal standard to be applied in this case. FIFRA defined an "imminent hazard" as,

> a situation which exists when the continued use of a pesticide during the time required for a cancellation proceeding would be likely to result in unreasonable adverse effects on the environment.[129]

Such an unreasonable adverse effect was defined as,

> any unreasonable risk to man or the environment taking into account the economic, social, and environmental costs and benefits of the use of any pesticide.[130]

In the view of the Administrator, therefore,

> the FIFRA and the cases make two points clear...with regard to a suspension proceeding. First, it is not necessary to find "conclusively" that actual harm to man will occur if the use of the pesticide in question is continued; rather the finding required is that continued use during the cancellation proceeding is "likely" to result in "unreasonable risk" to man or the environment. Second, the propriety of suspension turns upon an analysis in which the risks are balanced against the benfits...the mere fact that the evidence on either of these issues...is not complete, or that more evidence may be expected to be developed in the cancellation proceeding, is not a reason to deny suspension.[131]

Both Velsicol and EDF appealed the Administrator's decision to the U.S. Court of Appeals for the D.C. Circuit. Velsicol, supported by the Secretary of Agriculture as intervenor, argued that substantial evidence did not support the Administrator's conclusion that continued use of chlordane posed an "imminent hazard" to human health. They contended, furthermore, that in a suspension proceeding the ultimate burden of proof rests at all times on the Administrator. EDF, on the other hand, felt that the order did not go far enough. They sought an injunction

against the continued production and use of C/H on corn pests until 1 August 1976. In addition, they objected to the Agency's decision to allow the sale of C/H which remained in stock as of 29 July 1975 (the day the notice of intent to suspend was issued). In its opinion the Court of Appeals, in large measure, affirmed the Administrator's finding of fact and policy.[132]

On 6 March 1978, the three years of administrative hearings ended with a settlement agreement reached between the Agency, Velsicol and EDF. Under the terms of the agreement, Velsicol was to gradually phase out all agricultural uses of C/H over a five-year period ending in September, 1982. The settlement provided for the production of no more than 7.25 million pounds of C/H per year and only certified applicators and commercial seed treatment companies were to be permitted to use the insecticides during the phase-out period.

The cancellation and suspension of the use registrations for DDT, aldrin/dieldrin, and chlordane/heptachlor arose as a direct result of public participation in pesticide policy decisions. It is probable that these insecticides would still be on the market if non-agricultural interests had been barred from fully participating in formal and informal pesticide hearings. In the next section (Chapter 4) we shall examine a rulemaking proposal EPA published in August, 1980, which would have significantly abridged the rights of non-agricultural interests to participate in pesticide registration decisions.

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Chapter 4

A Critique of the Proposed Amendments to EPA's Current Rules of Practice Governing Pesticide Hearings

Since its inception EPA has utilized a formal adjudicatory hearing approach in its use cancellation/suspension proceedings against the pesticides DDT, aldrin/dieldrin, and chlordane/heptachlor. When the Agency has issued notices of intent to cancel[1] certain use registrations of a problem pesticide, the application of trial-type procedures has often resulted in protracted hearings. The reason for this is simple: FIFRA allows for the continued sale and use of "suspect" pesticides during the pendancy of a cancellation decision.[2] Thus, a registrant has a strong economic incentive to use every legal maneuver available to him to prolong a cancellation hearing.

"Both the formality of the hearings and the repetitive wrangling over science policy issues consume a great deal of agency and litigant time and resources."[3] On 7 August 1980,[4] EPA published a rulemaking proposal which would have significantly amended the procedures used by the Agency for conducting adjudicatory hearings under Section 6 of FIFRA. In the preamble to this proposal, EPA stated that,

> The objective of [this] proposal [was] to make the Agency's procedures for identifying and assessing potential problem pesticides, and implementing regulatory decisions concerning them more open, responsive and efficient. The fundamental theme...is to create an integrated system in which decisions about registration or cancellation of problem pesticides are made in the RPAR process...and in which adjudicatory hearings are utilized primarily to probe and challenge decisions reached in the RPAR process, after appropriate

> screening to identify disputed fact questions which [could] profitably be illuminated through the use of trial-type procedures.[5]

The Agency alleged that "these changes [were] designed to enhance public participation in the process."[6] Yet, "EPA's proposed RPAR/Hearing Reform regulations [not only] unlawfully abridged the right of [registrants and other adversely affected persons][7] to an evidentiary hearing quaranteed by Section 6 of FIFRA"[8] and the Administrative Procedures Act (APA)[9] but also significantly restricted the rights of intervenors to meaningfully participate in a hearing, should a request for a hearing be granted by the Agency.

