Cancer Battles

and the

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by Dave Bennett, PhD

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Policy And Science Need Not Be Related

Let us suppose a group of policy makers proposed to reduce the volume of chemical pesticides released into the environment. They made it a rule that only a small, selected number of chemical pesticides were to be allowed, based solely on agricultural need. Asked to defend their stance, they said that it was not a good idea to spread chemical poisons needlessly over the human and physical environment. There were effects on agricultural productivity, to be sure, but these could be mitigated by non-chemical pest control methods. Also, we should measure agricultural output by food value in relation to depletion of soil quality, not simply by volume of product.

Those opposed to the move replied that if there were any detriments to human health, they were small compared with the agricultural benefits of the unrestricted use of pesticides. Besides, most of the human detriments could be avoided by the licensing of pesticide applicators, strict rules over spraying and the use of personal protective equipment. No doubt, chemical pesticides degrade the environment, but here again the price is worth paying in terms of agricultural productivity. The degradation of the environment is minuscule compared with the move from wild country to agricultural settlement.

The idea of allowing only a small number of pesticides is essentially the pest control policy of Sweden, Austria and the erstwhile German Democratic Republic.

The point of this policy debate is that it can be conducted entirely without reference to scientific expertise. Such a scenario could not easily be envisaged in the U.S., not least because of the presence of a powerful business lobby representing a major manufacturing industry. More pertinent is the objection that the US is not a parliamentary democracy and the structure of its political system is such as to rule out radical moves of this sort. It may also be true that property rights in the U.S. Constitution would prohibit discrimination over manufacturers' different products. It is bad enough that whole classes of product are taken off the market, even worse when one manufacturer's pesticide for a given set of crops is allowed, when another's pesticide, chemically different but performing the same function, is prohibited. The proposed scheme is against equity and natural justice.

The least that would have to happen is that there be some rational way of deciding which products are to be allowed and which not. There are several ways in which this could be done. The first is determining a yardstick of agricultural efficacy (how well does the pesticide work) and allowing only those above a certain threshold to qualify. But this would be so uncertain, time-consuming, variable and tenuous that it would be likely in practice to be stalled indefinitely. Enter the scientific experts.

Risk Assessment is Not the Only Science of Environmental Health

The first reaction of most scientific experts would be to challenge the usefulness and propriety of the scheme proposed. They would offer the only tool on hand: risk assessment. Do a risk assessment on each of the candidates for approval or rejection. If a pesticide really does pose a significant risk: take it off the market. The characteristic of such assessments is that they are not based centrally on epidemiology or direct evidence of human health effects. When pesticides are first approved, there is little such evidence available and once the evidence does start to come in, it is mighty difficult to get the approval cancelled. So risk assessments are usually based on calculation from sources of non-human evidence and data. A threshold of harm also has to be established, estimated or calculated from the data. Under this procedure, almost nothing is ruled out. Risk assessment allows pretty much every application for approval to be accepted. It could certainly not be used to provide the rationale for a radical proposal such as the one described.

Some other scientific rationale must be sought. This can be discerned from the following table (1):

MOE SCORING SYSTEM SUMMARY CHART

ELEMENT NAME	ENDPOINT & UNITS	SCORING CRITERIA 0	4	7	10
Environmental Transport	Percent partitioning, measured or predicted	Any single medium contains >95% of the total amount released	No one medium contains >95%, and only one medium contains ≥5% of the total amount released	Two media each contain ≥5% of the total amount released	Three or more media each contain ≥5% of the total amount released or Substance is inorganic or is adsorbed to particles <10um in diameter when released
Environmental Persistence	t1/2 (days)	≤10	<10 to 50	<50 to 100	>100
Bio-accumulation	BCF Log k_{ow}	≤20 ≤2.0	>20 to 500 >2.0 to 4.0	>500 to 15000 >4.0 to 6.0	>15000 >6.0

