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ENVIRONMENTAL POLICY MAKING: ACT NOW OR WAIT FOR MORE INFORMATION?

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ABSTRACT:

This paper explores a central, paradigmatic problem in environmental decision making-- the problem of timing. Do we act now or do we hold out for more information? The frequently-voiced preference for waiting, I suggest, is based upon a strong but unstated assumption-- namely, that environmental policies are irreversible. That is, interventions by regulatory agencies impose large, sunken costs on private firms and consumers that cannot later be taken back.

Reviewing past experience with diesel emission controls, pesticide regulation, chloroflourocarbon limits, and other problems in environmental regulation, I find that the preference for waiting is often unwarranted. The longer the regulatory agency delays action, the more time private agents have to make large, sunken investments in the prevailing technology. If the agency delays too long, the stakes get too high.

While a strategy of delay is often framed as an opportunity to gather more information, I suggest that regulatory intervention is hardly incompatible with continued research. In fact, some regulatory actions are themselves a form of research because they provide essential information about the benefits and costs of future regulatory decisions

My analysis points toward a style of environmental regulation in which agencies take small, incremental regulatory steps at the early stages of a problem. These small steps would be designed to impose minimal sunken investments in compliance, yet provide essential information on the uncertain benefits and costs of future interventions.

INTRODUCTION

Environmental policy making is a dynamic process. Rarely do regulatory agencies make once-and-for-all choices between action and inaction. Instead, they choose over and over between degrees of action and waiting. The information base for such choices-- scientific, economic, political-- changes all the time.

This dynamic quality of environmental decisions poses serious problems for benefit-cost analysis. To evaluate a contemplated regulatory intervention, it is no longer enough to compare the intervention's currently estimated benefits and costs. In fact, it is insufficient to assess the whole future stream of expected benefits and costs. The decision maker also needs to estimate the benefits and costs of regulating later on as opposed to acting right now. If the regulator decides to act now, its experience with implementation may be informative about the costs and benefits of later policy choices, including future rescission of the regulatory action. In deciding to act now, the environmental decision maker thus needs to assess the future benefits and costs of taking back its mistakes.

The idea that policy choices are dynamic is hardly new. Most public policy decisions-- in fact most individual

decisions-- are dynamic ones. When a public utility commission disapproves a requested rate increase, it contemplates the benefits and costs of approving the increase later on. When a stock holder decides not to sell his holdings, he considers the benefits and costs of selling later. The same goes for seeking another job or deciding to go on a diet.

Environmental policy, however, is an extreme case of dynamic decision making. The reason is that regulatory decisions about environmental hazards are routinely made in the face of huge uncertainties-- uncertainties in estimates of health risks, in mechanisms of disease, in the extent of exposure, in the costs of risk control. Under such extreme uncertainty, the appearance of even a modicum of new data can swamp the decision maker's prior beliefs concerning the costs and benefits of regulatory intervention. As a result, regulatory action on suspected hazards can be triggered or stifled by the issuance of preliminary toxicological findings, by false-alarms concerning the measurement of environmental contaminants, by leaks of draft reports of blue-ribbon panels.

In the conventional model of research, repeated measurements tend to improve the precision of our estimates of benefits and costs. But with the extreme uncertainties encountered in environmental decisions, new research

findings can pose unexpected contradictions, thus enhancing rather than reducing uncertainty.

My task in this paper is to explore, at least in a preliminary way, these dynamic complications of environmental policy making. My method of analysis is essentially anecdotal. That is, I offer some generalizations and then cite selected case studies for support. The hypotheses generated in this paper need independent, more systematic testing on a representative sample of decisions faced by regulatory agencies.

In the next section, I set up the central, paradigmatic problem in the dynamics of environmental decision making-- that is, the problem of timing. Do we act now or do we hold out for more information? The frequently-voiced preference for waiting, I suggest, is based upon a strong but unstated assumption-- namely, that environmental policies are irreversible. That is, interventions by regulatory agencies impose large, sunken costs on private firms and consumers that cannot later be recovered.

I then inquire further about the realism of the irreversibility assumption. I find that in many cases, a contemplated environmental policy can grow more irreversible with continued delays. There are two mechanisms for this phenomenon of growing irreversibility. First, an environmental problem in its early phases may be amenable to

partially reversible interventions, such as restrictions on use or access, product labelling, or pollution fees. But if the problem gets a lot worse later on, then truly draconian, irreversible actions may be required. Second, regulation is a game between governmental agencies and the private sector. The longer the regulatory agency delays action, the more time private agents have to make large, sunken investments in the prevailing technology. If the agency delays too long, the stakes get too high.

In a subsequent section, I probe further into the issue of "research." While a strategy of delay is often coupled with a decision to invest in new data collection, I suggest that research is just as compatible with regulatory intervention. In fact, some regulatory actions are themselves a form of research because they provide essential information about the benefits and costs of future regulatory decisions. In principle, regulatory action can be a better investment in knowledge than pure research without intervention.

I thus propose that policy makers consider two types of questions when contemplating the benefits and costs of a proposed regulatory action: How irreversible is intervention? How informative is the intervention? In general, my analysis points toward a style of regulation in which agencies take small, incremental regulatory steps at

the early stages of a problem. These small steps would be designed impose minimal sunken investments in compliance, yet provide essential information on the uncertain benefits and costs of intervention.

IRREVERSIBILITY AND THE BIAS TOWARD WAITING

All too often one hears the following refrain from scientists and policy makers: We do not yet have sufficient information to take regulatory action. We would prefer to wait for better data to come in. We need more research.

This bias in favor of waiting and against action has been articulated in many forms. The following examples are illustrative.

> "It may be that a proportion of lung cancers in man are induced by tobacco smoke; at the moment we do not know, but let us be sure of our evidence before we scare our public." (Passey, 1953)

> "Thus, I conclude that in my personal view, given the current information, the banning of saccharin at this point in time is counterproductive, and I believe the ban should not be instituted until or unless some 'safer' nonnutrient sugar substitute is available." (Isselbacher, 1977)

> "DES could have been taken off the market immediately, without a hearing, if the FDA had declared it to be an imminent hazard to health. That is the only statutory basis for immediate withdrawal of a drug from the market without first offering a hearing. The agency went to the National Cancer Institute on this issue, and the NCI said that, in its judgment, DES was not an

> imminent hazard. The government's own scientists concluded that the risk was not of that magnitude. Therefore, there was no legal basis for taking that action." (Hutt, 1977)
>
> "EPA did not immediately suspend these uses [of ethylene dibromide as a grain and fruit fumigant] despite the carcinogenic potential because EPA management did not believe enough was known at the time about the risks from residues on food, the risks from substitute fumigants, or the risks from leaving crops and foodstuffs unprotected. ... It decided to await the results of studies then in progress." (Russell and Gruber, 1987)

Each of these statements is a variant on the same basic theme: Immediate action may be too costly in comparison to waiting. In Passey's view, the costs arose from scaring the public. For Isselbacher, the cost would be the absence of an alternative to saccharin. In the case described by Hutt, it was too costly to bypass standard regulatory procedure and ban diethylstilbestrol without a hearing. Russell and Gruber's discussion of ethylene dibromide suggests several types of costs, including the risks of substitutes for EDB.

