● *ABSTRACT*

The question is posed: why were two pesticides, Aldrin and Dieldrin, judged to be carcinogenic in the US but not in Britain when the same evidence was available to the public authorities in both countries? No single cause is identified; rather, a variety of mutually reinforcing factors account for the decisions by the two public authorities: the uncertainty of the scientific evidence; the application of different standards of carcinogenicity associated with different social and scientific commitments; the government agencies with primary responsibility for the decisions; the way in which pesticides are regulated; and several cultural and economic considerations. The case study illustrates the analytical inadequacy of the fact-value distinction, and the tendency of decision-makers to justify their decisions by recourse to science. It also supports the view that the traditional relationship between science and public policy is being redefined in complex, technical areas of decision-making like risk assessment.

Carcinogenic Risk Assessment in the United States and Great Britain: The Case of Aldrin/Dieldrin

Brendan Gillespie, Dave Eva and Ron Johnston

The nature and scale of the hazards associated with new technologies have required governments to make difficult decisions about the form of control they should exercise over new products and processes. Although governments have received advice from a large and growing body of scientists, this has not always simplified the decision-making process nor rendered it more rational and objective. In this paper, we try to explain why two governments reached contradictory conclusions to the same problem on the basis of the same scientific evidence. More specifically, we will try to explain why two chemical pesticides, Aldrin and Dieldrin (A/D), were judged to be carcinogenic in the US but not in Britain when the same data were available to public authorities in both countries.

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The issue here is not one of differing assessments of risk or of balancing risks and benefits. Rather, what requires explanation is the different regulatory statuses assigned to the same products by the US and British governments.

The observation that scientific experts disagree and oppose each others' views is hardly new — witness the appearance of a sizeable literature on the subject.[1] However, much of the literature incorporates weaknesses which inhibit our understanding of the role played by science in risk determination and, more generally, in controversies among experts in matters of public policy. Firstly, much of it focuses on one country, usually the US, whilst assuming that the roles of scientific advisers, and the problems which their use entails for governments, are everywhere the same. The comparative approach adopted in this paper should offset this, as well as a second, deficiency:[2] namely, the roles of science and scientists are generally analyzed in terms of universalistic categories which obscure the effects of different political and cultural settings on the type of science providing the basis for policy decisions. One frequently used variant holds that science provides the facts, and their evaluation from divergent value and ideological perspectives results in contrary interpretations. Conflicts among scientists are then explained in terms of the political views, bias or irrationality of one or more of the disputants, and little attention is paid to the content of the conflicting positions.

This approach has merit to the extent that it accepts that scientists become integrally involved in political conflicts. Nevertheless, two weaknesses severely limit its explanatory power, and call into question the fact-value distinction on which it is based. Firstly, it does not provide an accurate description of actual controversies. Nowotny, for instance, has argued that the traditional division of labour between science and society is, in practice, breaking down in complex technical spheres of decision-making like risk assessment.[3] Secondly, the essentially positivist view of science, on which the distinction is based, is overly restrictive in limiting explanations of scientific controversies to factors 'extrinsic' to science: values, bias, and the like. When science is examined as a form of organized, intellectual production, a much more complex relationship between scientific concepts, theories and methodologies, on the one hand, and ideological and value commitments, on the other, emerges, which also allows explanations of controversies in terms of factors 'intrinsic' to scientific development.[4]

Through a cross-national analysis of the contradictory A/D decision, we hope to set the pertinent features of the science-public policy relationship into sharper focus than before. We begin our analysis by looking at the differing interpretation of the evidence about A/D provided for the British and US authorities. Significant differences are found to exist in the criteria used to infer carcinogenicity, in the type of scientists providing the authoritative interpretations, and in the choices with which the respective public authorities were faced. This examination of the provision of scientific advice is complemented by an analysis of the factors affecting its reception. We identify those institutional, legal, cultural and economic features of the decision-making contexts which made them more or less receptive to the advice they were proffered.

BRITISH AND US DECISIONS[5]

Aldrin and Dieldrin are two organochlorine pesticides that were widely used in agriculture in the 1960s. Workers applying A/D and consumers eating residues in and on treated crops were exposed to short- and long-term toxic hazards. (Aldrin rapidly degrades to Dieldrin in plants and animals, so when we discuss the hazards of A/D we are really talking about the hazards associated with Dieldrin.)

The carcinogenic risk of A/D had been reviewed in a variety of national and international settings since the mid 1950s.[6] While several expressed uncertainty and requested more evidence, none of the reviews had been prepared to declare A/D carcinogenic. None, that is, until September 1974, when the US Environmental Protection Agency (EPA) found that A/D posed an unacceptable carcinogenic hazard.[7] Not surprisingly, the decision was viewed as controversial in the US, and was criticized in other countries.

EPA had initiated administrative hearings in August 1973 to determine whether A/D's registrations should be *cancelled* — that is, effectively banned.[8] These hearings had been in progress for a year when EPA held more hearings to determine whether A/D's registrations should be *suspended* — that is, their sale temporarily banned. Congress had provided EPA with these statutory alternatives to ensure that the agency could eliminate 'unreasonable' risks that might arise in the course of lengthy cancellation hearings. The two decisions are administratively distinct, and a decision to

suspend does not automatically prejudge the cancellation decision.

It was during the suspension hearing that the question of A/D's carcinogenicity came to the fore and, in fact, dominated the proceedings. An environmental group, the Environmental Defense Fund (EDF), had petitioned EPA for A/D's cancellation and suspension. Their case was supported, and effectively prosecuted, by EPA's Office of General Counsel (OGC). Shell, A/D's manufacturer, opposed these groups and was supported by the Department of Agriculture (USDA). The case was argued before Administrative Law Judge Perlman who was paid by EPA but independent of the agency for career advancement and tenure.[9] He recommended that A/D be suspended because of the carcinogenic risk the chemicals posed, and this was endorsed by EPA's Administrator, Russell Train. When the decision was upheld in the Appeals Court, Shell withdrew from the cancellation hearings.[10]

The following month, in October 1974, a group of British experts reviewed EPA's decision. They concluded 'that there was no reason to recommend any change in the UK action on Aldrin and Dieldrin as decided at the time of the 1969 reviews of organochlorine pesticides'.[11] No new evidence had been produced in the interim and the British experts had frequently discussed the evidence on A/D with Shell toxicologists over the years.[12]

The British experts were members of a Panel of the Advisory Committee on Pesticides and Other Toxic Chemicals. The Committee is an expert body that advises UK government departments on the hazards associated with pesticides. The departments then negotiate what action to take with manufacturers of the product in question. The negotiations are conducted within the framework of a voluntary (that is, non-statutory) agreement between government and industry — the Pesticides Safety Precautions Scheme (PSPS).[13]

It is quite clear, then, that the experts and decision-makers in PSPS and EPA reviewed the same experimental evidence of A/D's possible carcinogenicity and came to contradictory conclusions. Furthermore, although each group of experts and decision-makers was aware of A/D's status in the other decision-making forum, this did not change their conclusion. We therefore have a genuine paradox to explain.

LIMITATIONS OF TOXICOLOGY

The first possible explanation of the different decisions is the uncertainty in determining carcinogenic risk. Toxicology, the field most directly concerned with the harmful effects of chemicals, has developed a fragmentary and incomplete understanding of the cancer-causing process.[14] It is conceivable, then, that a number of competing and equally plausible interpretations of the same evidence could co-exist. The toxicologists' uncertain knowledge of cancer might make it difficult to choose between rival interpretations, or to completely rule one out.

Although this argument could be applied to all chemicals tested for carcinogenicity, it seems particularly relevant in this case. Firstly, other chemicals have been shown to be more definitely carcinogenic than A/D.[15] Secondly, even those scientists who thought A/D to be carcinogenic regarded them as considerably less potent than other known carcinogens.[16] Thirdly, Shell had spent $10 million producing evidence of the hazards associated with A/D, so there was a much better data base for these than for most other chemicals.[17]

Briefly, the evidence was of three types:

1) *Epidemiology* — that is, correlations between incidence of human cancer and exposure to particular substances. Although ingestion of A/D-contaminated foodstuffs had resulted in British and US consumers storing Dieldrin in their body fat, there were no control populations which could be used to evaluate the effects of these exposures. The best available epidemiological data derived from the medical records of workers employed in the production of A/D.[18] About 1,000 workers had been occupationally exposed, but only 69 of this pre-selected group had been exposed for 10 years or more. Since the latency period for cancer can be 20, 30 or even 40 years, it was difficult to know what significance to attribute to the two cases of cancer that had occurred in this group by 1974. Finally, Shell argued that indirect epidemiological evidence was available. Their scientists believed that humans metabolized A/D analogously to the drug phenobarbitone. They argued, therefore, that epidemiological data on phenobarbitone were relevant to the determination of A/D's carcinogenic hazard.

2) *Animal models*. These were experiments in which specific doses of A/D were administered to populations of laboratory animals, and the incidence of cancer tumours in exposed populations were compared with those in controls. The significance of

results from these experiments for humans is highly controversial. By 1969, experiments with A/D in mice, rats, dogs and monkeys had been performed.