ELEMENT NAME	ENDPOINT & UNITS	0	2	4	6	8	10
Acute Lethality	oral LD_{50} mg/kg	>5000	>500-5000	>50-500	>5-50	>0.5-5	≤0.5
	dermal LD_{50} mg/kg	>5000	>500-5000	>50-500	>5-50	>0.5-5	≤0.5
	inhal LD_{50} mg/m³	>15000	>1500-15000	>150-1500	>15-150	>1.5-15	≤1.5
	aquatic LC_{50} mg/L	>1000	>100-1000	>10-100	>1-10	>0.1-1	≤0.1
Sublethal Effects, Non-Mammals	aquatic EC_{50}, mg/L MATC, mg/L NOAEC,mg/L	≥20 ≥2 ≥0.2	2-<20 0.2-<2 0.02-<0.2	0.2-<2 0.02-<0.2 0.002-<0.02	0.02-<0.2 0.002-<0.02 0.0002-<0.002	<0.02* <0.002* <0.0002*	<0.02* <0.002* <0.0002*
	terrestrial subchronic NOEL mg/kg/d	≥1000	100-<1000	10-<100	1-<10	<1*	<1*
	chronic NOEL mg/kg/d	≥500	50-<500	5-<50	0.5-<5	<0.5* *in one genus	<0.5* *in different genera
Sublethal Effects, Plants	Water, mg/L Air, mg/m³ Soil, mg/kg						
	% Growth Reduction: ≤5 (=NOAEL) water air soil	 >10 >100 >100	 >1-10 >10-100 >10-100	 >0.1-1 >1-10 >1-10	 >0.01-0.1 >0.1-1 >0.1-1	 0.001-0.01 0.01-0.1 0.01-0.1	 <0.001 <0.01 <0.01
	>5-50(=EC_{50}) water air soil	 >100 >1000 >1000	 >10-100 >100-1000 >100-1000	 >1-10 >10-100 >10-100	 >0.1-1 >1-10 >1-10	 0.01-0.1 0.1-1 0.1-1	 <0.01 <0.1 <0.1
	>50 water air soil	 >1000 >10000 >10000	 >100-1000 >1000-10000 >1000-10000	 >10-100 >100-1000 >100-1000	 >1-10 >10-100 >10-100	 0.1-1 1-10 1-10	 <0.1 <1 <1
Sublethal Effects, Mammals*	oral NOEL mg/kg/day	>1000	>100-1000	>10-100	>1-10	>0.1-1	≤0.1
	inhal. NOEL mg/m³	>3000	>300-3000	>30-300	>3-30	>0.3-3	≤0.3
Teratogenicity	mg/kg/day	no terata, or terata only at >1000	terata or developmental anomalies at >50-1000	terata or developmental anomalies at >10-50	terata or developmental anomalies at >1-10	terata at >0.1-1, without overt maternal toxicity	terata at ≤0.1 without overt maternal toxicity
Genotoxicity /Mutagenicity	in vivo and in vitro cell assays	not genotoxic or mutagenic; negative results in vivo and in vitro	mutagenic in in vitro assays only; negative in vivo	mutagenic in prokaryotic cells only; negative results in eukaryotic cell assays	causes DNA induction or repair, with no direct interaction with nuclear material	causes clastogenic effects, sister chromatid exchange, crosslinks; no evidence of mutation	mutagenic in vivo (no negative results from in vivo assays)
Carcinogenicity	human and animal bioassay data	no tumours in adequate studies on at least two species, and does not interact with genetic material	tumours in only one animal species, negative results in others	causes benign tumours in more than one species, and does not interact with genetic material; promotor only; or causes cell transformation in vitro only (negative evidence in vivo)	tumourigenic in bioassays at doses causing metabolic enzyme saturation, or associated with lesions that predispose to tumours. No interaction with genetic material	indirect-acting carcinogen, no interaction with genetic material	direct-acting carcinogen that interacts with genetic material

* NOTE: The Sublethal Effects, Mammals criteria are based on studies of ≥90 days duration. If only shorter-term subchronic studies are available, the data are modified as follows, for scoring purposes:

Study duration 28-89 days - divide result by 10;
Study duration ≤28 days - divide result by 100.

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This table sets down a number of environmental and human health criteria, (under "Element Name"), then assigns an Endpoint and Unit (the type of test or data to be used), then scores each candidate substance a numerical value (0-10). The scores for each candidate, i.e. each substance subjected to the scoring matrix, are then ranked in numerical order.