The ultimate effect of these proposed rules would have been to make public participation a hollow exercise in the formulation of pesticide regulatory policy "with the Agency internalizing most if not all of the critical value judgements involved"[10] in their regulatory decisions. Through the proposals put forward in this rulemaking the Agency sought to achieve an administrative/"functional" resolution to what is really a polycentric controversy by infusing a managerial decision-making model into the realm of public policy formulation. Where there exist,

> ...complex problems involving large numbers of interested parties the concept of a single best solution is misleading. Quantitative techniques of decision making are of great value in solving many problems; however, they offer little prospect of serving as an impartial, irrefutable arbiter of the conflicts of interest involved in large policy problems.[11]

I. PROPOSED RULES

The proposed rules contained three principal elements: (A) the merging of the RPAR process with the Section 6 Hearing, (B) the establishment of a screening mechanism for cancellation hearings, and,

(C) modifications of the rights of intervenors to fully participate in administrative hearings.

A) MERGING of the RPAR PROCESS with the FIFRA § 6 HEARING PROCEDURES

The Rebuttable Presumption Against Registration (RPAR) process, initiated in mid-1975, was "designed primarily as a mechanism for identifying pesticide uses which might pose substantial questions of safety...and providing for a relatively brief informal exchange between the Agency and other interested persons on...whether a substantial question of safety in fact existed."[13] It was never intended to replace pesticide cancellation hearings.

> (T)he agency implemented [the RPAR process] in response to industry complaints that the agency was deciding whether to issue notices of intent behind closed doors and to environmentalist complaints that it was scrutinizing too few pesticides.[14]

As the RPAR system presently operates, there are two stages in the proceedings that provide for the receipt of public comments and submission of rebuttal evidence: at the initial (PD 1) publication phase and at the final publication (PD 2/3) phase. Should the Administrator decide that an RPAR has not been successfully rebutted, under the proposed regulations the RPAR "record" would automatically form the evidentiary foundation for any future cancellation proceeding.[15] This, i effect, constitutes the fusion of the RPAR process with the FIFRA §6 Hearing procedures. As the Agency stated in the preamble to this proposal,

> (t)he fundamental theme...is to create an integrated system in which decisions about registration or cancellation of problem pesticides are made in the RPAR process...[wherein]

> adjudicatory hearings [would be] used...to probe and challenge decisions reached in the RPAR process.[16]

The RPAR record furthermore, (irrespective of how irrelevant, immaterial or fallacious the information contained therein), would make up the entire body of evidence for such a § 6 proceeding "unless certain procedural hurdles were overcome to demonstrate that introduction of additional evidence (through direct testimony or cross-examination) was warranted."[17]

B) THE SCREENING TESTS

Under the proposed regulations, a registrant or other party adversely affected by a Section 6(b) notice would not be automatically entitled to an adjudicatory hearing upon timely request to the agency for a hearing. "The proposed regulations require the proponent of a formal hearing to identify those issues for which adjudicatory procedures would aid in clarifying issues and answering factual questions."[18] The keystone of the Agency's hearing reforms was a set of "screening tests" which would have been applied in hearings following a notice of intent to cancel.[19] The screening tests would have been used to determine the extent to which the Agency would permit the introduction of additional evidence, cross-examination, and referral of scientific issues to the National Academy of Sciences. As the preamble stated:

> The broad purpose of these tests is to ensure that formal hearings are focussed on the types of issues they are best qualified to address, and do not expend time and resources on matters which have little prospect of being further clarified or achieving decisional significance.[20]

The following criteria were to be used to guide the Agency's application of the screening tests:

> [A] party requesting further proceedings [must] show that: [i] a genuine and substantial question of fact is involved; [ii] the proceedings at issue are likely to resolve the issue; and [iii] the resolution of the issue one way or another has the potential to change...the outcome of the proceedings.[21]

Should a party request "further proceedings" (that is, an evidentiary proceeding for the introduction of additional evidence, through direct testimony or cross-examination) he would be under an affirmative duty to demonstrate to the presiding officer (an ALJ) that each item which he wished to introduce into evidence had satisfied each of the screening tests. The screening tests included,