3) *Biochemical tests.* In recent years, a variety of so-called 'quick tests' have been developed to predict carcinogenic hazards more cheaply and quickly than animal tests.[19] Shell scientists developed several *in vitro* tests which they used on A/D. Essentially, they tested for certain biochemical properties of chemicals that Shell claimed were correlated with carcinogenic activity.

Clearly, there are general problems in determining carcinogenicity, and these were exacerbated by the particular biological properties of A/D. The uncertainty gave rise to the possibility of different interpretations and hence different decisions. Even so, uncertainty cannot explain why the British and US decisions went one way rather than the other; that is, it cannot explain why A/D were judged to be carcinogenic in the US but not in Britain, and vice versa. Since the uncertainty of the evidence was common to both EPA and PSPS, it cannot be a sufficient explanation of their difference.

EVALUATION OF THE EVIDENCE

The different interpretations of the A/D evidence could have arisen in at least two ways, depending on the degree of consensus that existed over the standards for inferring carcinogenic risk. If there were consensus over the standards, the explanation of the differing interpretations would be different than if there were competing standards of carcinogenicity. Thus, the way in which the two decisions were reached is important to our overall argument; clarification of just how the decisions differed must logically precede any account of why they differed.

We will outline the arguments that Shell and EPA presented to Judge Perlman in the first part of this section, and the evaluation of the A/D data by British experts in the second.

(A) *US Evaluation*

At the suspension hearing, the Shell lawyers argued that a scientific approach requires *all* the evidence to be examined and that the results should be reproducible.[20] In a similar vein, the Director of Shell's Tunstall Laboratories argued that carcinogenicity could be inferred only when five criteria were met:

1. The exposed animals experience a higher incidence of tumors.
2. Tumors develop in more than one species.
3. The development of these tumors can be proven to be compound-related.
4. The animals have proven to be an adequate model for extrapolating to man.
5. Human data is available proving at least one incidence of cancer.[21]

Shell conceded the first criterion and accepted that five experiments — Davis and Fitzhugh (1962), Davis (unpublished), McDonald et al. (unpublished), Walker et al. (1973) and Thorpe and Walker (1973) — demonstrated that Dieldrin had increased the incidence of liver tumours in mice.[22] Shell then argued that this finding could not be extrapolated to humans because A/D did not meet the other four criteria.

Evidence was produced by several Shell witnesses that the mouse liver was not a valid predictor of human cancer (Criterion 4). They argued that Dieldrin had not induced tumours at sites other than the liver, and that the induction of hepatomas (liver tumours) in mice was highly dependent on a variety of genetic and environmental factors. More critically, Shell witnesses argued that the response of the mouse liver was unique and quite different from other species. Thus, they argued that evidence from rat, monkey and dog studies failed to indicate a carcinogenic response (Criterion 2). Next, they advanced a five stage process whereby liver tumours were formed, and argued that whilst all five stages occurred in mice, they did not occur in humans.

This latter argument was quite subtle, and drew on evidence from a variety of sources. The indirect epidemiological evidence, the results from the *in vitro* biochemical experiments and the medical histories of occupationally-exposed workers, were all marshalled to support an argument for a mechanism which, Shell argued, showed how A/D did *not* induce human cancer (Criterion 3). Finally, Shell argued that the direct epidemiological evidence indicated that A/D did not meet their fifth criterion: the demonstration of one A/D-induced human cancer.

The EPA case against A/D was quite different in form. Their lawyers argued that a number of principles had been well-established in the scientific community for assessing carcinogenic hazards, and that on this basis A/D must be considered carcinogens.[23] Nine cancer principles were presented in the A/D case. The most relevant ones 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...
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.[24]

We may demonstrate that EPA was using different standards than Shell by considering the agency's response to Shell's five criteria. EPA's witnesses had re-examined the pathological and statistical analysis of the animal data, and it was this which helped elicit Shell's concession of their first criterion. With this established, EPA strongly objected to Shell's 'two species' criterion (Criterion 2). They argued that the negative evidence from the rat, dog, and monkey studies was the result of poorly designed experiments. Further, the uncertainties surrounding the use of animal models and the dictates of 'prudent policy,' meant that, for them, positive evidence should supercede negative results.[25] In this case, however, evidence on the mouse derived from five different strains, and was therefore sufficient, in their opinion, to indict A/D as carcinogens.

Although EPA could hardly disagree with Shell that the induction of tumours in test animals be 'compound related' (Criterion 3), there was scope for disagreement on the meaning of this term. EPA witnesses insisted that they were not required to produce causal mechanisms, as Shell had demanded. Indeed, Epstein argued that such a requirement 'would define away the entire field of chemical carcinogenesis.'[26] EPA's ninth cancer principle declared that a positive result from a 'valid carcinogenesis test' was sufficient to consider that chemical carcinogenic. Since A/D fulfilled this condition, EPA concluded that they were carcinogens.

There were also objections to the mechanism Shell had presented. Farber argued that

> it is evident that many chemicals require metabolic conversion to active derivatives before they can initiate the development of cancer. However, the specifics of the metabolic processes which result in cancer in various test animals

> are not clear, to say nothing of the metabolic processes in man. No one as yet can draw any valid correlation between a particular pattern of metabolism and the induction of cancer in any species, and any judgments concerning carcinogenicity or lack thereof based on metabolic patterns have no scientific basis at this time.[27]

Similarly, the EPA witnesses rejected the analogy on which Shell's indirect epidemiological evidence was based and the validity of the biochemical tests as indicators of carcinogenicity.

If EPA were to demonstrate A/D's carcinogenicity, the agency had to accept Shell's fourth criterion, and justify the mouse as a valid indicator of human cancer. Shell's attack on the mouse had important regulatory implications.

> Health agencies in many countries recommend mice for routine testing of pesticides, food additives and drugs. If the argument that 'mice are of no value for carcinogenicity testing' were acceptable, the existing regulatory system would have been pitched into chaos.[28]

By the same token, the regulatory status of other pesticides would have been threatened if A/D were suspended. Aware of these implications, the EPA witnesses strongly objected to doubts about the reliability of the mouse liver, and argued that well-designed experiments controlled any genetic or environmental susceptibilities. They pointed out that the mouse was a standard test animal in national and international regulatory systems, and that it was used in Shell's own studies — Walker et al. (1973) and Thorpe and Walker (1973). Finally, evidence was produced suggesting that chemicals 'adequately' tested in mice had been shown to be carcinogenic when 'adequately' tested in other species.

EPA also objected to Shell's fifth criterion, requiring the demonstration of at least one A/D-induced human cancer. They argued that since animal tests were sufficient to predict carcinogenic risk, it was ethically unjustifiable to wait for the demonstration of human harm. Moreover, since the direct epidemiological data for A/D failed to meet methodological standards, it was not 'prudent' to reach a negative conclusion, which might subsequently be reversed by the accumulation of more evidence.

On all of the critical points, EPA convinced both Perlman and Train that theirs was the superior argument. Perlman had few doubts about his decision and justified his finding that A/D were

unacceptable carcinogenic hazards as follows:

> We believe that this conclusion represents established traditional and 'conventional wisdom'. The Shell Chemical Company has strenuously and with sophistication attempted to demonstrate that 'this truth' does not apply to Aldrin and Dieldrin for the reasons we have detailed above. We do not believe that traditional wisdom or science has been overcome thereby. Shell's presentation with respect to the shortcomings of the mouse as an appropriate test animal and its lack of significance for man is based, in part, on matters far from established in the scientific community, speculation and surmise. In reality, our knowledge with respect to cancer is very limited. Many, many years would be required to pursue the theories, hypotheses and correlations advanced by witnesses for Shell without any confidence that they could be proven.[29]

(B) *British Evaluation*

For reasons that will become clearer below, there is no public record of the deliberations preceding the British decision. Nevertheless, we may infer the standards employed by British advisers from several sources, and we will do so in chronological order. The first source of evidence derives from several Editorials in the principal British medical journals: *The Lancet* and the *British Medical Journal*.[30] It seems unlikely that they would have been written without at least consulting the PSPS advisers, and their 'anti-EPA tone' would tend to confirm this. The Editorials were dubious about a lay person/judge's ability to evaluate such complex issues and of the validity of using 'cancer principles' to make the decision. In dealing with the evidence, they showed a greater willingness to accept the available epidemiological evidence than the EPA witnesses. Moreover, they shared Shell's scepticism that human carcinogenic risk could be inferred from mouse data.

These themes are echoed in the views of several British committees that met in the 1960s. In 1969, PSPS's Advisory Committee reviewed the evidence and placed great stress on the negative epidemiological evidence derived from observations on Shell's workers.[31] In contrast, the Committee considered that evidence from animal experiments could only provide 'presumptive evidence.' They reported that:

> In consultation with a number of cancer experts and pathologists we were unable to obtain common agreement as to whether these lesions represented malignant tumours which would have indicated that Dieldrin had had an undoubted carcinogenic effect on these mice. Certain additional experiments were then suggested which might help yield results which would help to decide whether these

> liver lesions were capable of autonomous growth and had the accepted characteristics of malignant tumours.[32]

In 1967, a different Committee reviewed the evidence, but merely requested more data to clarify the issue.[33] PSPS's Advisory Committee had also reviewed the available evidence in 1964, and concluded in much the same way as the 1969 Committee.[34] They could obtain no consensus on whether the tumours were malignant or benign, and they hinted that their induction might be unique to the mouse.