In theory, the scoring system is an abstract schema, that can be used for different political purposes. It could be used, for example, to determine which substances should be investigated further, such as doing epidemiological studies, on the grounds that the highest scoring substances are likely to be most detrimental in practice. Or it could be used for regulatory purposes. In fact, the Ontario Ministry of the Environment in the early 1990s used the scoring system as a basis for the development of multimedia standards and water quality guidelines, for selecting chemicals of high priority for air emission control and for waste management (2). The policy purpose in question will determine where to draw the line under the score chart: for example, if there were only limited cash available, policy-makers might sponsor epidemiological studies on only the three top scorers over a certain volume of the substance in use or emitted. From the point of view of science, these lines are arbitrary. From the point of view of policy, they may be set pragmatically, eg how much environmental protection can we afford and how should we use our limited resources. Or: what is the tightest limit we can realistically set, which is compatible with the scoring results.

Less formal versions of this procedure can be found in "substitution analysis" for pollution prevention measures in the workplace: the toxicity profile of a substance in use is compared with the profile of a promising substitute. Only if there is a wide variation between the two will the substitute be adopted (3).

In the MOE system, certain decisions have to be made. For example, we need a decision procedure when there are no data for any given end-point. One option is to score 10, the maximum, on precautionary grounds, or on the contention that substances ought to have been tested for the end-point, prior to being put on the market. Uncertainties and non-scientific procedures there certainly are. But then there are uncertainties and scientifically arbitrary assumptions in risk assessment too. In risk assessment, we can adopt models and work on assumptions which maximize or which minimize risk (4). Both are equally legitimate from a scientific point of view: it depends on whether policy makers want to pursue a precautionary approach or whether they want to disturb chemical use-patterns as little as possible. No scientific evaluation is value-free.

Why Should Policy Makers Consult The Experts ?

In the debate so far, there is no need for policy-makers to rely on expertise outside the government. There are usually in-house resources enough to draft hazard assessment procedures and risk assessment protocols. But policy-makers will usually want advice from sources outside

the government, eg at the Committee stage of legislation. Among the sources will be scientific experts. One way of soliciting advice is simply to advertize hearings and ask the public to contribute. The Committee accepts written submissions from all parties, then selects a limited number to testify, on the basis of whose contribution would be the most valuable, in the short time available. This is the way things are done in Canada (5). Scientific experts are always included in environmental health deliberations, though rarely as the central or most numerous party.

One problem is that unless the experts are well-known to the public, it is difficult to identify experts whose contribution is likely to be highly valuable. If scientists always came under a banner like the Union of Concerned Scientists or Scientists Against Health Regulation Without Our Blessing, the Committee would at least know the direction of the advice it would likely receive. The Committee would know in advance that its proposals were either regarded as too little or far too much, always excessive. But this situation is relatively rare. Most of the time, there is a need to evaluate or grade experts as to their usefulness. Such an evaluation is provided by S. Robert Lichter and Stanley Hoffman in *Environmental Cancer - A Political Disease ?* (4), hereafter referred to as L&R with page numbers in square brackets, thus [L&R, l].

Their starting point is completely the opposite from the drift of this debate to date. L&R start from the implicit presumption - it is not asserted or argued - that for every area of health policy, there is an appropriate scientific discipline. Each discipline forms a natural constituency whose exclusive prerogative is to determine policy of which their discipline is the foundation. Policy

must be based on science. Thus "the unchallenged position of scientists as masters of their own political house has largely unravelled in the new political system in Washington." [L&R, 91]. The "relevant experts are largely ignored ..." [in the policy process; L&R 101]". We argue for the prudence of relying upon the consensus of the scientific community (as to the facts of the case, but not as to public values) in making policy decisions" [L&R, xii].

On the contrary, throughout L&R, there is no argument as to why it is prudent for policy-makers to rely on scientific experts, why scientists should be consulted at all. There is much discussion of the views of environmental activists versus those of scientists and how these parties are portrayed in the media. But the prudence of relying on experts is not argued; it is assumed. There is nothing in L&R in the spirit of the great Leibnitz: *calculemus* – let us reason together. There is no discussion either of the relationship between science and policy: the "public values" are to be those of scientists since they are the natural constituency to consult over policy issues.