All of the examples contain an implicit benefit-cost calculation. The benefits of a determination that smoking causes lung cancer, Passey argued, did not outweigh the costs of "scaring" the public. The cancer risks of saccharin, Isselbacher contended, were outweighed by its benefits as a non-nutritive sweetener.

But there is more to each of these examples than a one-time benefit-cost analysis. In each case, the decision to

act or wait was recurrent. In analyzing the benefits and costs of action and inaction, each writer needed to consider how such benefits and costs might change over time. The benefits and costs of action were really the benefits and costs of acting immediately as opposed to later.

Thus, Hutt's description does not imply that DES had no danger, merely that in NCI's opinion the danger was insufficient to act forthwith. Isselbacher likewise did not deny saccharin's cancer-causing potential. Instead, he urged action later, once a substitute was available. EPA did not deny the carcinogenicity of ethylene dibromide. Instead, the agency believed there was insufficient data for immediate suspension of the fumigant chemical.

This dynamic view of the decision-making process begs some hard questions: Why couldn't the FDA have banned DES immediately in 1971? If subsequent evidence proved contradictory, then couldn't the ban have been modified or lifted? Why couldn't EPA have immediately suspended ethylene dibromide's uses as a fumigant? Again, if subsequent data had shown extremely low residues in food stuffs, why couldn't the ban have been modified? Why couldn't the medical community (and manufacturers of cigarettes) have warned the public immediately in 1953 (and even earlier) of the serious, legitimate evidence that cigarette smoking may cause lung cancer? If further

research had shown otherwise, then couldn't a superceding statement of opinion have been issued?

Implicit in these examples is the assumption that an action now cannot be taken back-- more precisely, undoing the action is very costly. Thus, implicit in Passey's argument is the contention that it would be quitely costly for the public to recover from a false alarm about smoking and cancer. Implicit in Hutt's description is that the act of bypassing the normal hearing process on DES could have been a costly administrative and political error. It is an unstated assumption of irreversibility that creates the bias toward waiting.

The concept of irreversibility of decisions has not been considered in the literature on environmental policy-making. Yet economists have made a number of attempts to spell out its consequences-- especially in recent theoretical work in financial economics (Henry, 1974; Cukierman, 1980; Roberts and Weitzman, 1981; Baldwin, 1982; Bernanke, 1983; McDonald and Siegel, 1986; Majd and Pindyck, 1987).

In the economic models, a decision-maker is assumed to be continuously faced with three types of choices: to <u>invest</u>, to <u>proceed</u>, or to <u>abandon</u> a hypothetical project. Investing, on the one hand, is a noncommittal action. It may accelerate the arrival of new information about a

project's benefits and costs, but the project's ultimate fate remains undecided. On the other hand, the decisions to proceed with or to drop the project are assumed to be irreversible.

The assumption of irreversibility has a number simple consequences in the economic models. In particular, conventional static benefit-cost analysis is rendered misleading (Majd and Pindyck, 1987). Even if the expected benefits of a project exceed its expected costs at a particular point in time, the decision to proceed may be unwarranted. Instead, we need to modify our decision criterion to take into account the benefits and costs of waiting for more information. The modified decision rule is to take action only when expected benefits exceed costs by a fixed, predetermined amount. (Strictly speaking, we get this rule only when the stochastic process that generates new information is stationary. See, e.g., Roberts and Weitzman, 1981). Put differently, the expected net benefit of the project has to exceed an "option value" of waiting for more information.

These stylized, economic models of the wait-or-act decision have general application. The financial decision to proceed with or abandon a project is analogous to the public policy decision to approve or disapprove, say, a new drug application or a new clean-up technology. The

financial decision to invest parallels the regulatory decision to send back the drug or the technology for more study.

The critical issue in applying the economic models, however, is the validity of their assumption of irreversibility. We should not jump to label an environmental regulation as irreversible until we have actually measured the sunken costs expended to comply with the regulation.

In conducting such an empirical inquiry, we really need a typology of sunken costs. To take a first pass, I shall suggest three classes: producer compliance costs; consumer compliance costs; and credibility costs. The first two categories reflect responses by producers and consumers, respectively, to environmental policy decisions. Thus, banning saccharin might result in a permanent and costly shutdown in saccharin-producing facilities. Prohibiting the use of DES as a livestock fattening agent might result in permanent and costly changes in the consumer diet. Credibility costs, the third category, arise because policy decisions are interdependent. Consumers' and producers' responses to environmental policies dependent upon the credibility of the policy-making entity. If the FDA banned saccharin or DES immediately and if these actions turned out to be mistaken, then the agency's ability to enforce subsequent regulatory actions might be destroyed.

Still, we need to ask for hard evidence of that capital in the saccharin industry was nontransferable. We need to inquire whether consumers could go back to leaner meats if and when DES were reintroduced. We need to ask whether the credibility costs of policy mistakes all stacked in favor of waiting.

WAITING AND SUNKEN COSTS

The argument in favor of regulatory delay, we have seen, hinges critically on the proposition that government intervention may impose irreversible, sunken costs on private agents. In this section, I suggest that the irreversibility argument can be turned upside down: Waiting can have equally irreversible consequences.

When an potential environmental hazard is first recognized, its control may be amenable partially reversible interventions, such as restrictions on access or use, product warning or labelling, and pollution fees. But if the hazard later becomes quite large, then such small-scale interventions may be ineffective and only large-scale, irreversible interventions may be worth considering. Thus, the regulator who waits for more data runs the risk that only the most extreme, irreversible measures will be

available in the end. Acid rain and toxic waste disposal may be good examples of the problem of increasingly narrow regulatory choices.

It is no accident of nature that the costs of effective intervention grow larger when regulatory agencies delay action. Private economic agents, especially firms, have an incentive to make intervention costly. The longer the regulatory authority waits, the more breathing time firms may have to commit themselves to the suspect technology.

Diesel Emissions

Since the 1950s, the condensates from diesel fuel-burning engines were known to cause cancer in laboratory animals. Such particulate emissions were further known to contain carcinogenic polyaromatic hydrocarbons. There was, however, little sound epidemiological evidence on the cancer risks of workers exposed to such emissions.