The only other source that illuminates the British position is a report issed by the Panel on Carcinogenic Hazards in 1960.[35] The report outlines several principles to guide the interpretation of animal tests, and is significant because it is still recommended in the PSPS agreement.[36]

The Panel defined a carcinogen as a substance that increased the incidence of *malignant* tumours in animals or humans. The Panel was also prepared to differentiate carcinogens from co-carcinogens and initiators that were not carcinogenic in themselves, but which enhanced or augmented the effect of a carcinogen.[37] On the assumption that a carcinogenic response was specific to some test animals, the Panel recommended that two species be tested. The report does not make clear whether positive findings are required in both species, but it does suggest that: 'Negative results will reinforce confidence in the safety of the material; the significance of positive findings needs careful consideration in each case.'[38]

Putting these pieces of evidence together, we may conclude that British cancer experts were reluctant to label Dieldrin carcinogenic because of:

(1) the negative epidemiological evidence available;
(2) the lack of consensus as to whether Dieldrin had induced *malignant* tumours;
(3) concern about the mouse liver as a valid indicator of carcinogenicity; and
(4) the failure of Dieldrin to induce tumours in species other than the mouse.

Our review of the interpretation of the A/D evidence indicates that EPA and Shell witnesses employed different standards for the determination of carcinogenic risk. Similarly, EPA and PSPS experts required the evidence to satisfy quite different conditions

before labelling A/D 'carcinogenic', and this was the most immediate reason for the different British and US decisions. Moreover, insofar as we can compare the British evaluation with that of Shell, there are some striking similarities. Indeed, the only noticeable difference is that the British authorities required evidence of malignant tumours in test animals, whereas Shell's argument made no distinction between malignant and benign tumours. It seems reasonable to take as a working hypothesis, then, that the British authorities used the same, or very similar, standards as Shell to evaluate the A/D evidence.

ADVISERS AND THEIR ADVICE

Since different standards were applied to the same evidence, we must turn our attention to the scientists who provided EPA and PSPS with the authoritative interpretation of the evidence, and the nature of their advice. How can we account for the existence of such disparate standards, and what features of those standards, and their adherents, made them more or less compelling to decision-makers in PSPS and EPA?

In an earlier paper, Johnston and Robbins developed a model relating the type of occupational control exercised over scientists to the type of knowledge they produce, the specialties they work within, and the relations between those specialties.[39] These authors had previously analyzed one environmental controversy, and found that the disputants not only evaluated the same data differently, and derived contrary policy implications from the same, or similar, evidence, but that also, motivated by different social and scientific commitments, they were prediposed to produce different 'facts'. We may consider whether similar forces are operating in this case, and if they had any bearing on the British and US decisions, by examining the work of J. M. Barnes and Umberto Saffiotti, the principal advisers (respectively) to PSPS and the EPA lawyers. Both scientists occupied important organizational positions — Barnes as head of the British Medical Research Council's Toxicology Unit, and Saffiotti as head of the National Cancer Institute (NCI)'s Chemical Carcinogenesis Program. Their organizational roles meant not only that they could determine the type of work sup-

ported by these key organizations, but also that they could provide authoritative advice to those seeking it. Thus, Barnes was a member of every British Committee that has reviewed the toxic hazards of pesticides since the 1950s.[40] Similarly, Saffiotti had served on many US governmental committees, and was centrally involved in the development of EPA's cancer principles. Perlman described him as

> ...a world renowned expert whose initial testimony was cleared and approved by [the NCI] and whose demeanour and knowledge during his several days of cross-examination especially impressed us.[41]

Whereas the British Toxicology Unit has maintained close links with the chemical industry,[42] the NCI has been the leading agency in the US crusade against cancer.[43] Not surprisingly, then, the social commitments expressed by these institutional leaders in their approach to carcinogenic hazards are quite different:

> *Saffiotti:* That we take a position of caution and prudence in the matter of exposing the entire population to the potential hazards of chemical carcinogens is dictated by the tragic knowledge that — with the present trends in cancer mortality in the United States — out of 200 million Americans now living, 50 million will develop cancer and 34 million will die of it. Yet most cancers appear to be caused by environmental factors and therefore could be preventable.[44]

> *Barnes:* The safety of man from hazards presented by pesticide residues will not necessarily be increased by crying 'wolf' on every conceivable occasion that some direct or indirect carcinogenic activity can be detected in a substance filling a valuable role as a pesticide. Without pesticides many people would die for other reasons long before they reached the age at which they might develop cancer. Cancer was widespread long before modern pesticides were synthesized. If chemical carcinogens are responsible for any significant fraction of human cancer of unknown origin it is probable that such carcinogens will be of natural origin. With aflatoxin, cyasin, and the pyrroloziding alkaloids before us as examples of carcinogens found widespread in nature, it would be unwise if not irrational, to try to create undue alarm about carcinogenic hazards from pesticides that display no carcinogenic activity even faintly comparable with that of the compounds listed above.[45]

These quite different assessments of the significance of industrial chemical carcinogenic hazards support, and are supported by, competing scientific accounts of the carcinogenic process:

Saffiotti: ...extremely small amounts of chemical energy are required for the critical chemical interaction, which can trigger off a permanent change in the regulation of cell growth which in turn, as cells replicate, will become manifested as cancer. Such trigger effect of carcinogens is basically and completely different from most other toxic effects of chemicals, which require the continued presence of the agent to produce the effect, or a large number of target cells to be affected, to reveal a physiologic or pharmacologic effect, as is the case with hormones or vitamins. This is a fundamental point — well borne out by all modern molecular biology — which the 'traditional' toxicologist, used to the study of 'compound-dependent effects' rather than 'trigger effects,' is at times found to misunderstand. Because of this trigger effect, carcinogenesis is not like most other forms of toxicity and is not explained by the same generalizations.[46]

Barnes: There is already enough evidence to support the belief that for many carcinogens the latent period before the cancer develops bears some inverse relation to the size of the dose. If there are data on the response to high doses it may not be difficult to calculate that at some lower dose the latent period of response will exceed the life-span of the host. Thus, if the dose of a carcinogen is low enough, the response in an exposed person will be manifest only in the hereafter. What is needed to examine this belief is not more molecular biology but more studies on the response of the whole animal. ...A critical look at the limited amount of information that exists on the whole-animal response to carcinogens should help to disperse the prevailing gloom that there is neither a practical safe dose of a chemical carcinogen nor a reasonable experimental basis for attempting to derive one.[47]

Thus we can see how commitment to a 'trigger' mechanism of carcinogenesis, whereby a single molecule can initiate a cancerous response, legitimates the 'prudent' policy advocated by Saffiotti — whilst Barnes' commitment to the traditional toxicological mechanism, which requires prolonged contact between cells and chemicals, supports a more 'permissive' policy. This analysis suggests that a more detailed examination of the social and scientific commitments of the PSPS toxicologists and the EPA witnesses would reveal similar, systematic differences on issues such as the pathological classification of tumours, the evaluation of epidemiological evidence and the significance of mouse data.

It is beyond the scope of this paper to document these claims further, but we think the evidence presented is sufficient to conclude that the advice and the advisers that EPA and PSPS accepted were very different in the two cases. Moreover, the social and scientific commitments embedded in the advice were consistent with the two decisions: the more agriculturally-oriented advice with the British decisions, and the more health-protective advice with the US decision. But having clarified those features of the advice that seem

most relevant to the decisions, we must examine those features of the two decision-making contexts that made them more or less receptive to the advice they received. This would not be necessary if the British and US decision-makers had received only one set of arguments. However, the decision-makers in EPA had to choose between two rival arguments presented in a court-like setting, whilst British decision-makers asked a committee of experts to review their previous decision in the light of the opposed US decision. We must therefore determine what led the British and US decision-makers to favour differing interpretations of the same evidence.

Before doing so, we should mention one other way in which the two types of advice differed, and which may have affected their reception. As we indicated above, the type of occupational control exercised over scientists has implications not just for the content but also for the form of the knowledge produced. On Johnston and Robbins' model, the collegiate form of occupational control operating in basic research tends to result in universalistic and theoretically-oriented forms of knowledge that quickly diffuse into other scientific fields. In contrast, a greater degree of patron control, as occurs in more mission-oriented research establishments, is related to particularistic, atheoretical concepts and techniques, which are only locally intelligible. Whereas molecular biology, the approach underlying Saffiotti's 'trigger mechanism', has become the conceptual foundation for a significant part of contemporary biological sciences,[48] 'traditional toxicology', the approach underlying Barnes' 'continuous contact mechanism', is hardly represented in university scientific research. This evidence, therefore, tends to support the prediction of Johnston and Robbins' model, and suggests that there were differences not only in the social values of EPA advisers and the content of their advice, but also in the character of the science forming the basis of their advice.