That scientists as the proper arbiters of policy should be presumed without debate, comes from the splendid mutant notion of "science policy" [L&R, 91], a blatant conflation of distinct concepts that was demolished by Barry Commoner in a work, predictably, not referenced in L&R(6). Policy is about values while science is about facts and theories.

One central question addressed by L&R is to identify and describe "an expert consensus on cancer causation" [L&R,101]. It will then be axiomatic that this consensus will provide the rationale for public health measures aimed at the causes of cancer and thus their consequences in

society. A policy-maker who goes against the scientific consensus will realize measures that are ill-founded and therefore futile, ineffectual or socially disruptive and counter-productive.

L&R go about their project by sponsoring a survey of experts (1993). They polled 401 experts, randomly selected from the American Association for Cancer Research (AACR), who listed either carcinogenesis or epidemiology as their field of specialization. The aim was to determine expert opinion on the science of cancer causation. The experts were mainly academics who had published extensively in peer-reviewed journals; 92% said on the phone that they were currently involved in research on the causes or prevention of cancer [L&R, 102-103].

On the face of it, selecting epidemiologists and carcinogeneticists as the relevant experts for the survey was a sound idea, since these are likely to have the most relevant opinions on cancer causation. But when this move is explored, it is less convincing than it seems. Many of the controversies in epidemiology occur when a potential hazard is newly recognized, such as electromagnetic radiations and their effects on office workers, linemen and on children living near high-voltage electrical sources. The experts doing the research notice a connection between apparent effects and potential causes and a controversy erupts (7).Controversy is occasionally a dispute about statistical significance. More often, the lines are drawn between those who are convinced that "there is something there" and those who aren't. The controversy then centres on the methodology of the studies. Since there is no such thing as the perfect epidemiological study, *any* study can be faulted on methodological grounds and used as the occasion for discrediting apparently positive results. Since most epidemiologists do not in fact study health physics, most

will express the view that as a potential cause of cancer, electromagnetic radiations are dubious or a minor cause in the big picture.

All this can be quite bewildering to the educated layperson who has to evaluate the controversy in relation to public policy. Epidemiologists are a quarrelsome bunch. We can see this in the controversy over the pesticide Alar [L&R, Table 4.2, 107]. Most experts rated Alar low as a possible cause of cancer and as a minor concern. But *of course,* the effects of Alar are less certain than tobacco smoke and *of course* the risks to the public are smaller in size than tobacco. This is on L&R's assumption that risk analyses are the only scientific grounds for policy [L&R, x, 33, 36 ff, 39, 82]. L&R imply that Alar should not be taken seriously because, unlike activists and journalists, [L&R, Figure5.5, l6l], scientists rate it of low concern. This is like saying that while a big war is going on, no one should be concerned about little wars. But Alar is easy to fix: you just ban the pesticide, with the important proviso that it not be replaced by something just as bad [L&R, 77](8). If the experts then reply that the costs outweigh the benefits [L&R, back cover, quoting Bruce N. Ames], the response must be that the experts are no more experts on cost- benefit accounting than you or I or the Man in the Moon.

It gets worse. Reputable epidemiologists differ greatly as to the relationship between statistical associations and inferences of causality, with studies establishing a tenuous relationship between hazards and their supposed effects subject to scepticism as to whether they indicate anything at all as to causality (9). Epidemiologists who believe that such studies do not indicate a causal connection would disqualify themselves as informed commentators over most of those hazards which have attracted public controversy in recent years. The extreme case is to deny that epidemiology establishes causation at all, a position that is none the less philosophically defensible.

As with epidemiology, so with carcinogenesis. We should not expect an expert on metastasis to have an informed opinion on the causes of cancer. An expert who studies the mechanism whereby asbestos fibres in the lungs lead to cancerous tumours may not have an informed opinion on the relationship between the number of fibres inhaled to the incidence of lung cancer, estimates of which form the basis for workplace exposure limits. An expert on mutagenesis such as Bruce N. Ames may not be an expert on carcinogenesis. (10). Experts who say they work in the area of cancer prevention may confine their work to early detection and intervention (secondary prevention). If so, they are not experts on primary prevention, i.e. on how to prevent outbreaks of cancer in the first place.