In the late 1970s, in the face of increasing pressures for fuel economy, American automobile manufacturers announced plans to convert 25 percent of the light-duty passenger car fleet from gasoline to diesel-fuel burning engines. If implemented, such plans would raise population exposures to such emissions by an estimated factor of 1,000.

The auto makers' proposal stimulated new research into the combustion process and the physical chemistry of the particulate matter contained within diesel and other emissions. By 1979, EPA scientists had determined that the organic solvent extracts of diesel particulates were highly mutagenic in the Ames Salmonella assay. Directly mutagenic nitroaromatic compounds were identified as the likely culprits.

EPA lauched a major research program in laboratory testing of fossil fuel combustion products. The carcinogenicity and mutagenicity of diesel and other emissions were confirmed in multiple laboratory models. Mathematical extrapolations suggested a small individual risk of cancer, but the estimated number of exposed persons was quite large. There was renewed interest epidemiological studies of exposed workers, but very little hard evidence on humans was available. A study of London Transport Workers was negative, but of sufficiently low power that some lung cancer risk from diesel emissions was not excluded (Harris, 1983).

A scientific panel of the National Research Council could do no more than reiterate the substantial uncertainty about the health risks of the proposed diesel technology (National Research Council, 1981). Moreover, while the biological data base became more refined, the uncertainty on

population exposures grew. Changes in the relative prices of diesel and gasoline fuels, as well as unanticipated changes in consumer preferences made the large-scale introduction of diesel passenger cars less likely. What is more, there were continued uncertainties about the feasibility of effective, low-cost particulate control technologies.

In the face of all this uncertainty, EPA proposed immediate particulate emission standards for diesel cars (at a level of 0.6 grams per mile). This action hardly settled the issue, for it remained unclear whether the proposed standards should remain in effect, or whether they should be tightened in the future. At the time, a stricter standard (0.2 grams per mile) was contemplated. Even if particulate standards were to be tightened, the Agency still needed to know when to impose them.

By the early 1980s, EPA could reasonably conclude that diesel emissions had at least the potential to cause cancer in humans. With virtually no solid epidemiological evidence, however, the Agency could not draw definite conclusions about the extent of human cancer risk. From the purely scientific standpoint, the prudent decision was to wait for the results of newly commissioned epidemiological studies. Concrete results from such studies were expected within five years.

But EPA's decision was not so simple. The planned conversion to a diesel-driven auto fleet would require a major investment in a new engine technology. Auto makers could not simply modify the existing production technology for gasoline-burning engines. If diesels were to make up to 18 percent of new car sales by 1990, investments on the order of $3 to $4 billion would be required. Moreover, it was unclear whether auto makers might later be able to convert such diesel technology back to producing gasoline-burning engines. As the National Research Council reported, "Based on the current state of knowledge, an irrevocable decision by the EPA . . . could run a danger of costly mistakes." (National Research Council, 1982).

Anyway, what did the Agency really expect to get out of the additional planned research? EPA could reasonably conjecture that by 1985, retrospective studies of diesel-exposed workers might show an elevated risk of lung cancer. Such studies might bolster the case for regulation of diesel particulates. Still, high-dose exposures in the workplace could not be simply extrapolated to low-dose ambient exposures from tailpipe emissions. Moreover, detailed laboratory studies of the composition and biological actions of diesel particulate emissions might still not settle a key, lingering question: Did the apparently unique nitroaromatic constituents in the particulate extracts make diesel fumes a uniquely dangerous species of emissions?

What made EPA's regulatory dilemma so acute was not the laboratory discovery that diesel emissions were mutagenic, and not the paucity of direct, human evidence, but the announced intention of manufacturers to sink billions into a new diesel technology.

In fact, the Agency was engaged in a prototypical regulatory game with the car makers. The longer EPA waited for new information, the further down the diesel road the car makers would be. The investment in diesel technology would not be instantaneous, but gradual over a period of a decade or more. By the time EPA had sufficient information to satisfy the blue-ribbon scientific panels, the industry might have invested so much in diesel technology as to make tight emission controls too costly.

In this regulatory game, both EPA and the car makers knew the dilemma the Agency might soon face. Hence, car makers had a strong incentive to accelerate their investments in diesel technology, that is, to build up their sunken costs as rapidly as possible. While EPA and some auto companies were conducting their own biological research, information on the likely pace of such research was common knowledge. On the other hand, the car makers possessed far more information on the irreversibility of investments diesel production technology. In fact, EPA's lack of expertise in this area was perhaps its central difficulty in reaching a decision.

In the end, EPA stuck with its proposed emission controls, if only to avoid more drastic interventions later. As it turned out, however, the anticipated major demand for diesel cars never materialized, and the Agency bought more time to wait for new data.

Cyanazine

In order to obtain a registration for a pesticide under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA; 7 U.S.C. 136 et seq.), an applicant for registration must demonstrate, among other things, that the pesticide performs its intended function without causing "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." (Section 2(bb) of FIFRA). EPA, the enforcing agency, interprets this standard to require "a finding that the benefits of the use of the pesticide exceed the risks of use, when the pesticide is used in compliance with the terms and conditions of registration or in accordance with widespread and commonly recognized practice." (U.S. Environmental Protection Agency, 1988). If at any time EPA should determine that this benefit-cost standard has been violated, then the

Administrator may modify the conditions of registration or cancel the registration entirely.

In April 1985, EPA initiated a "special review" of all pesticide products containing the active ingredient cyanazine (U.S. Environental Protection Agency, 1985). The review (formerly called the "Rebuttable Presumption Against Registration" or RPAR process) was instigated by the recent finding that cyanazine produced teratogenic and fetotoxic effects in laboratory animals. EPA was proposing that a warning be added to the pesticide label concerning cyanazine's potential to cause birth defects in laboratory animals. Moreover, because the main route of occupational exposure was through skin contact, the product label was to specify that cyanazine's use was restricted to certified applicators or persons under their supervision.

EPA was also concerned about ground water contamination from agricultural uses of cyanazine. Preliminary monitoring studies had identified residues of cyanazine in a small percentage of sample wells from five states. While most positive samples showed cyanazine concentrations of 0.2 parts per billion, a small percentage were at levels close to 1 part per billion.

> "Cyanazine has the potential to move (leach) through the soil and contaminate ground water which may be used as drinking water. Cyanazine has been found in surface and ground water as a result of agricultural use. The Agency does not have the data necessary to assess the

> health risks associated with consuming drinking water which has been contaminated with cyanazine." (U.S. Environental Protection Agency, 1985)

Accordingly, the Agency imposed labelling requirements that advised users not to apply cyanazine to highly permeable soils or where the water table is close to the surface. It also required registrants to conduct ground water and surface water monitoring studies.