DECISION-MAKING INSTITUTIONS

In general, the location of regulatory responsibility in different types of agencies will have direct implications for the evaluation of the benefits of pesticides and the hazards associated with their use. The considerable discretion inevitably vested in government agen-

cies allows the bureaucracy to define its mandate in close relation to its own interests and goals. We need to evaluate, therefore, how the institutional missions of the British and US governmental agencies with primary responsibility for the regulation of pesticides influenced the reception of the rival interpretations of the A/D evidence.

In Britain, several government departments are party to the PSPS Agreement, but there is little doubt that the Ministry of Agriculture, Fisheries and Food (MAFF) plays the leading role. The Minister of Agriculture, Fisheries and Food answers Parliamentary questions about pesticides, and MAFF provides most of PSPS's supporting staff. Eighty percent of the British usage of pesticides is agricultural, so MAFF is the agency with the most direct interest in pesticides, and particularly in their contribution to agricultural production. Indeed, there is good evidence that this interest tends to dominate MAFF's regulation of pesticides.[49]

It was just such a conflict of interest that led President Nixon to transfer responsibility for the regulation of pesticides from USDA to EPA in 1970.[50] Critics of USDA had argued that pesticides would be more effectively regulated by an agency with sole responsibility for environmental protection, and with no role in the promotion of agricultural production. When the agency was established, many of the activists who had supported the move were attracted by EPA's mandate, and found work in quite senior positions. The vigorous manner in which EPA has subsequently endeavoured to protect the US environment has led critics to accuse the agency of 'capture' by environmentalists.[51]

There are good grounds for believing, then, that MAFF's and EPA's institutional missions have led these agencies to regard pesticides in quite different ways, with MAFF emphasizing their contribution to argicultural efficiency and EPA predominantly concerned with the hazards that their use entails. Moreover, we can see how these missions would have made them receptive to different types of scientific advice: EPA to the more health-protective type and MAFF to the more agriculturally-oriented advice. Still, the organization of the two decision-making processes suggests that any connections of this sort are less direct than, at first, they would appear. Both processes are formally designed to attenuate the political pressure that government agencies can apply to the providers of technical advice: in the US, by insulated, quasi-legal pro-

ceedings; and, in Britain, by the operation of an expert-based committee. What role, if any, can we therefore attribute to EPA's and MAFF's institutional responsibilities in explaining the British and US decisions?

In the US, the lawyer who led the OGC team has opined '. . . that there simply would not have been an Aldrin/Dieldrin case to review. . . if the matter had been left up to the scientists designated within EPA's Office of Pesticides.'[52] When the OGC lawyers were preparing their brief for the A/D suspension hearing they naturally consulted with EPA's principal source of expertise: the Office of Pesticide Programs (OPP). This Office had been formed from the scientific divisions of EPA's regulatory predecessors — USDA, the Food and Drug Administration (FDA) and the Department of the Interior.[53] Until 1970, these scientists had effectively controlled the regulation of pesticides in the US. Now Karch has argued that, despite being employed by EPA,

> the views of many OPP scientists (on cancer testing) were generally along the lines of those in industry and USDA, who felt, for example that evidence of advanced stages of malignancy were necessary to characterize tumors as carcinogenic. Also many scientists in OPP had extensive toxicological experience in industry, USDA or FDA with substances that induce reversible effects below a certain 'threshold' of action. Thus they believed 'no-effect' levels should be set for carcinogens as they are for substances posing acute and certain other chronic hazards. Furthermore, many in OPP believed rodents (mice especially) were inappropriate animal models for cancer testing.[54]

Not surprisingly, the OPP scientists did not believe that the available data would support a case alleging the carcinogenicity of A/D. The OGC lawyers were not satisfied by the response from OPP and a bitter conflict developed between the two divisions within EPA. The intensity of the disagreement can be gauged by the fact that Kent Davis, who had transferred to EPA from FDA, and who had co-authored two of the A/D mouse studies, offered to support Shell's defence of A/D.[55]

The OGC lawyers, however, were not compelled to rely exclusively on EPA's in-house expertise. They were aware of the controversial nature of the toxicological field, and the diversity of institutions engaged in research. They therefore sought, and found, scientists who were prepared to oppose the OPP/Shell interpretation of the A/D evidence. In fact, they did not have to look very hard. Several of the scientists who subsequently appeared as EPA

witnesses, such as Saffiotti and Epstein, had been highly critical of the way in which toxicologists in FDA and OPP had discharged their regulatory duties.[56] The suspension hearing therefore provided an excellent opportunity for them to carry on this attack and establish the superiority of their arguments within an important regulatory forum.

What the transfer of regulatory responsibility achieved, then, was to shift the critical regulatory locus away from the scientists in OPP to the lawyers in OGC. This, in turn, allowed the introduction of a new group of scientific advisers into the decision-making process, whose advice was more in keeping with the interpretation which the OGC lawyers had given EPA's mandate. The lawyers could not be certain that their advisers' evaluation of the evidence would be accepted by Perlman and Train, but, by introducing the rival interpretation, they were attacking the hegemony that OPP scientists had enjoyed until that time, and providing a possible replacement.

Turning to the British case, we need to clarify how the decision-making process operates before we can evaluate how MAFF's institutional goal influenced the reception of the rival interpretations of the A/D evidence. By 1974, the PSPS Agreement had been in operation for nearly 20 years, and, in this time, a considerable amount of decision-making authority had been delegated to the Advisory Committee. Although no industrial representatives sit on the Committee or its Sub-Committee, a company may send a representative to attend any meeting at which its products are being discussed. Considerable efforts are made to achieve a consensus between government advisers and industrial scientists. The success of this process in generating consensus has meant that government departments have effectively been able to delegate their authority, and rely on PSPS advisers to negotiate a satisfactory settlement.

In this way, PSPS advisers have developed a long tradition of working closely with pesticide manufacturers and advising a government department with a predominant interest in the promotion of agricultural production. Neither of the groups with a direct interest in the regulation of potentially carcinogenic substances — workers and consumers — are represented in PSPS's decision-making process. It is hardly surprising, then, that after working in a regulatory system like PSPS, advisers like Barnes develop a very positive evaluation of pesticides. But we have also seen that this commitment informed the scientific principles used to determine

carcinogenic risk. We are now in a position to understand why PSPS advisers were so receptive to Shell's argument and, as we suggested earlier, shared the same or similar standards for inferring carcinogenicity. The tradition of close cooperation between PSPS advisers and industrial toxicologists within a context that placed a high value on agricultural production and the achievement of consensus meant that PSPS advisers had been actively involved in the development of Shell's case and in the standards they applied to their evidence.[57] In so far as the PSPS advisers had become the de facto decision-makers, they were highly receptive to an argument which they had helped develop, and which was congruent with their social and scientific commitments.

The toxicologists in OPP had developed a similar regulatory-regulatee relationship before 1970, and they employed standards for determining carcinogenic risk that were much closer to those of Shell's and the PSPS advisers than they were to those of EPA's witnesses.[58] Indeed, it seems likely that the activities of multinational companies like Shell, and the need for international regulation of foodstuffs contaminated with pesticides, provided the opportunity for these scientists to develop a good measure of consensus among themselves on the scientific basis for the regulation of potentially carcinogenic pesticides. Moreover, since little toxicological work was performed independently of government and industry, there were few scientists to challenge the approach they developed, especially outside the US. When seen in this light, EPA's finding that A/D were carcinogenic emerges as a radical departure from the 'regulatory orthodoxy' that had prevailed in the US until that time, and that probably persists in most other national regulatory systems.[59]

The closed nature of the PSPS decision-making process is also relevant to the reception of the rival interpretations of the A/D evidence. Since there was virtually no way for 'outsiders' to contribute to the British decision, there was no way that EPA's witnesses' case could be presented systematically. But even if there were, it is not clear whether any British scientific equivalents to Saffiotti and his colleagues existed, or whether they would contribute to the decision-making process if they did. So there was neither the demand, nor the supply, nor the opportunity to play a role in the British decision-making process for scientists advocating an interpretation of the A/D evidence like EPA's witnesses. The result was that the PSPS advisers had little difficulty in dismissing the EPA

decision and the rival approach on which it was based.

To sum up, the British institutions with responsibility for regulating pesticides — MAFF and PSPS — were motivated by a commitment to agricultural production that resulted in a highly favourable reception of Shell's argument. For the US decision, we concluded that EPA's institutional mission of environmental protection could be afforded only an indirect role. Through the agency of the OGC lawyers, a new group of scientists was introduced into the decision-making process who challenged Shell's and OPP's interpretation of the evidence. Once there, EPA's advisers and Shell's witnesses had to convince two laypeople that theirs was the better argument. Perlman's and Train's evaluations of the arguments were, in turn, constrained by the statute governing the regulation of pesticides. We need to determine, therefore, how far this statutory framework affected the reception of the rival arguments in EPA, and whether the absence of a similar statutory authority in Britain helps to account for the different British and US decisions.

REGULATORY STANDARDS

In general, systems of pesticide regulation employ two types of standard that influence the determination of carcinogenic risk — substantive standards and standards of proof. Taken together, these standards specify what evidence is required for the public authorities to classify a pesticide as a carcinogenic hazard. It is important, therefore, to determine how the standards used in EPA and PSPS affected the reception of the rival interpretations of the evidence on A/D.