When the L&R survey addresses particular policy issues, it is even more suspect. When asked whether the U.S. faced a cancer epidemic, 67% of experts said No and 31% said Yes [L&R, Figure 4.1,110]. Since an epidemic can consist of a handful of cases, the correct answer would have to be affirmative. The question is too vague to be useful. Besides, an expert on the epidemiology or carcinogenesis of radon may have nothing valuable to say about the patterns and the incidence of cancer generally.

Another question in the survey was " Should chemicals and additives be banned from food and drugs if they ever cause cancer in any species ?" To this question, 85% of the respondents said No. But this question is a straight policy question, the expert answer to which is no more or less pertinent than that of the educated layperson, in fact less so if it claimed that expertise in something or other gives their opinions special weight. Policy makers can choose any one of a

number of criteria for policy. "Carcinogen" can be defined in a number of ways, including evidence from structure-activity relationships, which now play a central part in chemical evaluation by the US federal government. Other governments use different criteria. Neither can be said to be scientifically right or wrong: the choice will depend on what policies you want to pursue and on how to make the best use of evaluation resources.

Again, the question: Are cancer-causing agents unsafe regardless of the dose ?" If this is a factual question, the 9% who said Don't Know gave the correct answer. For the vast majority of carcinogens, it is simply not known whether there is a threshold of harm. We cannot even construct a well-founded dose-response curve, never mind establishing a threshold of safety (11). When we look at the way that thresholds are actually used in the construction of policy, they appear not as statements of fact but as assumptions as to where we should draw the line over permitted exposures. They are rules of procedure, not facts. For instance, most health physicists work on the assumption that there is no lower threshold of harm from exposure to ionizing radiations. It is idle to ask whether it is true or false that there is a threshold.

Further, "Should human cancer risks be assessed by giving animals the maximum tolerable dose of a suspected cancer-causing agent ?" A reasonable answer is No (63%), but this does not tell us what "tolerable" means in scientific practice, nor is it evident that this bears any relation to the reality of the laboratory. If we did a 90-day animal test for chronic toxicity at the "maximum tolerable dose", we would expect all or most of the surviving test animals to die shortly after the

ninetieth day. That things are not done this way, can be verified by consulting the authoritative OECD Guidelines on Chemical Testing: "The highest dose level should be sufficiently high to elicit signs of minimal toxicity without substantially altering the normal life span due to effects other than tumours" (12).

Later, L&R state [Figure 5.3, 154] that 27% of experts polled, rejected the idea that we can base human cancer risks on animal tests; this is supposedly "based on" the AACR survey. But this is a supposition completely different from the question of tolerable doses given to test animals. By an amazing coincidence, the 27% of respondents who said that test animals should not be given the maximum tolerable dose is the same figure for those who (we are told) rejected the idea of basing human cancer risks on animal studies. The International Agency for Research on Cancer, a prestigious public body, does indeed classify carcinogens on the basis of animal test evidence, as to most other cancer experts (13). But policy makers do not have to infer human cancer risks from animal evidence. They can simply accord a lower degree of regulatory oversight to animal carcinogens than they do to known human carcinogens. The controversies over extrapolation of animal evidence to human cancer risks, [L&R, 256], then become irrelevant. L&R are forced to address the issue of extrapolation of animal test evidence to humans because of their axiom that the regulation of environmental health hazards must always be risk-based.