In a January 1987 review, the Agency proposed a number of additional requirements for cyanazine registration, including use of protective gloves, closed loading systems, and chemical-resistant aprons. The pesticide label was to include statements regarding the cleaning of protective gloves, and separate laundering of protective clothing. The label was to state that cyanazine was classified for Restrictive Use because "cyanazine has caused birth defects in laboratory animals and has been found in ground water." (U.S. Environental Protection Agency, 1987a)

By early 1988, however, new data suggested that cyanazine was not so serious a threat to ground water. In particular, further sampling from 200 wells in hydrogeologically vulnerable areas revealed no detectable residues. The Agency thus lifted its prior restriction on the spraying of cyanazine where the water table is high or the soil is highly permeable.

> "As a result of newly generated monitoring data and the previously available data, the Agency no longer believes that cyanazine has significant ground water contamination potential. Therefore, EPA no longer believes that ground water contamination should be a reason for classifying cyanazaine for Restricted Use. Therefore, all cyanazine labels will include a statement that cyanazine products have been classified for Restricted Use only because cyanazine has caused birth defects in laboratory animals. However, because some instances of contamination were reported in the earlier studies, the Agency believes the ground water advisory statement should remain on the label." (U.S. Environmental Protection Agency, 1988)

In the case of cyanazine, EPA altered its position several times as new evidence accumulated on the pesticide's potential toxicity and routes of environmental exposure. The Agency in fact reversed itself on the issue of ground water contamination. However, the only clear effect of these multiple regulatory changes was to alter the contents of the pesticide's warning label.

Ninety-six percent of the cyanazine produced in the U.S. was used as a herbicide on corn. About 3 percent was used on cotton and less than 1 percent was used on sorghum and wheat. About 14-16 percent of the total U.S. corn acreage was treated with cyanazine in 1982. Several close substitutes to cyanazine were readily available, and there was little evidence that switching to these substitutes would be costly.

EPA was thus in a position to make a series of incremental changes in its regulation of cyanazine use

without imposing large sunken costs on the private sector. Users of cyanazine were required to make investments in closed loading systems and protective equipment, but none of these investments was specific to a single chemical. Producers of cyanazine were required to reissue warning labels. But in the absence of an outright ban on the use of cyanazine, the question of irreversible, cyanazine-specific investments did not arise.

Ethylene Dibromide

Table 1 traces scientific developments concerning ethylene dibromide (EDB) from the 1910 to 1976. EDB was first used by lead antiknock producers in the 1920s. By the late 1940s and early 1950s, the compound was widely employed as a fumigant of imported fruits and vegetables, grain, storage silos, and grain milling machinery.

Data on EDB's acute and subacute toxicity go back to the early 20th century. The evidence on EDB arose from reports of accidental human exposure, and from studies of ingestion, inhalation and dermal exposure in various laboratory animals. By the mid-1960s, additional reports appeared on EDB's reproductive toxicity in farm animals. Still, residues of EDB remained essentially undetectable in the food supply.

Two things happened in the early 1970s. First, EDB was linked to mutagenicity and carcinogenicity. Second, the technology for detecting EDB markedly improved.

In 1971, EDB was found to be a direct-acting mutagen in the Ames mutation assay. By 1974, the chemical's genotoxicity had been confirmed in other experimental systems. At this time, scientists were increasingly interested in the possible role of genotoxic events in the genesis of cancer. The finding that EDB was a mutagen stimulated whole-animal carcinogenicity studies by the National Cancer Institute (NCI).

NCI's preliminary results showed that EDB was carcinogenic when directly instilled into the stomachs of rodents. To be sure, there was concern that the NCI results were somehow artefactual, since the experimentally-induced stomach cancers appeared near the site of EDB application. Still, the prospect of EDB's carcinogenicity changed the entire perspective on the chemical's risks. Now, EDB was a potential carcinogenic contaminant of the food supply. Since many scientists believed that there was no dosage threshold for carcinogenicity, it was possible that even traces of residual pesticide were causing cancer in humans.

By 1975, an EPA study had shown detectable gasoline station exposures in the range of 0.01 parts per billion (ppb), and manufacturing site exposures in the range of 10-

15 ppb. These findings heightened the concern over the long-term consequences of low-dose EDB exposures.

Table 2 picks up the chronology from 1977. The table displays not only the salient scientific developments, but also some key regulatory actions. The table constitutes a preliminary attempt to show the timing of regulatory decisions in relation to the emergence of new scientific and economic information.

Not shown in Table 2 are contemporaneous developments in the media and public opinion. As shown in the table, the scientific evidence on EDB's potential hazards continued to expand after 1977. Yet media coverage of EDB erupted only after the chemical was discovered in ground water in Florida, Georgia, California and Hawaii in 1983. In that year, EPA issued an emergency suspension of soil fumigation of EDB. In the following year, the Agency announced suspension of all further use of EDB in the production of grain products (Russell and Gruber, 1987).

EPA's suspensions of ethylene dibromide in 1983 and 1984 were not the first regulatory actions taken with respect to the pesticide. Nor did 1983 see the first instance of damning evidence on EDB. The question then arises: What exactly happened between 1977 and 1983?

By 1977 the International Agency for Research on Cancer had already classified EDB as an animal carcinogen and

mutagen. A review by the National Institute on Occupational Safety and Health (NIOSH) noted that EDB can interact chemically with DNA, the basic genetic material. Still, EDB had thus far been found to be carcinogenic in only one incomplete animal experiment. Moreover, attempts to identify elevated cancer rates among EDB-exposed workers were negative. If EDB in fact posed a cancer threat at low doses, the magnitude of the cancer risk remained uncertain.

In the face of this uncertainty, the Occupational Safety and Health Administration (OSHA) proposed a tightening of its EDB exposure standard for workers. EPA, in parallel, began a special review (Rebuttal Presumption Against Registration or RPAR) under FIFRA. The linchpin of EPA's regulatory analysis was a risk assessment, performed by its Carcinogen Assessment Group (CAG).

The CAG's initial risk assessment proved to be problematic. The initial dosages of EDB in the NCI oral gavage study-- which CAG relied upon-- proved to be too toxic, so the dosage schedule had to be reduced in mid-experiment. This changing dosage schedule complicated CAG's attempts at high-dose to low-dose extrapolation. Moreover, CAG's analysis predicted a substantial cancer risk from long-term EDB exposures at the levels seen among chemical workers. Yet limited surveys of EDB-exposed workers showed no evidence of a significant cancer increase.