The enactment of the Federal Environmental Pesticide Control Act (FEPCA) by Congress in 1972 established a strict, new standard whereby a pesticide could only be marketed as long as there were 'no unreasonable adverse effects on the environment.'[60] This standard was defined in FEPCA 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.'[61] Particular types of risk, such as carcinogenic ones, were not explicitly mentioned in FEPCA, but they were, nevertheless, covered by this standard. The Act also specified that, whenever required, manufacturers had to demonstrate that the use of their product

conformed to this standard — in other words, at all times, the burden of proof of safety remained with the manufacturer.

Even though the PSPS Agreement is non-statutory, it does declare that '...if a chemical is known or shown to be a carcinogen it will not be permitted to occur as a residue in food.'[62] Presumably, this standard applies both before and after products are marketed, though manufacturers will not necessarily have to provide the evidence in the latter case.[63] The burden of proof, therefore, is more ambiguous in the British system of regulation than in the American.

Insofar as these standards do not define what is meant by 'carcinogen', 'unreasonable risk', 'burden of proof', and so on, they are too broad to account for the differing US and British decisions on A/D. To evaluate the significance of these standards, we need to see how they were interpreted in the two decision-making contexts and whether they were influenced in this by their statutory or voluntary form.

(A) *US Standards*

The EPA suspension decision apparently did not turn on the distribution of the burden of proof. Perlman stated in his opinion that

> ...the respondent [i.e. EPA], who has the burden of going forward to present an affirmative case for suspension, but not the ultimate burden of persuasion as to safety, has in fact satisfied the burden of proof, which is not his, that the chemicals in question pose a high risk of causing cancer in man.[64]

More substantively, Perlman's and Train's interpretation of the FEPCA standard did not require EPA to unequivocally demonstrate A/D's carcinogenicity: '...suspension is to be based upon potential or likely injury and need not be based upon demonstrable injury or certainty of future public harm.'[65] They were supported in this by similar interpretations of other environmental statutes. A number of precedents had been established in the US courts so that if a hazard was thought to be sufficiently serious, the decision-maker was required to make a policy determination of the likely effects of the hazard-producing activity, rather than waiting for definite evidence of harm to accumulate before deciding whether the risk was acceptable or not.[66] For serious hazards, then, rather less evidence would be required before

treating an activity as if it were hazardous, than would be the case if the strictest standards of scientific causality were to be met. Both Perlman and Train believed that cancer fell into this category of 'serious hazard' and that it should be dealt with accordingly.

The strong Congressional aversion to carcinogens in the US food supply, expressed in the Delaney Clause, supported Perlman's and Train's view of the seriousness of carcinogenic hazards, and provided additional justification for a cautious approach to decision-making on pesticides. The clause was an amendment to the Federal Food, Drug and Cosmetic Act and stated that

> ...no [food] additive shall be deemed safe if it is found to induce cancer when ingested by man or animal, or if it is found, after tests which are appropriate for the evaluation of the safety of food additives, to induce cancer in man or animals.[67]

Train was aware of the fuzzy distinction between 'intentional' food additives and 'unintentional' contaminants and argued that

> ...since the Delaney Amendment does prohibit the setting of safe levels/tolerances of carcinogenic food additives, and since Aldrin-Dieldrin is present as a residue in processed foods, the Administrator has a particular burden to explain a basis for a decision permitting continued use of a chemical known to be carcinogenic in laboratory animals.[68]

It is likely, then, that Perlman's and Train's interpretations of the FEPCA standard would have made them more receptive to EPA's argument than Shell's. EPA's use of cancer principles as guides to decision-making was tailored to their needs. The principles assumed the necessity of making policy judgments when determining carcinogenic risk, and expressed a preference for minimizing carcinogenic risk. In contrast, Shell's argument seemed to reflect an insufficient appreciation of the novel regulatory situation in which they found themselves. Their stress on causality and the demonstration of harm was precisely the approach that had been rejected in recent court decisions.

Nevertheless, Perlman did not justify his decision in these terms. As we saw earlier, he thought his decision represented the conventional wisdom of the scientific community. But if this were so, why didn't experts in other countries, and in the US before 1974, adopt the same view? Even if one accepts that these other decision-making forums were dominated by pro-pesticide interests, this still leaves the problem of explaining why Perlman and Train preferred

EPA's interpretation of the evidence. Should we accept Perlman's argument that the greater scientific merit of EPA's case was the decisive factor? Or should we afford the legislative tradition, within which Perlman evaluated the rival interpretations, the greater role, despite Perlman's indications to the contrary?

Unfortunately, it is not so easy to disentangle these factors. Nevertheless, we think that the legal framework undoubtedly helped structure the way in which Perlman evaluated the evidence. Perlman's and Train's acceptance of EPA's cancer principles, and the policy judgments associated with them, can only be understood in terms of their interpretations of their statutory responsibilities. Moreover, FEPCA determined that two laypeople, rather than scientists, were in the key decision-making roles.[69] So, unlike the PSPS advisers, EPA's decision-makers were not committed to a particular way of assessing carcinogenic risk, and they were unaware of the 'regulatory orthodoxy' that had prevailed until that time. At the same time, we think it is possible to identify some reasons, independent of immediate legislative concerns, that may have influenced Perlman's decision.

Firstly, EPA's witnesses were generally highly qualified scientists, working in prestigious institutions rather than in private industry or government regulatory agencies. The claim that they represented 'the most advanced research findings and policy of both national and international cancer experts and agencies',[70] appeared, therefore, to have some substance. In contrast, Shell's witnesses had the disadvantage of representing a narrower, sectional interest. Furthermore, Shell's attempt to bolster the universality of their argument by relying on witnesses associated with international agencies backfired somewhat when Perlman discovered that the views of the Joint FAO/WHO Committee on Pesticide Residues were largely determined by one of Shell's witnesses, Francis Roe.[71] As the report was being used to support a controversial argument — Shell's 'two species' criterion — this discovery was quite damaging to a case purporting to represent the 'state-of-the-art' views of the scientific community.

Secondly, the evidence that EPA's witnesses presented within the framework of the cancer principles was generally coherent and consistent. The only disagreement was on an issue that, as it happened, was not critical to the outcome of the case.[72] Shell, too, had disagreements among their witnesses and their position shifted during the course of the proceedings. More significantly, their demand for evidence of causality contrasted unfavourably with their use of

poorly corroborated biochemical evidence, and emphasized an apparently narrow dimension to their argument. The quality of Shell's work was also brought into question by EPA's reinterpretation of existing data in a way which suggested that Shell had systematically underestimated A/D's carcinogenic risk. Furthermore, the scientists who developed the methodology that Shell employed in their two mouse studies testified that Shell's analysis was 'almost guaranteed to give non-significance for even the strongest carcinogens.'[73]

Finally, the form of the two arguments may have influenced Perlman's and Train's judgment as to which approach would provide the better scientific basis for future regulation. We saw in an earlier section how the scientific approach of the EPA witnesses was generally more theoretical and universal in character than Shell's. In the context of the suspension hearing, this may have influenced Perlman's assessment of the relative scientific merit of EPA's and Shell's arguments.

We may conclude, then, that both the legal framework within which the A/D evidence was considered, and various features of the arguments themselves, led Perlman and Train to prefer EPA's interpretation of the A/D evidence to Shell's. Considerably more research would be needed to clarify which of these factors was the more important. Nevertheless, we would emphasize the fundamental importance of the legal framework in this connection. Whereas it is not clear that other laypeople, or uncommitted scientists, would be persuaded by EPA's case alone, it seems more likely that they would reach the same conclusion as Perlman and Train when they evaluated the evidence within the framework of FEPCA, the Delaney Clause, and US environmental law. Thus, we would argue that the motivation for the US decision on A/D is to be found in the American legal system rather than the 'conventional wisdom of the scientific community.'

(B) *British Standards*

The interpretation that PSPS advisers gave the PSPS cancer standard was subject to none of the same constraints as the interpretation of the FEPCA standard. To the extent that there was no statutory framework governing the interpretation of the cancer standard, the institutional constraints we considered in the last section were correspondingly more important in determining the outcome of the A/D review.

The PSPS Agreement did not express a strong commitment to the prevention of cancer, and, if Barnes' views are typical, British decision-makers did not think that banning pesticides would do much to prevent cancer anyway. Similarly, no new tradition had emerged in British legal, or political, or scientific thinking to guide the specification of serious but uncertain risks. As a result, PSPS and other British regulatory systems have expected the traditional requirements of scientific causality to be satisfied before labelling a chemical carcinogenic, just as OPP scientists had done when USDA exercised regulatory responsibility for pesticides. This approach was clearly more congenial to the PSPS scientific advisers and their industrial counterparts. Not only was it more in keeping with their professional training than any other, but also the way in which the balance of doubt was distributed (in favour of pesticides) was more consistent with the ethos of PSPS.

Thus, the very 'weaknesses' of Shell's argument in the suspension hearing became their 'strengths' in PSPS. For example, the demands for causal mechanisms and demonstration of harm were all quite reasonable, and compelling, in the PSPS context. Similarly, PSPS advisers had been closely involved in FAO and WHO activities, and the Shell witness who had largely determined the views of the report that Perlman rejected had served on a number of British advisory committees. It is not surprising, therefore, that British medical opinion was irritated by Perlman's treatment of Roe's evidence, nor that the EPA decision was regarded as 'unscientific' and the unfortunate result of leaving such decisions to laypeople.