L&R are at their worst when they deal with scientists' ratings of levels of confidence in expertise on environmental cancer [L&R, Table 5.2, 162]. "Expertise on environmental cancer" can mean a whole range of different things. Bruce Ames is at the top of the list but (from his articles cited)

he knows nothing worth knowing about cancer epidemiology. If we wanted to know about the cancer epidemic in industrialized societies, we should certainly solicit the opinions of Richard Doll, Richard Peto and John Higginson. But we would not go to the late Irving Selikoff, who was an expert in asbestos-related cancers. Experts may have 100% confidence in Selikoff's opinions on *asbestos* and cancer, but none at all when it comes to his views, if he ever had any, on the relationship between *diet* and cancer. Similarly, scientists may have a high regard for Samuel Epstein's peer-reviewed articles but no confidence in his views on cancer policy. What we have here is a full-blooded blend of asking for subjective opinions about the wrong objects of scientific inquiry, a case of *ad hominem* and *ignoratio elenchi* in a single Fallacy of the Grand Slam. This is an intellectual accomplishment unique in the annals of the cancer wars. Science reduced to a popularity contest. A little course in Logic 101 would not go amiss. After all, L&R are professors of media and political science. We should not expect them to be experts in logical thinking.

Why then should policy-makers consult the experts at all ? After all, we do not expect a gifted carpenter to be an expert on housing policy. Nor are ace fighter pilots experts on air power. Where this has proved to be the case, as with Adolf Galland and Chuck Yaeger, this is arguably because they had experience of high command before they became civilian experts on policy, not because they had been brilliant fighter pilots with a well-deserved public reputation.

The answer to the question about experts is a simple one: experts are to be consulted whenever policy-makers consider their advice to be essential or useful.

Like carpenters and fighter pilots, scientific experts are capable of saying some exceedingly stupid things. Sir Richard Doll has contended that the children of Sellafield nuclear workers in the United Kingdom contracted leukemia because their over-clean homes made them susceptible to leukemia viruses.

He goes on. Since most cancers are caused by naturally occurring substances, the best thing to do is research on strategies whereby human systems can counter the action of these natural carcinogens (Doll and Ames). The body has natural defence mechanisms against synthetic chemicals, such as DNA repair, but evidently none against natural carcinogens which are allegedly thousands of times more important. That is why we need to redirect research. This is the sort of stuff we would expect from an imaginative high school student, not two of the world's most distinguished scientists.

A Case Study: Occupational Hazards [L&R, 68-72]

L&R begin by stating that the figures for cancers caused by work have been variously estimated as 6% of the total (Higginson & Muir) for men, 2% for women (Wynder and Gori) and 4% (Doll and Peto), with a lower limit of probability at 2% and a higher limit of 8%. Then came a federal government study in 1978, estimating that between 20% and 38% of cancers were occupational. "Long-simmering conflicts with management over workplace safety made labour leaders ready believers once the OSHA paper estimates were published" [L&R, 72].

As an historical perspective, this is dubious. Chronic diseases of work have been a concern of labour for centuries. The latest round of concern arose in the 1960s with the publication of *Work Can Be Dangerous to Your Health* in 1972 and *The Hazards of Work – How to Fight Them* in in 1973. *Chemicals, Work and Cancer* appeared in 1980 (14). This book downplayed the importance of epidemiology in constructing an occupational cancer policy and made only a passing reference to the OSHA paper as evidence of the seriousness of the workplace cancer epidemic.

In L&R's cosmos, labour leaders are a crowd of well-meaning duffers, always liable to be "hoodwinked" by the likes of Ralph Nader and "ready believers" when a major government study shows they had a problem on their hands which was bigger than they had ever imagined. The reality is that the labour movement employs its own experts, including risk analysts, epidemiologists, physicians and industrial hygienists, who are quite capable of evaluating studies such as the OSHA paper on its merits, giving corresponding policy advice to the leadership.

This historical image is not unique to L&Rs' treatment of occupational hazards. We repeatedly get the impression of scientists hammering on the doors of government to persuade policy-makers that their grounds for policy-making are false [L&R, 58; 94; 96 and n. 48 of Chapter 3; 113; 121]. The reality was that chemical manufacturers found a way of legitimizing their carcinogenic products through the scientific procedure of risk assessment and the adoption of thresholds in the form of "negligible risk" and "reasonable certainty of no harm" [L&R, 39].