By 1979, however, additional laboratory studies confirmed EDB's carcinogenicity. The chemical caused cancers by skin painting in mice, and an NIOSH-sponsored study showed cancers by inhalation in rats. By 1980, EDB was found to be carcinogenic in a separate NCI-sponsored inhalation study of rats and mice. In that year, the American Conference of Governmental Industrial Hygienists also classified EDB as a suspect human carcinogen.

EPA's special review (RPAR) continued in 1980. An internal study estimated the probable residue level for EDB in wheat bread derived from fumigated grain to be less than 0.1 ppb, with a realistic worst-case residue of 31 ppb. Based upon such exposure estimates and extrapolating from the original NCI oral gavage experiment in rodents, CAG projected a 0.03 percent increased lifetime cancer risk due to the dietary burden of EDB. The Agency proposed cancellation of EDB's use as a fumigants of stored grains, milling machinery and fruits and vegetables by mid-1983. It further ordered studies of potential ground water contamination.

By 1981, new measurements of EDB residues in fruit and grain products showed that previous estimates may have been misleading. One study found EDB residues of 36 ppb in biscuits. Another found 57 ppb in the edible portions of fumigated fruits. Concurrently, OSHA proposed a further

tightening in the occupational standard for EDB exposure; California imposed a temporary emergency occupational standard.

By 1982, EDB levels as high as 100ppb were found in three wells in Georgia. The California Department of Food and Agriculture (CFDA) estimated EDB residues in fumigated citrus fruits up to 210-880 ppb. By Spring 1983, CFDA had found EDB concentrations of 0.1-31 ppb at depths greater than 20 feet. By June 1983, EPA had been detected at levels of 0.02-5 ppb in 16 counties.

EPA moved in September, 1983 to suspend soil fumigation immediately. Based upon the new exposure data, as well as a reanalysis of the NCI oral gavage experiment, CAG revised the estimated lifetime risk from dietary EDB to 0.3 percent. In February 1984, the Agency suspended further use of EDB in the production of grain products. It did not order an immediate ban on the sale of all EDB-containing products. Instead, it issued recommended guidelines to the states for acceptable levels of EDB in currently marketed foods.

Why did EPA wait six years (from its initial review in 1977 until its emergency suspension in 1973) to take action on EDB? Evidence of EDB's toxicity was long-standing. Its mutagenicity was established in 1971; and its carcinogenicity was reported by 1977. While the initial NCI study needed confirmation, independent findings of

carcinogenicity were available by 1979. While EDB was initially thought to be virtually undetectable in the food supply, contrary evidence was available by 1981. Ground water contamination was an issue as early as 1980, when EPA commissioned a study by CDFA, and residues were found in wells as early as June 1982.

Perhaps it is unfair to juxtapose EPA's regulation of cyanazine during 1985-1988 with the Agency's drawn out response to ethylene dibromide during 1977-1984. By the mid-1980s, the Agency surely had learned how better to handle the procedural and notification burdens built into FIFRA, which was enacted in 1972. Still, the cyanazine case shows the Agency moving quickly in incremental, reversible steps to establish warning labels and restrictions on use. In the case of EDB, the Agency essentially got caught having to ban the pesticide late in the game, years after other federal and state agencies had moved on the problem. Had EPA accelerated the information-gathering process, especially in the measurement of food residues and ground-water contamination, then less extreme measures might have been necessary.

By 1984, the sunken investment in EDB had become enormous. There were $29 billion in grain stocks and $4.3 billion in manufacturer and retail inventories of grain products and baked goods. Between 50 and 60 percent of

stored grains and grain products likely contained detectable levels of EDB. Comminglying of grains during storage, transport and manufacture raised the possibility that nearly all such products had detectable levels of the chemical (Temple, Barker and Sloan and Economic Perspectives, 1984). Immediate removal of EDB-containing foods would be quite costly. In the end, EPA chose an intermediate course: suspension of use without confiscation of existing stocks of potentially EDB-contaminated food.

REGULATION AS RESEARCH

Scientists and policy makers may recommend delaying regulatory action until they see the results of current research. But the need to perform more research does not preclude concurrent regulatory intervention. EPA imposed a ground water advisory on cyanazine's label even as it sought further testing of pesticide residues. The Agency imposed a standard on particulate emissions from diesel-powered cars even as it awaited the results of epidemiological studies on diesel workers. While EPA did not restrict ethylene dibromide until 1983, earlier action should not have barred further toxicological and exposure studies.

In fact, there is no clear dividing line between regulatory intervention and research. The reason is that we learn from the experience of regulatory intervention. In some instances, the best way to assess the benefits and costs of regulation is to regulate and see what happens. By contrast, we may learn little or nothing by further delay.

Our experience with environmental controls may provide the best source of information-- and sometimes the only source of information-- on the costs of complying with even stricter controls. At issue here is whether the public or private sectors are best suited to perform the necessary research on new control technologies. When the development of new controls entails highly specialized or proprietary knowledge, it may be impractical for regulatory agencies to fund public research into cleanup technologies. Instead, the most effective way to instigate the necessary research is to impose environmental controls, thus changing the incentives of private firms.

Conversely, our experience with regulatory controls may be the best or only means of assessing the benefits of environmental regulation. The idea is that laboratory experiments can measure small-scale individual effects, while environmental controls operate on a large scale. Thus, laboratory experiments and meteorological modelling can only imprecisely gauge the aggregate effect of curbing

sulfur oxide emissions on acid rain. Measurement of individual tailpipe emissions, in combination with dispersion modelling, may be inadequate to predict the aggregate effect of installing auto pollution control devices.

The main idea is that small-scale "micro" models and experiments may be inadequate to understand or predict the "macro" consequences of large-scale policy interventions (Harris, 1985). At best, basic research and data acquisittion can only disentangle individual mechanims. Such research cannot by itself show the interaction of multiple mechanisms of environmental damange and multiple routes of toxic exposure. The only way to assess such large-scale effects is by natural experiments, that is by regulatory intervention.

Chloroflourocarbons

In 1974, Molina and Rowland proposed that long-lived, stable chloroflourocarbons (CFCs) could slowly migrate to the stratosphere, where they would release chlorine upon contact with high levels of radiation. The resultant free chlorine could in turn act as a catalyst to break apart ozone molecules. Thus, CFCs might be steadily depleting the

stratospheric layer of ozone, the shield that stops the penetration of ultraviolet-B radiation to the earth's surface.

The ozone-depletion hypothesis was taken seriously by the scientific community, including a 1976 report by the National Academy of Sciences. In 1977, Congress amended the Clean Air Act (42 U.S.C. 7457(b)), authorizing EPA's Administrator to issue regulations for controlling substances or activities "which in his judgment may reasonably be anticipated to affect the stratosphere, especially ozone in the stratosphere, if such effect in the stratosphere may reasonably be anticipated to endanger public health or welfare. Such regulations shall take into account the feasiblity and the costs of achieving such control." The statutory language permitted EPA to act in the face of scientific uncertainty (U.S. Environmental Protection Agency, 1987b).