> (1) That there [was] a genuine and substantial issue of fact for resolution.
>
> (2) The factual issue [was] not one which may properly be decided on the basis of official notice of matters within the expert knowledge of the Agency.
>
> (3) The factual issue [was] capable of being resolved by available and specifically identified reliable evidence. A request [would] not be granted on the basis of mere allegations or denials or general descriptions of positions and contentions.
>
> (4) Good cause existed for not presenting the material in question to the Agency for inclusion in the administrative record. "Good cause" mean(t) either that the material was not available at the stage of the RPAR process at which it should have been presented, or that the material [was] of such a nature that it [could] only be presented meaningfully in a trial-type hearing.
>
> (5) The material in question if accepted as valid would be adequate to justify resolution of the factual issue in the way sought by the person. A request [would] be denied to the extent the Administrator concluded that, even assuming the truth and accuracy of all of the data and information sought to be introduced, they [were] insufficient to justify the factual determination urged.
>
> (6) Resolution of the factual issue in the way sought...[was] adequate to justify granting some or all of the relief sought by that person. A request [would] not be granted to the extent the

> Administrator concluded that his action would be the same even if the factual issue were resolved in the way sought.[22]

EPA's use of the screening tests, placing the burden of identifying material issues of fact upon hearing proponents, closely paralleled the "summary judgment"[23] approach used by FDA in determining whether an adjudicatory hearing was justified.[24] The Agency claimed that its proposed screening tests would be used to "formalize and define the standards"[25] used by the hearing officer to "structure the course of a formal proceeding..."[26] Yet, in essence, the screening tests would have the ultimate effect of denying an adjudicatory hearing to an adversely affected party except in those cases, and for those issues, "for which trial-type procedures [could] functionally aid the agency."[27]

C. MODIFICATIONS OF THE RIGHTS OF PUBLIC PARTICIPATION

In the preamble to this proposed rulemaking, the Agency stated that "the objective of this proposal is to make the Agency's procedures...more open, responsive and efficient."[28] The Agency further stated that, "the merger of the RPAR process and the Registration Standards System would not affect...the opportunities for public participation in the RPAR process."[29] Yet, while espousing the goal of making the regulatory process more open, responsive, and efficient, the EPA was, in reality, describing "a process which [was] more closed than before."[30]

STANDING

There are two key tests for standing: "injury in fact" and "whether the particular interest asserted is arguably within the zone of interests to be protected or regulated by the statute or Constitutional guarantee in question." If a person has rights recognized by law and has been injured or threatened with injury by a governmental action then he has a legal right to challenge that action. As used in the context of standing to request a hearing in response to a notice of intent to cancel a registration the EPA defined the term "adversely affected person" very narrowly. An "adversely affected" person included "only persons who want to prevent proposed actions from becoming effective, and to litigate with the Agency an unreasonable adverse effects problem."[31] In other words, an adversely affected person is one who has an economic or property interest in the regulatory outcome of a cancellation decision. "The term does not include persons who believe that the Agency did not go far enough and who therefore want the Agency to take actions more restrictive than those"[32] which the Agency has proposed to do.

The proposed regulations would also have severely limited participation by environmental groups, and others who might be adversely affected by the notice but do not belong to the class of persons who are registrants. The Consititutional test for standing is the "injury in fact" test. In the case of Sierra Club v. Morton[33] the Supreme Court held that injury in fact "may reflect 'aesthetic, conservational, and recreational' as well as economic values." EPA's economic injury requirement for standing would be contrary to the liberalized law of standing reflected in the Sierra Club decision. "(T)he proposal would

afford this...class of persons an opportunity to demonstrate to the Administrator...that an action which he has proposed is inadequate."[34] This demonstration would be "based on a showing [by them] that the RPAR has failed to satisfactorily resolve substantial factual issues which could have a significant impact on the final regulatory outcome."[35]

With respect to intervention rights, environmental groups would only be afforded an opportunity to support the Agency's position at a formal hearing, and "arguing that a section 6(b)(2) hearing [notice of intent to hold a hearing to determine whether (a pesticide's) registration should be cancelled or its registration changed] is necessary to explore their contention that the Agency did not go far enough."[36] The net result of the Agency's narrow definition of standing would be the effective elimination of meaningful public participation in pesticide regulatory decisions and any statutory right they might have had to fully participate in a Section 6 hearing. This state of affairs had led one commentor to observe that,