The story of occupational carcinogenesis did not end there. The American Industrial Health Council (AHIC), an employers' group, hired Professor R. Stallones of the University of Texas to reevaluate the OSHA estimates. Stallones concluded that work-related cancers were of the order of 20%, at the lowest end of the OSHA figures, describing them as a "public health catastrophe" (15). Thus OSHA had not "exaggerated" the figures, nor was it guilty of a "confidence trick" [L&R, 71]; it had maximized them for policy purposes. Later Canadian studies put the occupational figure at 9%, well above the high end of the Doll and Peto estimates and more than double the mean (16). None of these developments are mentioned by L&R, though they are central to the occupational cancer controversy.

This one-sided view of the science pervades L&R's work: carcinogenic pesticides passed over lightly in favour of a past controversy over DDT [L&R, 15; 39]; the dismissal of pollution prevention as an environmental protection strategy [L&R, 37]; the failure to discuss the European middle way between socialist and capitalist approaches to environmental protection [L&R, 31-32]; downplaying the current asbestos-related cancer epidemic [L&R, 71; 180]; the supposition that nitrites are the only way to cure meat products [L&R, 77]; ignoring the evidence that trihalomethanes in drinking water cause cancer [L&R, 79 and 92]; the supposition that the only way to sterilize drinking water is by chlorination [L&R, 79 and 92]; and failing to cite sources that connect the medical administration of natural and synthetic hormones with cancer [L&R, 73-74].

The worst example is over the connection of dietary fats with cancer [L&R, 64] when in a space of two paragraphs, L&R move from saying that if certain scientific hypotheses were correct, fat would have to be regarded as a carcinogen - from this to the categorical statement that a low fat, high fibre diet will prevent up to 35% of all cancers. No mention is made of contrary theories, such as those concerning circumpolar societies, where we should expect those with a meat and fish diet, high in *uncontaminated* fats, to show high rates of cancer of the alimentary canal. All this is due to an obsessive desire to minimize the role of man-made chemicals in cancer causation, a truly scientific attitude.

Environmental Cancer – A Political Disease? contains many interesting observations and insights, particularly over the ideology of the environmental movement (there is no corresponding examination of the ideology of scientists). But on the rational interplay of science and policy, the book is worthless and intellectually dishonest. The book is shot through with the supposition that laypeople are incapable of evaluating the work of the experts. Judges (17) and juries routinely have to evaluate the testimony of experts. They cannot solve the problem by appealing from one expert to another. But if judges have to do this, as sure as hell, policy-makers can do the same. And so must we all if we are to avoid what George Bernard Shaw referred to as the conspiracy of every profession against the laity. Experts may not be the trained dogs in Einstein's critique but their value to society will be minimal if they present themselves as a mysterious elite, beyond the pale of the grand jury of society writ large. L&R comprise a modern example of "Aristotle hath said it", whereby all criticism is to be silenced by appeal to the authority of the master. Ames and Doll take the place of Aristotle, with lay commentators

such as L&R assuring us that whatever they say is true and wise. Give us a break.

References

1. *The Ontario Ministry of the Environment Scoring System for Assessing Environmental Contaminants,* Queen's Printer for Ontario, 1990, page 29.

2. *Opus supra,* Preamble, page ii.

3. For substitution analysis, see Rossi M., Ellenbecker M., and Geiser K., "Techniques in Toxics Use Reduction", *New Solutions,* 2.2, Fall, 1991. For a primitive attempt to construct a rating system for chemical pesticides, see Bennett D., "Pesticide Reduction, A Case Study from Canada",*New Solutions,* Fall 1991, pp 59-63. Hazard Assessment is a small but well-established scientific discipline in North America (though not, curiously, in Europe). See for instance, "Criteria to Identify Cremical Candidates for Sunsetting", by Foran, Jeffrey A. Ph.D., and Glenn Barbara A., MPH; *New Solutions,* Vol. 4, No 3, Spring 1994; "Comparative Evaluation of Chemical Ranking and Scoring Methodologies", CCPTC, University of Tennessee, 1994.

4. *Environmental Cancer - A Political Disease ?* by Lichter, S. Robert and Rothman, Stanley, Yale University Press, 1999, p 81.

5. The Chair of the House of Commons Standing Committee on Environment and Sustainable Development, the Hon. Charles Caccia MP, has a healthy scepticism about the testimony of all witnesses, whether they are experts, activists or businesspeople. This has forced organizations like the Canadian Labour Congress to be utterly rigorous in their arguments and proposals, which is surely the way that public debate ought to be conducted.