Finally, if we speculate on the decision that would have been reached if PSPS were statutorily based, there is no guarantee that the British decision would be reversed. On the contrary, it is quite likely that A/D would not be considered carcinogenic as long as the following conditions pertain: the assessment is based on the traditional notion of scientific causality; the burden of proof of safety is not unequivocally with Shell; the PSPS decision-making organization (with its strong commitment to agricultural production) remains intact; and there is an absence of scientists and citizens advancing more health-protective arguments. In Britain, as in the US, the critical point is not the existence of a statute, but the type of framework, legal or otherwise, that guides the determination and assessment of uncertain risks.

CULTURAL AND ECONOMIC FACTORS

In complex policy issues such as this, where the outcome is determined by the interaction of several causal factors, the roles of cultural and economic factors are diffuse and difficult to evaluate. Their influence is generally mediated through specific institutions and practices, but is, nonetheless, real. For our purposes, we need consider only four aspects of the British and US cultural and economic contexts: environmental movements and their definition of the pesticide issue; economic well-being; availability and type of expertise; and styles of government — particularly their use of law and science in environmental decision-making.

There is little doubt that the environment was a much more significant issue, politically, in the US than in Britain.[74] For whatever reasons, the environment became one of the major foci for the unrest that characterized American society in the late 1960s. The movement that mobilized around environmental issues became sufficiently strong to elicit a major response from US political and legal institutions. The creation of EPA, the enactment of FEPCA, and the reformulation of the principles on which technological risks were managed — all important factors in the US A/D decision — must be seen within the context of this movement.

In Britain, political conflicts did not develop around environmental issues in the same way, nor with the same intensity. The British environmental movement — if one may call it that — did not secure the same support, nor develop the same strength, as its US counterpart. Thus, in contrast to the US, pesticides were not a political issue in Britain after 1969.[75] There was no group lobbying for the transfer of primary regulatory responsibility from MAFF to a less compromised agency, for the enactment of more effective statutory controls nor for the removal of A/D from the British market. Finally, there was no constituency that could provide senior administrators for a new regulatory agency, nor monitor their activities, as groups like EDF did for EPA.

It is also interesting to contrast the type of concern expressed about pesticides in the two countries. In Britain, this concern was largely confined to wildlife conservation, an issue that traditionally has mobilized strong support. In the US, public health issues, and particularly cancer hazards, have also concerned critics of pesticides.[76] Indeed, environmentalists have generally capitalized on the widespread fear of cancer among the US population, and

have tended to redefine environmental protection as health protection, and health protection as prevention of cancer. It was no accident, therefore, that the OGC lawyers based their suspension case on the carcinogenic hazard that A/D posed, rather than some other 'unreasonable adverse effect on the environment'.

Fear of cancer has not carried the same political weight in Britain as in the US, especially in policy circles. British decision-makers and their advisers have not been convinced that cancer rates would be much reduced by the costly regulation of technologies like pesticides. It is not surprising, then, that there has been virtually no expression of public concern about A/D in Britain either before, during, or after the suspension hearing.[77]

One reason why the US political system has been more responsive to environmental demands than the British is the relative strengths of their national economies. The US has a much larger and wealthier economy than Britain's, and this has allowed it to absorb the costs of environmental and health protection more readily. Technologies have been regulated on the assumption that American industry is sufficiently robust to operate within these new constraints. In contrast, Britain's weaker economic position has legitimated a continued stress on the promotion of economic development through technological change. British decision-makers have been very reluctant to encumber industry with unnecessary regulation, and this interest has continued to inform the British management of technological risks.

Taken together, the different national resources and evaluations of dread diseases such as cancer have affected the availability, and type, of expertise for the assessment of carcinogenic risk.[78] In both countries, toxicology has been, historically, most closely associated with the private sector and the government agencies responsible for the regulation of toxic hazards. This has been reinforced by the failure of toxicology, as a scientific field, to develop a unique identity within national university systems. Still, the greater resources and commitment to health protection in the US has resulted in the development of a larger, more diverse biomedical research system, in which more basic research institutions, independent of regulator-regulatee interests, operate. These, in turn, have proved to be appropriate locations for the development of alternative approaches to the assessment of carcinogenic risk — an important precondition for the US A/D decision. This contrasts with the British biomedical research system, which is smaller, more closely tied to

regulatory work, and apparently lacking the diversity of approaches to the assessment of carcinogenic risk that exists in the US.

In this connection, the different scientific traditions that the cultural and economic contexts of the two countries supported may also have contributed to the different decisions. McGinty, for instance, has argued that the British emphasis on epidemiology owes much to the influential tradition associated with Sir Richard Doll.[79] Doll and his co-workers had carefully demonstrated how cigarette smoking was the main cause of increasing rates of lung cancer in Western countries. This experience, and the authority it conferred on British epidemiology, may, therefore, have structured the way in which British scientific advisers regarded 'environmental' cancer hazards and the best means for their detection. This contrasts with the US situation, in which the NCI has tended to emphasize animal rather than human studies because of their relative costs.[80] Considerably more research would be needed to evaluate the contribution of these differing traditions to the British and US A/D decisions. Nevertheless, they are consistent with the respective decisions and would at least have supported their rationales.

Turning, finally, to the styles of government, we can see how the British and US systems for regulating pesticides are guided by the norms that generally inform the execution of public policy in these two countries. In Britain, consensus is generally achieved by restricting both access to, and information of, the decision-making process, whilst allowing maximum flexibility for negotiation among the most directly involved parties. This contrasts with the American pluralist tradition in which great importance is attached to the clash of conflicting ideas and the evolution of policy through adversarial processes.

The different approaches are clearly illustrated by contrasting the British and US attitudes to the legal process. In Britain, the law is often thought to breed inflexibility and rigidity, and to induce confrontation, polarization and irrationality — all of which hinder the achievement of consensus. In policy fields like the environment, therefore, a voluntary approach is preferred, and the law is used only as a last resort. Within the American pluralist tradition, however, the courts are the archetypal mechanism for conflict resolution. The participation, openness, and confrontation that they allow have served to assimilate disaffected groups back into the system. Thus, in contrast to Britain, the courts have been a

critical locus for the development of US environmental policy and the management of technological risks.

The British and US styles of government have also structured the roles that science and scientists played in such decisions as that on A/D. While toxicology and its practitioners have generally been 'politicized' by their involvement in government decision-making[81] (for instance, by governments intervening in what was traditionally the autonomous sphere of the scientist to prescribe 'good laboratory practice,' and endorsing particular experimental designs and interpretive principles to be used in risk assessment), the respective styles of government have politicized them in different ways. In the US, toxicologists have been enlisted by conflicting social groups, especially industrialists and environmentalists, to support their arguments, and, as a result, have been drawn into adversarial political processes. Indeed, the political and legal forums in which US toxicologists confront each other have become important new battlegrounds for competing groups of scientists trying to establish the superiority of their approach. Pfizer has described this changed environment in which the toxicologist works as

> ...at once both frustrating and stimulating; frustrating because he doesn't have the answers to many of the perplexing questions being asked; frustrating to some toxicologists because their time honoured methods for making judgments about safety evaluations are being challenged; stimulating because suddenly there are a multitude of people interested in his professional activities... He can expect his data to be scrutinized by non-scientists, to be interpreted in the newspapers and in legal hearings, to face requirements for exactness and statistical validity with increasing rigour. The life of the toxicologist will never be the same.[82]

British toxicologists have not escaped politicization by their contact with government, but it has assumed a less obvious form. Rather than publicly confronting each other, toxicologists have been enlisted by the British government to generate a consensus and legitimate political decisions. In contrast to the conflicts among experts that characterize many American decisions in this field, British decisions emerge from a closed decision-making process with the apparently uncontroversial and authoritative support of science. Whereas US decision-making institutions depend upon, and, to some extent, generate conflicts among experts, British institutions tend to rely upon singular sources of expertise. In the A/D case, the result was the availability of an adversarial forum in

the US, and scientists eager to enter it, whereas in Britain there was neither the forum nor, apparently, the scientists.

CONCLUSION

This case study should, at the very least, rid readers of the notion that the relationship of scientific knowledge to public policy is straightforward. In order to provide a reasonable account of why two countries derived different conclusions from the same scientific evidence, we had to invoke a set of interacting, and mutually supporting factors for each decision: the uncertainty inherent in the relevant scientific field; the application of different scientific standards, motivated by different scientific and social commitments; the bureaucratic politics of the agencies with responsibility for regulating pesticides; the way in which standards are defined in particular systems of regulation; and, finally, a series of contextual factors. Whilst many interesting questions were raised by the analysis, we will conclude this paper by reconsidering, in the light of this case study, some features of the science-public policy relationship outlined in the introduction.

Firstly, although this study has highlighted some important differences between the British and US approaches to the regulation of potentially carcinogenic pesticides, it is significant that both decisions were ultimately justified by recourse to science — in the British case by the traditional notion of scientific causality, and in the US case with reference to the consensus of the scientific community. Both decisions were, thus, presented as in some sense 'springing directly from the facts'. This testifies to the considerable cultural authority of science in these countries, particularly as a means of solving policy problems.