> (d)espite the lip-service repeated throughout the Preamble to the "objective of...making regulatory decisions...more open, responsive, and efficient,"[37] in fact this proposal describes a process which is more closed than before. The increased public participation mentioned as a goal in the Preamble[38] is upon analysis a triumph of form over substance. Public participation, other than by manufacturers and users, is in essence the right to submit written comments...The proposal eliminates the opportunity of the public at large to put the Agency, or the Agency and pesticide industry and growers, to their proof. Their facts and value judgements will never be publicly subject to challenge, except possibly by intervenors in industry/grower-requested hearings, whose scope will be strictly limited to issues delineated by the Agency...[39]

Hence, the proposed rules would have effectively closed members of the public (other than registrants and agricultural groups) out of the cancellation/suspension decision process on suspect pesticides.

II THE RIGHT TO A FORMAL ADJUDICATORY HEARING GUARANTEED BY FIFRA AND THE APA WOULD BE ABRIDGED BY THE PROPOSED RULES

The proposed hearing regulations were an attempt to "modernize" the historical approach used by EPA to reach pesticide cancellation/suspension decisions. Traditionally, decisions to revoke or amend a pesticide's use registration(s) were reached after a formal adjudicatory hearing. By effectively eliminating the access of registrants and other adversely affected persons to a meaningful formal hearing, the Agency attempted to elevate the function of an RPAR review into that of an informal rulemaking. In the Preamble to the proposed regulations the position of the Agency was "that the RPAR process...with its multiple opportunities for public input and with its requirements of carefully articulated decisions based on specifically identified records--together with the requirements of Part 164 for screening requests for further proceedings...[would] comprise the requisite 'public hearing' for purposes of judicial review."[40] It is thus not surprizing that the majority of the comments received by the Agency on this proposal addressed the conflict between the EPA's proposed hearing reforms and the type of hearing FIFRA and the APA require.[41]

As legal authority for proposed "improvements" of its current Rules of Practice, the Agency had relied upon the 1975 and 1978 FIFRA amendments and accompanying Congressional guidance.[42] As noted in Chapter 2, the 1975 and 1978 amendments established certain procedural requirements for the Administrator to fulfill prior to making a determination as to what type of regulatory action to take against a

problem pesticide. These amendments did not, however, alter or compromise the fundamental role of adjudicatory hearings under FIFRA.

The legal authority for an adjudicatory hearing to be held pursuant to Section 6 of FIFRA is contained in the provisions of the Administrative Procedures Act (APA).[43] An "adjudication" is defined by the APA as an "agency process for the formulation of an order."[44] "In every case of adjudication required by statute to be determined on the record after opportunity for an agency hearing,"[45] the procedures for formal hearings "in accordance with sections 556 and 557"[46] are to be applied.

Sections 6(b)(1) and 6(b)(2) of FIFRA provide that upon publication by the Administrator of a notice of intent to cancel or a notice of intent to hold a hearing, a person "adversely affected" by the notice(s) may request a hearing. Such a hearing under section 6(d) "shall be held...for the purpose of receiving evidence relevant and material to the issues raised by the objections filed by the applicant or other interested parties..."[47] At the end of this hearing the Administrator "shall issue an order" which "shall be based only on substantial evidence of record of such hearing..."[48]

> Accordingly, because a hearing conducted under Section 6(b) requires an order to be determined on the record, the formal adjudication procedures required by the APA are applicable.[49]

Adjudicants (i.e., persons adversely affected by an agency action) are afforded the following procedural rights by the APA:

> A party is entitled to present his case or defense by oral or documentary evidence, to submit rebuttal evidence, and to conduct such cross-examination as may be required for a full and true disclosure of the facts.[50]

Section 6(d) of FIFRA states that the public hearings held pursuant to the provisions of section 6(b) of the Act shall be "for the purpose of receiving evidence relevant and material to the issues raised by the objections [to an agency action filed by a registrant or other adversely affected person]."[51] The Agency states that "a formal evidentiary public hearing" under Section 6 would begin with the publication in the Federal Register of a notice to hold a hearing.[52] Yet, because of the procedural hurdles contained in the proposed "screening tests" mentioned earlier, the type of hearing guaranteed by the APA and FIFRA would be held only when the Agency chose to hold a hearing. This result is clearly contrary to the affirmative duty placed upon the Agency by law to hold an adjudicatory hearing which would result in an order.