In 1978, EPA and the U.S. Food and Drug Administration moved to ban the use of CFCs as aerosol propellants in all but "essential applications." During the early 1970s, aerosol propellants constituted about 50 percent of the total CFC use in the United States. Thereafter, CFC use in propellants declined markedly.

Largely in response to a series of National Academy of Sciences studies in the late 1970s, EPA issued in 1980 an

Advance Notice of Proposed Rulemaking under the Clean Air Act. The Notice proposed to freeze the production of certain CFCs and suggested the possible use of marketable permits to allocate CFC production among various industries.

In the early 1980s, however, new data and models suggested that many other factors contributed to ozone depletion in the stratosphere. Carbon dioxide and methane, two atmospheric gases that have been increasing in concentration, appeared to buffer the ozone-depleting effects of CFCs. Moreover, while CFCs continued to be used as foam-blowing agents, refrigerants and solvents, the decline in CFC aerosol propellant use had resulted in a levelling off of world-wide CFC production.

Beginning in about 1983, demand for non-aerosol uses of CFCs accelerated. Total production expanded to the point where it now exceeds 1974 levels. CFC-11 (primarily used as a foam-blowing agent) and CFC-12 (primarily used as a refrigerant) are now rising at 5 percent annually, while CFC-113 (primarily used as solvent for electronics and metal cleaning) has risen an esimated 10 percent annually. Moreover, there have been increases in demand for certain brominated compounds that are also thought to deplete stratospheric ozone (such as Halon-1211 used in specialized firefighting applications). These changes have been parallelled by continued increases in carbon dioxide and methane.

In 1985, the World Meteorological Organization (WMO) conducted a review of all ground and satellite-based atmospheric ozone measurements to date. WMO concluded that ozone levels in the upper atmosphere had in fact decreased by 0.2-0.3 percent annually during the 1970s. Moreover, these decreases were offset by increases in ozone in the lower atmosphere, so that the total "column" ozone had remained unchanged.

In May 1985, however, Farman, Gardiner and Shanklin reported that ozone levels in Antartica during the months of September to November had declined by 40 percent since 1957, with most of the decline occurring since the mid-1970s. The discovery of this Antartic ozone hole was completely unexpected; a 40 percent decline was not predicted by current atmospheric models of ozone depletion. By 1987, additional measurements of a key compound-- chlorine monoxide-- suggested that anomalous chlorine chemistry may play a role in the development of the Antartic hole. Such findings left open the possibility that seasonal declines in ozone above Antartica were idiosyncratic and not reflective of global chemistry. Still, researchers have yet to determine the exact mechanisms for the finding of high levels of chlorine monoxide in the Antartic hole. They cannot ascertain whether such unknown mechanims are indeed unique to Antartica.

Moreover, recently published evidence (Kerr, 1987) has challenged the conclusion that total column ozone is stable. Ground-based and satellite measurements now suggest a 3-5 percent annual decline during the 1980s. As in the case of the Antartic ozone hole, these measurements fall outside of the uncertainty bounds computed from current atmospheric models, which predict that column ozone should not have decined by even one percent. A review of the newer data has now been instituted by the National Aeronautics and Space Administration and the National Oceanographic and Atmospheric Administration.

Why did the models fail to predict the 1987 results? One possibility is that the results are artefactual (e.g., misinterpreted satellite measurements). Another is that the models have failed adequately to consider the solar cycle or volcanic activity. Still, the main problem is that current models-- which now include approximately 50 chemical species and simulate over 140 different reactions-- may not be able accurately to replicate atmospheric chemistry. Have they failed to predict the limits by which the lower atmosphere can compensate for stratospheric ozone losses? Have they failed to predict the buffering effects of carbon dioxide and methane? Are estimates of the half-lives of certain CFCs inaccurate (75 years for CFC-11 and 110 years for CFC-12)?

On September 16, 1987, the United States and 23 other nations signed the Montreal Protocol on Substances that Deplete the Ozone Lay. The agreement set forth a timetable for reducing specified ozone-depleting chemicals, including a freeze on production at 1986 levels, followed by reductions during the 1990s. EPA, in anticipation of U.S. ratification of the Montreal Protocol, has already mandated the reporting of 1986 production, import and export by American firms (U.S. Environmental Protection Agency, 1987b).

Formal benefit-cost analysis of CFC regulation is a formidable task. We need to model the future decline in stratospheric ozone levels; the possible compensating increase in lower atmospheric ozone levels; the potential adverse effects of changes in atmospheric ozone, including increased incidence of skin cancers and cataracts, damage to acquatic organisms, accelerated weathering of outdoor plastics; and the overall effects of global warming. We need to ascertain the economic dislocation resulting from restrictions of CFCs and halons, including losses in refrigeration, foam production, cleaning of electrical equipment and firefighting applications.

But that is not enough. We need to know whether we can really wait for better data and models on atmospheric chemistry ozone depletion. What is more, we need to assess

the future evolution such scientific information. Here, we need to ask whether implementation of CFC and halon controls now may provide a critical source of data in understanding the ozone problem.

Regulation of CFCs and halones is hardly an all-or-none proposition. Should the Montreal Protocol go into force, and should the United States ratify the Protocol, the EPA will need to implement the freeze at 1986 levels and the planned reduction for the 1990s. The Agency currently proposes a system of marketable licenses. Production or use charges are also under consideration. It is unlikely that EPA can project the consequences of these proposed regulatory schemes. Accordingly, in choosing which scheme to adopt, the Agency needs to ask what near-term interventions are likely to provide information about future regulatory designs.

CONCLUDING COMMENTS

In environmental decision making, inconclusive scientific evidence is a commonplace occurrence. Still, regulatory agencies continue to make decisions in face of such uncertainty.

In evaluating such regulatory choices, it is hardly enough to assess the static benefits and costs of each

regulatory option. Instead, regulatory agencies need to solve the problem of timing. That means assessing the benefits and costs of intervening now versus later.

To attack the problem of timing, I have suggested that regulatory agencies ask two types of questions: Will we be able to take back the regulatory action? Will intervention be informative about future regulatory choices?

Environmental regulation takes many forms: requiring private firms to conduct studies or report data; suspending some uses of a chemical while permitting others; mandating or changing warning labels; issuing emergency suspensions; and scheduling phase outs.

In general, my analysis points toward a style of regulation in which agencies take small, incremental regulatory steps at the early stages of a problem. These small steps would be designed impose minimal sunken investments in compliance, yet provide essential information on the uncertain benefits and costs of intervention.