Our analysis of the A/D case, however, calls into question any exclusively scientific justification for such decisions. Thus, the British demand for evidence of causality, and the hostility towards the EPA decision expressed in British medical journals, both tend to conceal that the decision to wait for definite evidence of harm to accumulate is just as much an ethical and political choice as the decision to treat risk determination as a policy issue. Whether or not one agrees with the ethical and political commitments informing the US decision, the acknowledgement of this dimension is

surely preferable to presenting decisions as the result of a methodological imperative.

Despite this insight, Perlman's treatment of the A/D decision as a policy issue, and his justification of it as the conventional wisdom of the scientific community, was rather like having one's cake and eating it. It may well be, as we have tried to show, that Perlman found 'good reasons' for preferring EPA's argument to that of Shell. But it is not clear whether they would have been compelling without the supporting *statutory* framework. Moreover, it is difficult to see in what sense the views of EPA's witnesses were more representative of the scientific community. The notion of 'community' seems inappropriate for a collection of scientists working largely in industrial and governmental laboratories, and, if anything, Shell's argument was more widely accepted in these circles than was EPA's. In cases such as this, then, science can only inform but not determine policy decisions. The attempt to ground policy decisions exclusively in science, for whatever motive, is inappropriate and ill-conceived.

Secondly, we found that the fact-value distinction was of little use beyond a trivial level for analyzing either the decisions or the roles of the scientific advisers. The apparently factual determinations of A/D's regulatory status involved considerably more than 'the facts', as did the advice on which they were based. Positivist-inspired views of science, which incorporate the fact-value distinction in their analysis, are thus inherently limited by their failure to examine the context of scientific advice and the way in which it can transmit social commitments.

Similarly, this case study supports the view that the traditional division of labour between science and public policy, or at least the way it is usually perceived, is breaking down in complex areas of decision-making like risk assessment. Scientists do not operate in an exclusively factual arena, and decision-makers play a more active role than is usually realized in determining what is to count as a fact. If both parties are to operate effectively, and in good faith, they will have to face up to this, and adjust their roles accordingly. We would argue that the minimum requirements for this are for scientists to accept responsibility for the commitments they inevitably make when devising approaches for the study of poorly understood phenomena. Moreover, they should be prepared to elaborate and clarify the implications of those commitments, especially in relation to the ethical and political considerations that

arise in risk assessment. Lay participants in risk decision-making, in their turn, will need to develop a more subtle understanding of both the strengths and limitations of the contributions that science can make to the clarification of these difficult issues. Unless they do, it seems likely that the social control of technological change will continue to be impaired by the manipulation of outmoded ideologies of science by competing or dominant social groups.

• NOTES

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1. S. Hadden, 'Technical Advice in Policy Making', in J. Haberer (ed.), *Science and Technology Policy* (Lexington, Mass.: Lexington Books, 1977), 81-98, and J. Milch, 'Technical Advice and the Democratic Process', paper presented to AAAS meeting, Washington, DC, 12-17 February 1978, provide good reviews of this literature.

2. The need for comparative studies in the social studies of science field has been most recently argued by R. Brickman and A. Rip, 'Science Policy Advisory Councils in France, The Netherlands and the United States: A Comparative Study', *Social Studies of Science*, Vol. 9 (1979), 167-98.

3. H. Nowotny, 'Scientific Purity and Nuclear Danger: The Case of Risk Assessment', in E. Mendelsohn et al. (eds), *The Social Production of Scientific Knowledge* (Dordrecht/Boston, Mass.: D. Reidel Publishing Co., 1977), 243-64.

4. R. Johnston and D. Robbins, 'The Development of Specialties in Industrialised Science', *Sociological Review*, Vol. 25 (1977), 87-108; and D. Robbins and R. Johnston, 'The Role of Cognitive and Occupational Differentiation in Scientific Controversies', *Social Studies of Science*, Vol. 6 (1976), 349-68.

5. The initial analysis of these decisions appeared in B. Gilllespie, *British Control of Pesticide Technology* (unpublished PhD thesis, University of Manchester, 1977), Chapter 8.

6. M. Sloan, 'A US Historical Account — Aldrin/Dieldrin Registration, Uses and Sales', evidence presented at EPA's cancellation hearing (no date).

7. L. McCray, 'Mouse Livers, Cutworms and Public Policy: EPA Decision Making for the Pesticides Aldrin and Dieldrin', in R. Burt et al. for the Committee on Environmental Decision Making, *Decision Making in the Environmental Protection Agency*, Vol. IIa (Washington, DC: NAS, 1977), 58-118, has provided a good discussion of this decision.

8. See P. Spector, 'Regulation of Pesticides by the Environmental Protection Agency', *Ecology Law Quarterly*, Vol. 5 (1976), 233-63, for a relevant discussion of the statute governing the US regulation of pesticides.

9. McCray, op. cit. note 7, 65.

10. Environmental Defense Fund, Inc. v. Environmental Protection Agency, 210 F2d (1975), 1292.

11. Personal communication (BG), MAFF, 17 June 1976.

12. 'Insecticides and Cancer' (Editorial), *British Medical Journal* (25 January 1975), 170.

13. For a more detailed discussion of the British regulation of pesticides, see Gillespie, op cit. note 5, and B. Gillespie, 'British "Safety Policy" and Pesticides', in R. Johnston and P. Gummett (eds), *Directing Technology* (London: Croom Helm, 1979) 202-24.

14. T. Maugh, 'Chemical Carcinogens: I. The Scientific Basis for Regulation,' *Science*, Vol. 201 (29 September 1978), 1200-05, and 'II. How Dangerous are Low Doses?', *Science*, Vol. 202 (6 October 1978), 37-41; and D. Eva, *Toxicology and Society* (unpublished MSc thesis, University of Manchester, 1975).

15. Maugh, ibid., II, 1200, lists 26 'accredited chemical carcinogens'.

16. Shell Chemical Company, 'Brief. Aldrin/Dieldrin Consolidated Suspension Hearing' (Washington, DC, 13 September 1974), III.

17. Shell Chemical Company, 'Objections and Supplemental Brief to the Administrator on Suspension' (Washington, DC, 24 September 1974), I-9.

18. See K. Jager, *Aldrin, Dieldrin, Endrin and Telodrin* (London/Amsterdam/New York: Elsevier, 1970).

19. Maugh, op. cit. note 14, I.

20. Shell Chemical Company, op. cit. note 16, III.

21. 'Consolidated Aldrin. Dieldrin Hearing', *Federal Register*, Vol. 39, No. 102 (18 October 1974), 37269. We shall refer to this below as 'EPA hearing'.

22. K. Davis and O. Fitzhugh, 'Tumorigenic Potential of Aldrin and Dieldrin for Mice', *Toxicology and Applied Pharmacology*, Vol. 4 (1962), 187-89; K. Davis, 'Pathology Report on Mice for Aldrin, Dieldrin, Heptacholor Expoxide for Two Years', internal FDA memo (1965); W. MacDonald et al., 'The Tumorigenicity of Dieldrin in the Swiss Webster Mouse', unpublished (1972); A. Walker et al., 'The Toxicology of Dieldrin (HEOD) I: Long Term Oral Toxicity Studies in Mice', *Food and Cosmetic Toxicology*, Vol. II (1973), 415-32; E. Thorpe and A. Walker, 'The Toxicology of Dieldrin (HEOD) II: Comparative Long Term Oral Toxicology Studies in Mice with Dieldrin, DDT, Phenobarbitone, ß-BHC and γ-BHC', *Food and Cosmetic Toxicology*, Vol. II (1973), 433-42. These and other experiments involving A/D were reviewed in S. Epstein, 'The Carcinogenicity of Dieldrin. Part 1', *Science of the Total Environment*, Vol. 4 (1975), 1-52.

23. Office of General Counsel, Environmental Protection Agency, 'Respondent's Brief. Proposed Findings and Conclusions on Suspension' (Washington, DC, 16 September 1974).

24. N. Karch, 'Explicit Criteria and Principles for Identifying Carcinogens: A Focus of Controversy at the Environmental Protection Agency', in Burt et al., op. cit. note 7, 134.

25. 'Prudent policy indicates that every possible measure should be taken to eliminate human exposure to chemical compounds as soon as their carcinogenic nature is identified': Office of General Counsel, op. cit. note 23, 41.

26. Epstein, op. cit. note 22, 207.

27. EPA hearing, 37258-59.

28. I. Nisbet, 'Measuring Cancer Hazards', *Technology Review*, Vol. 78 (1975), 8-9.

29. EPA hearing, 37259.

30. 'Insecticides and Cancer', *British Medical Journal* (25 January 1975), 170; 'Does This Chemical Cause Cancer in Man?', *The Lancet* (14 September 1974), 629-30; and 'Seventeen Principles About Cancer, Or Something', *The Lancet* (13 March 1976), 571-73.

31. *Further Review of Certain Persistent Organochlorine Pesticides Used in Great Britain*. Report by the Advisory Committee on Pesticides and Other Toxic Chemicals (London: HMSO, 1969), para 79-83. This was the evidence that Shell conceded was methodologically flawed at the suspension hearing.