A substantial body of federal case law affirms the right of adversely affected persons to a formal adjudicatory hearing contemplated by Section 6 of FIFRA. In EDF v. Ruckelshaus, Chief Judge Bazelon noted that the statutory scheme of FIFRA contemplated that,

> when [the Administrator] reaches the conclusion that there is a substantial question about the safety of a registered item, he is obliged to initiate the statutory procedure that results in...a public hearing...Public hearings bring the public into the decision-making process, and create a record that facilitates judicial review.[53]

In United States v. Florida East Coast Railway Co.,[54] the Supreme Court analyzed the procedural significance of statutory language which required an agency hearing on the record. While the Court did not construe magical significance to the terminology "on the record" and "after hearing" it did establish that these words would trigger the applicability of section 556 of the APA. (Section 556 of the APA, it will be remembered, prescribes the procedures to be followed by agencies

engaged in formal rulemaking and adjudication.) In Seacoast Anti-Pollution League v. Costle,[55] the First Circuit stated that,

> the FWPCA requires the EPA to afford an opportunity for a public hearing. We do not believe that an opportunity to submit documents constitutes a public hearing. Nor do we believe that the Administrator can comply with the statute merely by taking some evidence at a public hearing and then taking the rest in written form.[56]

Finally, in Pactra Industries, Inc. v. Consumer Product Safety Commission[57] (CPSC), the CPSC attempted to regulate the sale of self-pressurized containers, containing vinyl chloride monomer used as a propellant gas, (classifying them as "banned hazardous substances"), by denying requests to hold a formal hearing on this action. CPSC felt that a hearing was unnecessary since the result of the regulation would not be changed by a hearing. The Ninth Circuit held that where material issues of fact were raised by an objecting party they could not be dismissed by the Commission without first conducting a formal hearing and compiling a formal administrative record.

It is clear that the Agency wishes to move away from trial-type to informal procedures in making regulatory decisions on "problem" pesticides. Yet, in as much as FIFRA, the APA and legal precedent contemplate that an "order" be the result of an adjudicatory hearing, why then did EPA try to amend its current rules of practice in a way that would effectively eliminate the opportunities for formal hearings and public participation? The conclusion one comes to is that the Agency wished as a matter of Agency policy to make cancellation decisions through settlement agreements with pesticide registrants. By making it very difficult, if not impossible, for a registrant to obtain an agency hearing on a pesticide registration decision and by, simultaneously, depriving persons adversely affected by a given

regulatory decision the right to effectively and fully participate in agency decisions, the result sought by the agency--to make regulatory decisions through settlements with the registrants--becomes a self-fulfilling prophecy.

The agency clearly recognizes in their current rules of practice that environmental groups and others have a right to participate fully in cancellation/suspension hearings.[58] But this right to public participation in agency actions is a double-edged sword. While it affords a person adversely affected by an agency action his "day in court" it can also "prevent settlement agreements between registrants and the Agency that fall short of the group's expectations."[59]

> An environmental group that is a party to a cancellation proceeding has a statutory right to appeal to a federal court of appeals any aspects of an Agency's final decision that it dislikes, and the court must remand any aspects of the final order not supported by substantial evidence. Because there is no completed hearing prior to a final Agency order effectuating a settlement between EPA and a registrant, there is no record on which the court can base its review in determining whether substantial evidence exists to support the Administrator's decision. Therefore, an "adversely affected" environmental group can have the order remanded so that it may present arguments under the statutory criteria for cancelling more uses of the pesticide. In this way, the environmental group can exercise a "veto" over settlement.[60]

While the foregoing statement directs its comments specifically to the veto power environmental groups can exert in any pesticide settlement decisions it can also be applied to user groups, labor organizations and consumer groups. The only way to neutralize the veto power of these potentially adversely affected persons is to deprive them of their right to fully and effectively participate in pesticide regulatory decisions.