The supporting evidence, however, has been largely anecdotal. I have cited a few possibly unrepresentative examples. To assess the performance of our past environmental decisions, and to formulate guides for future environmental choices, we will need a much wider array of case studies.

Still, I see broad application of the idea that environmental decision makers often wait too long to take

action in the face of uncertainty. The reasons for delaying action, I suggest, are at best poorly articulated. Assertions that proof is not yet available, or that attention will be diverted from fundamental causes, or that the public will be needlessly alarmed should be subject to more careful scrutiny. The refrain that "we need more research before we can act" likewise needs to be questioned. It is unfair to state the problem as "regulation versus research" when the main issues are the synergies between regulation and research.

TABLE 1. SCIENTIFIC DEVELOPMENTS CONCERNING EDB, 1910-1976

Year	Scientific Developments
1910	Marmetschke reports on acute human toxicity of EDB after accidental administration.
1923	EDB first produced on commercial scale for sale to producers of lead antiknock compounds.
1925	Neifert reports efficacy of EDB as a fumigant.
1927	Thomas and Yant report EDB absorption in toxic amounts through the skin of exposed workers; acute toxicity reproduced in laboratory by inhalation and dermal exposure in guinea pigs.
1928	Kochmann reports on subacute toxicity in a worker repeatedly exposed to EDB; confirms acute toxicity in laboratory rabbits and cats exposed by inhalation.
1929	Glaser and Firsch confirm acute toxicity of EDB in guinea pigs.
1938	Pflesser reports on acute toxicity in workers exposed to EDB.
1946	Aman reports acute toxicity of EDB by oral administration in rats and guinea pigs.
1950	EDB begins to be used on widespread basis for quarantine treatments of imported fruits and vegetables, control of interstate movement of insect pests, fumigation of grain, spot fumigation of milling machinery, and soil fumigation.
1952	Adams et al. report acute toxicity in workers exposed to fumigant mixtures of EDB, ethylene dichloride and carbon tetrachloride. Rowe et al. report acute toxicity by oral instillation, dermal and eye contact and inhalation in rats, guinea pigs, rabbits, mice, chickens and monkeys.
1955	McCollester et al. report acute toxicity to albino rats of EDB and of fumigant mixtures containing EDB. Bondi et al. report decreased egg production and egg weight in hens fed grain fumigated with EDB.

TABLE 1. SCIENTIFIC DEVELOPMENTS CONCERNING EDB, 1910-1976

Year	Scientific Developments
1960	Olmstead reports case of acute toxicity after accidental oral ingestion of EDB capsules.
1965	Amir and Volcani publish initial report on spermicidal action of EDB in bulls given EDB-containing feed.
1968	Alumot reports reduction in egg size and egg fertility in hens given EDB-fumigated feed.
1970	Edwards et al. report rapid absorption and wide organ distribution of EDB in mice.
1971	Ames reports direct mutagenicity of EDB in Salmonella revertant assay.
1972	Buselmaier et al. confirm direct mutagenicity of EDB in Salmonella revertant assay.
1973	Olson et al. report preliminary results of National Cancer Institute (NCI) oral gavage study in Osborne-Mendel rats and B6C3F1 mice; squamous cell carcinomas of stomach observed in experimental animals near sites of application. Amir reports that spermicidal effect of EDB in bulls results from direct action on spermatogenesis; effects appear reversible.
1974	U.S. production of EDB reaches 330 million pounds, of which 200 million pounds were used in lead antiknock formulations. Brem et al. confirm experimental mutagenicity and DNA-modifying effects of EDB. Vogel and Chandler confirm mutagenicity in Drosophila.
1975	Powers et al. report additonal results of NCI oral gavage study in rats and mice. EPA study shows gasoline station exposures to EDB in the range of 0.01ppb, manufacturing site exposures in the range of 10 to 15ppb.
1976	Plotnick and Conner confirm wide organ distribution of EDB in guinea pigs after experimental administration.

TABLE 2. SCIENTIFIC AND REGULATORY DEVELOPMENTS CONCERNING EDB, 1977-1984

Year	Scientific Developments	Regulatory Developments
1977	Ott and Scharmweber report on 156 Dow Chemical employees in two EDB production facilities; no significant increase in mortality or cancer found. International Agency for Research on Cancer (IARC) classifies EDB as experimental mutagen and animal carcinogen. Hunt, in Great Lakes Chemical Corp. submission to Occupational Safety and Health Administration (OSHA), reports current worker exposure during EDB fumigant application to be in range 60-520ppb, depending upon adherence to label directions. In risk assessment based upon NCI oral gavage study, EPA's Carcinogen Assessment Group (CAG) predicts almost 100% lifetime incidence of cancer predicted from 40-year exposure to 400ppb.	National Institute on Occupational Safety and Health (NIOSH) reviews data on pharmacokinetics, metabolism, acute and chronic toxicity, reproductive effects and carcinogenicity of EDB; OSHA recommends tightening of standard for occupational exposure to 20ppm time weighted average (TWA) of EDB. Environmental Protection Agency (EPA) issues "Position Document 1" on EDB; initiates "Rebuttable Presumption Against Registration" of EDB under Federal Insecticide, Fungicide and Rodenticide Act (FIFRA).
1978	Rausch, in Dow Chemical submission to OSHA, reports on current and history occupational exposures to EDB; exposures were 1-24ppm in 1949 and 1952, less than 5ppm in 1971 and 1972. Ter Haar, in Ethyl Corporation submission to OSHA, issues preliminary report on mortality and reproductive function in	

TABLE 2. SCIENTIFIC AND REGULATORY DEVELOPMENTS CONCERNING EDB, 1977-1984

Year	Scientific Developments	Regulatory Developments
	workers exposed from 3 months to 10 years. At dosages ranging from less than 0.15ppm to 4.5ppm, no elevation in death rates was detected. Sperm counts compared favorably to general population. Trend of sperm counts in relation to EDB exposure, but of questionable significance. National Cancer Institute (NCI) publishes results of oral gavage study in rats and mice.	
1979	Van Duuren et al. report on skin painting study in Ha:ICR Swiss Webster mice; EDB found to be carcinogenic. Wong et al. report retrospective evaluation of reproductive performance of workers exposed to EDB; no effects seen in three of four plants. Plotnick et al. publish preliminary results of NIOSH inhalation study in Sprague-Dawley rats. Ramsey et al. report that CAG's 1977 risk assessment would predict 54-85 cancer cases among 156 exposed Dow Chemical employees in the Ott-Scharmweber study, whereas 8 cases were observed.	EPA cancels registration of soil fumigant dibromochloropropane (DBCP), probably resulting in increased use of EDB.