32. Ibid., para 81. There is no evidence that these experiments were ever performed, which is perhaps not surprising in view of Shell's research expenditure on A/D.

33. *Aldrin and Dieldrin Residues in Food*. Report by the Food Additives and Contaminants Committee (London: HMSO, 1967).

34. *Review of the Persistent Organochlorine Pesticides*. Report by the Advisory Committee on Poisonous Substances Used in Agriculture and Food Storage (London: HMSO, 1964), para 77.

35. Panel on Carcinogenic Hazards, 'Carcinogenic Risks in Food Additives and Pesticides', *Monthly Bulletin of the Ministry of Health*, Vol. 19 (1960), 108-12.

36. Ministry of Agriculture, Fisheries and Food, *Pesticides Safety Precautions Scheme Agreed Between Government Departments and Industry* (London: MAFF, Pesticides Branch, revised March 1971), para 1.4. We shall refer to this as 'PSPS Agreement'.

37. In accepting the arguments of EPA witnesses on this point, Perlman ruled: 'Whether the agent actually is a sine qua non of the observed response or merely enhances a virus or some other factor found in the host animal is irrelevant unless and until we know that similar factors are not also found in man': EPA hearing, 37257.

38. Panel on Carcinogenic Hazards, op. cit. note 35, Appendix, para 7.

39. Johnston and Robbins (1977), op. cit. note 4.

40. Gillespie, op. cit. note 5, 283-84.

41. EPA hearing, 37255.

42. Since its foundation in 1947, the starting point for all its research projects had been 'substances of potential interest to industry and agriculture about whose toxicity little was known': *MRC Annual Report*, April 1968-March 1969, HC 299, 82.

43. R. Rettig, *Cancer Crusade: The Story of the National Cancer Act of 1971* (Princeton, NJ: Princeton University Press, 1977).

44. U. Saffiotti, 'Comments on the Scientific Basis for the "Delaney Clause",' *Preventive Medicine*, Vol. 2 (1973), 128-29.

45. J. Barnes, 'Carcinogenic Hazards from Pesticide Residues', *Residue Reviews*, Vol. 13 (1966), 79.

46. Saffiotti, op. cit., note 44, 127.

47. J. Barnes, 'Assessing Hazards from Prolonged and Repeated Exposure to Low Doses of Toxic Substances', *British Medical Bulletin*, Vol. 31 (1975), 198-99.

48. Although the NCI is a government laboratory, its employees tend to be well-integrated into the open, peer review processes characteristic of collegiate forms of

occupation control. On molecular biology, R. Young, in 'Evolutionary Biology and Ideology: Then and Now', *Science Studies*, Vol. 1 (1971), 179, has written:

> ...the fundamental paradigm of explanation — the goal of all science — has been to reduce or explain all phenomena in physico-chemical terms. The history of science is routinely described as a progressive approximation to this goal. This is the metaphysical and methodological explanation for the fact that molecular biology is the queen of the biological sciences and the basis on which other biological (including human) sciences seek, ultimately, to rest their arguments.

49. This argument is documented in Gillespie, op. cit. note 13.

50. J. Blodgett, 'Pesticides: An Evolving Technology', in S. Epstein and R. Grundy (eds), *The Legislation of Product Safety*, 2 Vols. (Cambridge, Mass.: MIT Press, 1974), Vol. 2, 197-287.

51. P. Weaver, 'Regulation, Social Policy and Class Conflict', *Public Interest*, Vol. 50 (Winter 1978), 45-63.

52. J. Kolojeski, 'Federal Administrative Trial of a Carcinogen: the EPA Aldrin/Dieldrin Pesticide Case', in H. Hiatt et al., *Origins of Human Cancer*, Book C (Cold Spring Harbor, Mass.: The Laboratory, 1977), 1719.

53. See Spector, op. cit. note 8, for further details of this regulatory reorganization.

54. Karch, op. cit. note 24, 161.

55. McCray, op. cit. note 7, 104.

56. The chairperson of the Mrak Commission on Pesticides and Their Relationship to Environmental Health (Washington, DC: US GPO, 1969) reported: 'Pathologists of the (National) Cancer Institute made it known that they did not think much of the pathologists representing the Food and Drug Administration'. See E. Mrak, 'Some Experiences Relating to Food Safety', in F. Coulston and F. Korte (eds), *Environmental Quality and Safety: Global Aspects of Chemistry, Toxicology as Applied to the Environment*, Vol. 15 (Stuttgart: George Thieme, 1976).

57. Shell's '...research workers discussed the significance of their findings on many occasions with the scientists advising the [PSPS] committee': *BMJ* Editorial, op. cit. note 12.

58. Political scientists are familiar with the idea of regulators being 'captured' by their regulatees: see, for example, Weaver, op. cit. note 51. We would argue that this notion can be extended to encompass the scientific approaches that are developed for regulatory purposes.

59. This argument differs from Karch, op. cit. note 24, who accepts that EPA's witnesses' position was the better established in the scientific community. Still, we would argue that this underestimates the novelty of the EPA position, and overestimates the 'unscientificity' of their opponents.

60. PL 92-516, Section 3. See also Spector, op. cit. note 8.

61. Ibid., Section 2 (bb).

62. PSPS Agreement, Appendix F, para 1.1

63. Personal communication (BG), MAFF, 28 April 1977.

64. EPA hearing, 37259.

65. Ibid., 37265.

66. For a more detailed discussion of this difference, see M. Gelpe and A. Tarlock, 'The Uses of Scientific Information in Environmental Decision Making', *Southern California Law Review*, Vol. 28 (1974), 371-427.

67. 21 USC Section 348 (c) (3) (A) (1970).
68. EPA hearing, 37267.
69. We are using the term 'layperson' in the sense of non-scientist. Of course, Perlman's training as a lawyer would also have predisposed him to approach the evidence from a particular perspective which, in this context, cannot be regarded as neutral.
70. Office of General Counsel, op. cit. note 23, 29-31.
71. EPA hearing, 37256.
72. Some of EPA's witnesses disagreed over whether the rat experiments indicated a carcinogenic hazard associated with A/D. This was also the only matter that Perlman and Train disagreed on. But it was not important because they both accepted that the evidence from the mouse was sufficient for their purposes.
73. EPA hearing, 37268, fn. 55.
74. C. Enloe, *The Politics of Pollution in Comparative Perspective* (New York: David McKay Company, 1975).
75. Gillespie, op. cit. note 5, Chapters 6 and 7.
76. See, for instance, R. Carson, *Silent Spring* (Boston, Mass.: Houghton Mifflin, 1962), Chapter 14.
77. With the exception of Gillespie et al., 'A Tale of Two Pesticides', *New Scientist* (9 February 1978), 350-52.
78. S. Strickland, *Politics, Science and Dread Diseases* (Cambridge, Mass.: Harvard University Press, 1972), and J. Sadler, *Elites in Science: the Case of Cancer Research* (unpublished MSc thesis, University of Manchester, 1976).
79. L. McGinty, 'Controlling Cancer in the Workplace', *New Scientist* (22/29 December 1977), 758, and 'Human Guinea Pigs?', *New Scientist* (10 August 1978), 386.
80. Kolojeski, op. cit. note 52, 1722.
81. S. Blume, *Toward a Political Sociology of Science* (New York: Academic Press, 1974), 212-14.
82. E. Pfizer, 'Toxicology', in L. Cralley (ed.), *Industrial Environmental Health* (New York: Academic Press, 1972), 71-72.

The authors originally worked together in the Department of Liberal Studies in Science at the University of Manchester. Since then, *Brendan Gillespie* has examined US policies for controlling carcinogenic and other hazards whilst a post-doctoral associate in Cornell University's Program on Science, Technology and Society. *Dave Eva* has joined the Workers' Educational Association and teaches shop stewards about the control of hazards in the workplace. *Ron Johnston* has worked in the Policy Division of the Australian Department of Science during a sabbatical leave of absence from Manchester. He has recently been appointed to the Foundation Chair of History and Philosophy of Science at the University of Wollongong.

Authors' addresses (respectively): Program on Science, Technology and Society, Cornell University, Ithaca, NY 14853, USA; 191 Rake Lane, Wallasey, Merseyside, England; Department of History and Philosophy of Science, University of Wollongong, Wollongong, New South Wales, Australia 2500.

Notes

[2] **Science Policy Advisory Councils in France, the Netherlands and the United States, 1957-77: A Comparative Analysis**

Ronald Brickman; Arie Rip

Social Studies of Science, Vol. 9, No. 2, Theme Issue: Politics and Science. (May, 1979), pp. 167-198.

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David Robbins; Ron Johnston

Social Studies of Science, Vol. 6, No. 3/4, Special Issue: Aspects of the Sociology of Science: Papers from a Conference, University of York, UK 16-18 September 1975. (Sep., 1976), pp. 349-368.

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[14] **Chemical Carcinogens: The Scientific Basis for Regulation**

Thomas H. Maugh II

Science, New Series, Vol. 201, No. 4362. (Sep. 29, 1978), pp. 1200-1205.

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[48] **Evolutionary Biology and Ideology: Then and Now**
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