The decision of EDF v. Costle[61] held that the language of section 16(b) of FIFRA (judicial review of agency actions) does not obligate the agency to hold a hearing responsive to an environmental group's assertions that the agency improperly failed to cancel all unsafe uses of a carcinogenic pesticide (chlorobenzilate).[62] The court in the chlorobinzilate case focussed on the narrow procedural question of which federal court was appropriate to review a challenge to an Agency non-cancellation decision. The court of Appeals for the D.C. Circuit held that:

> ...persons seeking more stringent regulation may sue in the district court without first enduring an administrative hearing; those complaining of regulation as too strict must first exhaust their administrative remedy of a formal hearing before seeking judicial review.[63]

In commenting on the import of this decision with respect to the Agency's proposed regulations, Thomas O. McGarity (former attorney for the Office of General Counsel, US EPA) stated that,

> (t)he proposed regulations constitute the final step in effectively closing members of the public other than registrants and agricultural groups out of the pesticides cancellation process. The regulations consolidate [the chlorobenzilate victory] and thereby limit environmental groups to submitting evidence and arguments in the RPAR process, supporting EPA's position at the formal hearing, and arguing that a section 6(b)(2) hearing is necessary to explore their contention that the agency did not go far enough. Environmental groups cannot object to the adoption of one of the novel procedures...The regulations also let the agency off the hook with respect to deadlines... Hence, the proposed regulations will govern the dialogue between EPA and the registrants.[64]

Given the proposed screening tests which a registrant would have to satisfy in order to be granted a hearing by the Agency he would perhaps be more willing to seek other forums in reaching a registration decision with the Agency.

This situation has led the EDF to assert that,

> (t)he Agency's proposal would put enormous discretion in the hands of EPA personnel in the area of pesticide regulation, where history clearly shows that there is a trend in government regulation to support and indeed become captured by the regulated industry.[65]

III. THE NEED FOR PUBLIC PARTICIPATION IN PESTICIDE REGULATORY DECISIONS

Pesticide regulatory decisions are typically polycentric issues. They are often,

> characterized by a large number of possible results and by the fact that many interests or groups will be affected by any solution adopted.[66]

Where, as in the area of pesticide regulations, the issues are complex and the costs of making an incorrect decision are unknown and unknowable, the Agency should actively encourage public participation.

Several important values are served by an agency actively promoting public participation in its decision-making. One of the most fundamental values is that of fairness. This is the right of any person who might be adversely affected by an agency action to have his day in court. "The 'right to be heard' before government acts adversely to important private interests is well-established in American constitutional law."[67] As was discussed earlier, both FIFRA and the APA fully protect this fundamental right.

The decisions weighing the inherent benefits and risks of pesticide use are highly complex. No one agency, or member of an agency, is omniscient. This being true, a second value which can be served by encouraging full-public participation is that of accuracy.

All regulatory decisions are fundamentally political decisions. Regulatory agencies use scientific/technical information to rationalize their policy pronouncements. In the DDT case, discussed in Chapter 3, the Secretary of Agriculture used the lack of conclusive human health effects information to support his decision not to stringently regulate DDT. Less than 18 months later, the new Administrator of the EPA, William Ruckelshaus, decided to cancel almost all registered uses of this insecticide. He based his cancellation decision on the same data base that was considered insufficient, by the Secretary of Agriculture, to support such a regulatory outcome.

The,

> (q)uantitative techniques of decision-making are of great value in solving many problems...(but) they offer little prospect of serving as an impartial, irrefutable arbiter of the conflicts of interest involved in large policy problems.[68]

Hence, policies must enhance the participation by the broadest spectrum of experts outside of the agency whose unique knowledge could clarify the accuracy of regulatory determinations.

Finally, public participation lends balance to regulatory decision-making. The information upon which a regulatory decision is based is often incomplete, inadequate or speculative. Thus, determining the weight to be placed upon one set of information as opposed to another is at best a subjective exercise. "Because decisions are left to regulators with often only delphic congressional guidance, agency procedures should facilitate broad participation and vigorous debate to assure agency understanding of diverse viewpoints."[69]

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11. Bauer, "The Study of Policy Formation: An Introduction," in R. Bauer and K. Gergen, The Study of Policy Formation 1, 9 (1968).

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13. Supra, note 6, at 52630.

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23. 41 Fed. Reg. 51706, 51721-51722 (1976).

24. Supra, note 18, p. iii.

25. Supra, note 4, at 52645.

26. Ibid.

27. McGarity, supra, note 3, at 768.

28. Supra, note 4, at 52628.

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30. EDF Comments, supra, note 10, at 2.

31. Supra, note 4, at 52639.

32. Ibid.

33. Sierra Club v. Morton, 405 U.S. 727 (1972).

34. Supra, note 4, at 52639.

35. Ibid.

36. McGarity, supra, note 18, at vi.

37. Supra, note 28.

38. Supra, note 29.

39. EDF Comments, supra, note 10, at 2.

40. Supra, note 4, at 52650.

41. See, for example, comments of CSMA and NACA on this issue.

42. Ibid., at 52630-52635; 52643.

43. 5 U.S.C. 551; 554; 556; 557.