TABLE 2. SCIENTIFIC AND REGULATORY DEVELOPMENTS CONCERNING EDB, 1977-1984

Year	Scientific Developments	Regulatory Developments
1980	Ott et al. publish follow-up report on Dow Chemical cohort; inconclusive findings due to small cohort size, incomplete exposure data, incomplete follow-up, and confounding with other chemical exposures (arsenicals). Terr Haar publishes follow-up report on cohort of 53 employees exposed to EDB; cohort too small to assess cancer risk. NCI inhalation study on Fisher-344 rats and B6C3F1 mice submitted for internal peer review; EDB found to be carcinogenic. Wong et al. publish intermediate results of NIOSH inhalation study in rats; EDB found to be carcinogenic. American Conference of Governmental Industrial Hygienists classifies EDB as a suspect human carcinogen. EPA internal review estimates probable residue level for EDB in wheat bread derived from grain fumigated after harvest with EDB to be 0.07ppb; "realistic worst case" residue estimated to be 31ppb. EPA's CAG issues cancer risk assessment, based upon one-hit mathematical model; estimated lifetime cancer risk of dietary burden of EDB estimated at 3.3 per 10,000. EPA commissions	EPA issues "Position Document 2/3" (Notice of Preliminary Determination Concluding the Rebuttable Presumption Against Registration of EDB); proposes to continue registration of EDB for preplant soil fumigation, but to cancel EDB registrations for fumigations of stored grains and spot fumigation of grain milling machinery, and to cancel post-harvest fumigation of fruits and vegetables by July 1, 1983. EPA requires soil fumigant registrants to conduct ground water contamination studies. U.S. Supreme Court requires that a "significant risk" be adduced to justify OSHA regulations (Industrial Union Department v. American Petroleum Institute, et al.).

TABLE 2. SCIENTIFIC AND REGULATORY DEVELOPMENTS CONCERNING EDB, 1977-1984

Year	Scientific Developments	Regulatory Developments
	ground water contamination study by California Department of Food and Agriculture (CDFA).	
1981	Publication of final results of NIOSH inhalation study in rats; EDB found to be carcinogenic. EDB is used in California to fight the Mediterranean fruit fly. Maddy et al. (CDFA) estimate EDB residues up to 57ppb in edible portions of fumigated citrus fruits. Raines and Holder find average EDB residue of 35.7ppb in biscuits, contrary to early EPA estimates of 0.07ppb; reported levels in flour range from non-detectable to 4.2ppm.	State of California (Cal/OSHA) issues temporary emergency standard of 130ppb. OSHA issues Advance Notice of Proposed Rulemaking regarding occupational exposure to EDB, proposing reduction of standard from 20ppm to 15ppb and requesting comments on quantitative risk assessment (Federal Register, December 18).
1982	Publication (March) of final results of NCI inhalation study in rats and mice; EDB found to be carcinogenic. EPA scientists notified (June) that three wells in Seminole County, Georgia were contaminated with EDB levels as high as 100ppb. SRI International publishes NIOSH-commissioned risk assessment based upon NCI and NIOSH inhalation studies in rats and mice (June); chronic exposure	OSHA interprets Supreme Court ruling as permitting mathematical risk assessment in support of agency regulations (Federal Register, April 9). Cal/OSHA's emergency standard of 15ppb rejected by California Office of Administrative Law; California adopts as a permanent regulation a standard of 130ppb.

TABLE 2. SCIENTIFIC AND REGULATORY DEVELOPMENTS CONCERNING EDB, 1977-1984

Year	Scientific Developments	Regulatory Developments
	to 130ppb predicted to yield 4 to 26% lifetime human cancer risk. CDFA (June 2) revises estimates of EDB residues in fumigated citrus fruits up to 210 to 880 ppb. Wade and Sakura report two acute lethal reactions among workers exposed to EDB.	
1983	National Toxicology Program reports that inhalation of EDB (10 to 40ppm) in Fisher 344 rats produced testicular degeneration. EPA-commissioned study of ground water contamination by CDFA issues preliminary report (Spring), finding EDB at concentrations between 0.1 and 31 ppb in the soil at depths greater than 20 feet, moving down to ground water. Follow-up report (June) reveals ground water levels between 0.02 and 5ppb in 16 counties in four states. New EPA risk analysis issued as part of Position Document 4. Original one-hit model of Position Document 2/3 is modified to include "Weibull timing." Estimated average EDB content of grains is revised upward markedly to 31ppb. CAG's new estimate of lifetime cancer risk from dietary burden of EDB is 3.3 per 1,000, based	EPA issues "Position Document 4" (September 27), with revisions in mathematical risk assessment methodology. EPA issues emergency suspension of soil fumigation with EDB; gives notice (September 28) of intent to cancel registration of EDB as a grain and fruit fumigant under "unreasonable hazard" standard of FIFRA. EDB use in fumigation to be eliminated by 1986. State of Florida issues emergency regulations restricting EDB in uncooked grain products to 1ppb (level of detection).

TABLE 2. SCIENTIFIC AND REGULATORY DEVELOPMENTS CONCERNING EDB, 1977-1984

Year	Scientific Developments	Regulatory Developments
	upon lifetime consumption of current levels of EDB in grain products.	
1984	Grocery Manufacturers of America (GMA), modifying the Rains and Holder (1981) detection methodology, find 79% of ready-to-eat grain-derived products contain EDB levels below 1ppb; also report on disappearance of EDB through cooking raw grain products. Environ Corporation, under sponsorship of GMA, issues (January 20) risk assessment of exposures to EDB residues in consumable grain products, based upon NCI oral gavage assay and assumptions of no further grain fumigation and of depletion of EDB in grain stores by 1986; upper limit of lifetime cancer estimated to be 1 in 4 million. Temple, Barker & Sloane and Economic Perspectives, Inc. issue economic analysis of impacts of immediate removal of EDB from the food supply; if 50-60% of stored grains and 67% of grain products were immediately restricted from any use, they conclude, grain prices would nearly double, with consumer expenditure increases of $35 billion and grocery manufacturers losses of $2.8 billion in inventories.	EPA announces (February 3) immediate suspension of further use of EDB in production of grain products, recommends guidelines to states for acceptable levels of EDB in foods, including 900ppb in raw grain products, 150ppb in processed products requiring further cooking, 30ppb in ready-to-eat foods. Massachusetts Department of Public Health recommends (February 6) emergency regulation at 10ppb for all food products, with transition in 30 days to 1ppb. ("The Department's position is that the only safe level of exposure to a carcinogen is one that is zero or near zero. The Department therefore believes that it is appropriate to move rapidly to levels of EDB in food of less than 1ppb.")

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