44. 5 U.S.C. §551(7) emphasis added.

45. 5 U.S.C. §554(c)(2).

46. 5 U.S.C. §554(c)(2).

47. 7 U.S.C. §136d(d).

48. Ibid.

49. NACA comments, supra, note 17, fn. 3, at 9.

50. 5 U.S.C. § 556(d) (adjudication begets orders; rulemaking begets rules).

51. 7 U.S.C. § 136d(d).

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53. EDF v. Ruckelshaus, 439 F. 2d 584, 595 (D.C. Circuit, 1971) [DDT case].

54. United States v. Florida East Coast Railway Co. 410 U.S. 224, 238, 241 (1973) [case deciding whether formal rulemaking was required in a ratemaking proceeding under the Interstate Commerce Act.]

55. Seacoast Anti-Pollution League v. Costle, 572 F. 2d 872 (1st Cir, 1978) (meaning, under the Federal Water Pollution Control Act, of the requirement for an "opportunity for a public hearing" to be afforded operators of effluent sources prior to an effluent limitation).

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Conclusion

Public interest/advocacy organizations played a significant part in the political decisions to regulate problem pesticides. Through the use of adversarial procedures they shed light on the value judgements and political trade-offs inherent in these controversies. Regardless of how one views the results of the regulatory decisions made on DDT, A/D and C/H, public interest organizations put pressure upon regulatory agencies to support their regulatory decisions by a full and complete weighing of the factual and policy issues raised by the controversy.

While it is readily acknowledged that full public particpation in formal adjudicatory hearings is time-consuming, the problem cannot be equitably solved in the manner evisioned by the Agency in this proposal. One solution proposed to shorten the length of these hearings, put forward by a former EPA attorney,

> would be the establishment by the Administrator of a maximum time limit for the hearing in his notice of intention to cancel or deny registration or to change a classification, the time to be divided equally between proponents and opponents... (This would) force the parties to budget their time carefully, to present only their best witnesses, and to discuss only the most important issues, thereby focussing the attention of the decisionmaker on the strongest arguments for each side.[1]

There is no reason why the EPA cannot adopt this modest proposal in its conduct of adjudicatory hearings under FIFRA, if it were truly committed to increasing the efficiency and accuracy of its pesticide regulatory decisions.

On July 22, 1981, Dr. John Hernandez, Deputy Administrator of the EPA, in testimony before the U.S. House of Representatives, informed the Subcommittee on Department Operations, Research and Foreign Agriculture

that the Agency would not go forward with any amendments to their Rules of Practice governing hearings pursuant to section 6 of FIFRA.[2] No explanation was given to the committee by Dr. Hernandez for this decision. Furthermore, a recent committee print of the draft House Bill to amend FIFRA section 3(c)(3) contains the following provision:

> The Administrator shall not by regulation or otherwise utilize a public interim administrative review process to abridge entitlement to a public hearing in accordance with section 6 of this Act.[3]

Should this provision become law it would effectively prohibit the Agency from proposing rulemaking like the one discussed in Chapter 4.

As pesticide legislation evolved, from 1910 to the present, a greater and greater emphasis was placed upon product safety. In the event that a pesticide, when used as directed, caused unwanted side effects, provisions were included in FIFRA to cancel or suspend that pesticide's uses. Since 1972, the FIFRA (actually FEPCA) established the right of pesticide registrants, applicants, and others who might be adversely affected by a regulatory decision on a pesticide, or adversely affected from exposure to a registered pesticide to petition the Agency to hold a public hearing on the merits.

Public interest organizations (primarily EDF) played an important role in the cancellation/suspension decisions on carcinogenic, biopersistent insectides. The registrations of DDT, aldrin/dieldrin and chlordane/heptachlor were cancelled through the teamwork of sympathetic agency attorneys and environmental organization litigants. "There would have been no cancellations or suspensions without the OGC to act as a catalyst."[4] In the appellate court review of these three pesticide decisions, EDF established that public interest organizations have a right to challenge regulatory actions; that notices of intent to cancel

a registration shall be issued whenever a substantial question of safety existed on a given pesticide; that a pesticide registration shall be suspended whenever the use of that pesticide created an imminent hazard situation, and, certain principles of carcinogenicity which could be prospectively applied to regulatory decisions on presumptively carcinogenic pesticides.

Through its rulemaking proposal, the EPA sought to deny adversely affected persons their statutory right to a public hearing. More importantly, the Agency sought to make public participation a meaningless exercise in the formulation of pesticide regulatory policy. It is useful to remember that,

> [W]hen Congress creates a procedure that gives the public a role in deciding important questions of public policy, that procedure may not lightly be sidestepped by administrators...The statutory scheme contemplates that these questions will be explored in the full light of a public hearing and not resolved behind the closed doors of the Secretary. There may well be countervailing factors that would justify an administrative decision, after committee consideration and a public hearing, to continue a registration despite a substantial degree of risk, but those factors cannot justify a refusal to issue the notices that trigger the administrative process...Public hearings bring the public into the decision making process, and create a record that facilitates judicial review. If hearings are held only after the Secretary [and now the Administrator] is convinced beyond a doubt that cancellation is necessary, then they will be held too seldom and too late in the process to serve either of those functions effectively.[5]

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APPENDIX 1

APPENDIX I[1]

Major Events in the History of Pest Control

Date	Event
400,000,000 B.C.	First Land Plants
350,000,000 B.C.	First Insects
250,000 B.C.	Appearance of Homo sapiens
12,000 B.C.	First records of insects in human society
8,000 B.C.	Beginnings of agriculture
4,000 B.C.	Silkworm culture in China
2,500 B.C.	First records of insecticides
1,500 B.C.	First descripton of insect pests
950 B.C.	First description of cultural controls (burning)
300 A.D.	First record of use of biological controls (predatory ants used in citrus orchards in China)
1650-1780	Burgeoning of insect descriptions (after Linnaeus) and biological discoveries in the Renaissance
1732	Farmers first begin to grow crops in rows to facilitate weed removal
1750-1880	Agricultural revolution in Europe
early 1800s	Appearance of first books and papers devoted entirely to pest control
1840s	Potato blight in Ireland (no controls available to curb disaster)
1870-1890	Grape phylloxera and powdery mildew controlled in French wine country (introduction of Bordeaux mixture; Paris Green; use of resistant root stalks and grafting)
1880	First commercial pesticide spraying machine

1888	First major biological control agent importation success (Vedalia beetle imported to U.S. from Australia for control of cottony cushion scale in citrus in California)
1890s	Introduction of lead arsenate for insect control
1896	Recognition of arthropods as vectors of human disease
1899-1909	Development of strains of cotton, cowpeas, and watermelon resistant to *Fusarium* wilt (first breeding program for pathogen resistance)
1901	First successful biological control of a weed (lantana in Hawaii)
1910	The Insecticide Act of 1910 (first U.S. pesticide statute)
1912	U.S. Plant Quarantine Act
1915	Control of disease-vectoring mosquitoes allowed completion of Panama Canal
1921	First aircraft pesticide spray operation (in Ohio, for Catalpa sphinx)
1929	First area-wide eradication of an insect pest (Mediterranean fruit fly)
1930s	Introduction of synthetic organic compounds for plant pathogen control
1939	Recognition of insecticidal properties of DDT
1940	Use of milky disease to control Japanese beetle (first sucessful use of pathogen for insect control)
1940s	Organophosphates developed in Germany; Carbamates in Switzerland

1942	First quccessful breeding program for insect pest resistance in crop plants (introduction of wheat strain resistant to Hessian fly)
1944	First hormone-based herbicide (2,4-D)
1945/1946	DDT marketed as "miracle" insecticide
1946	First report of insect resistance to DDT (housefly in Sweden)
1947	The original Federal Insecticide, Fungicide, and Rodenticide Act enacted
1950s, 1960s & 1970s	Widespread development of resistance to DDT and other pesticides
1950s	First applications of systems analysis to crop pest control
1959	Introduction of concepts of economic thresholds, economic injury levels and integrated control
1960	First insect sex pheromone isolated, identified and synthesized (gypsy moth)
1962	Publication of Silent Spring
1970	EPA created--given control over pesticide regulation from USDA
1972	Banning of DDT
1972	FIFRA amended and renamed the Federal Environmental Pesticide Control Act (FEPCA)

REFERENCE FOR APPENDIX

1. Flint, Mary Louise and Robert van den Bosch: A Source Book on Integrated Pest Management, DHEW Publication, 1980.

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