6. Commoner, Barry, *Making Peace With the Planet,* Pantheon, New York, 1990, pp 76-78. "The environmental failure has been very costly, not only in money but in burdening the still unresolved environmental crisis with a heavy heritage of poor public policy disguised as science and poor science disguised as policy" (Commoner, p 78).

7. See for example, Carstensen, Edwin L.,*Biological Effects of Transmission Line Fields,* Elsewier, New York 1987; *Health and Well-Being at VDU Work,* National Institute of Occupational Health, Solna, Sweden 1992; *New Evidence of Cancer from Electromagnetic Fields, NIOH, Solna, Sweden 1992;* "Intermittent 50 Hz Magnetic Field and Skin Tumour Promotion in SENCAR Mice, *Carcinogenesis* 15, 1994; plus the works listed in Note 9 below.

8. Substitutions are likely to be counter-productive unless they are done in the context of an overall materials policy. See Geiser K., *Materials Matter, Towards a Sustainable Materials Policy,* MIT Press, Cambridge, 2001, Ch 8, "Reconsidering Materials Policies," 8.4

"Detoxification".

9. For aspects of this controversy, see for instance, Rothman Kenneth J. ed., *Causal Inference,* Epidemiology Resources Inc., Chestnut Hill, Mass., 1988; Lillienfeld, Abraham E. M.D., M.P.H., D.Sc, and Lillienfeld David E., A.B., M.S. Eng., *Foundations of Epidemiology,* 2nd. Ed.O.U.P., New York 1980; Hernberg, Sven, M.D., "Epidemiology in Occupational Health" in *Developments in Occupational Medicine,* ed. Zenz, Carl M.D., D.Sc., Chicago-London, 1980, Ch. 1; Doll R., "Interpretation of Negative Epidemiological Evidence of Carcinogenicity", *IARC Scientific Publications #65,* Lyon, 1985, p 6. For the view that epidemiology does not establish causation at all, see the letter in the *New York Times* by Perl, Daniel, M.D., February 11, 1991.

10. The reason that L&R [Ch. 3, What Is Environmental Cancer ?] allow mutagenesis to slide into carcinogenesis is based on (i) the contention that most mutagens are cancer initiators and (ii) the presumption that the only test for mutagenicity is the Ames test. Neither is correct. Not all chemicals with mutagenic potential are cancer initiators, far from it. There are more than a dozen lab tests in genetic toxicology. Researchers typically select a battery of such tests and can use the results for a number of purposes, eg a regulatory requirement that a certain set of results will indicate further testing; or to determine the future direction of research eg in pharmacology; or to rule out some substances from further exploration in finding practical applications for the substance or product. In these ventures, it is not as if a test or any set of tests establish whether or not a particular substance is to be classified as a "mutagen". It is rather that "mutagenic potential" is measured in relation to an objective, such as one or more of those mentioned above. The formula "mutagen, therefore cancer initiator" is misleading and simplistic.

11. Roach S.A., and Rappaport S.M., "But They Are Not Thesholds: A Critical Analysis of the Documentation of Theshold Limit Values", *American Journal of Industrial Medicine,* 17 (1990), 727-753.

12. *OECD Guidelines for the Testing of Chemicals*, Paris 1993, updated regularly, Section 453, Combined Chronic Toxicity/Carcinogenicity Studies, Test Conditions - Dose Levels.

13. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans,* Vols. 1-69, IARC, Lyon, France, 1972-95).

14. Stellman J.M., PhD and Daum S.M., M.D., *Work is Dangerous to Your Health,* Vintage, New York, 1971; Kinnersly, P., *The Hazards of Work: How to Fight Them,* Pluto Press, London, 1973; LeServe A. PhD, Vose C. Ph.D, Wigley C. Ph.D, and Bennett D. PhD., *Chemicals, Work and Cancer,* Nelson, London 1980, pp 32-36.

15. Miller A.B., "Planning Cancer Control Strategies", *Chronic Diseases in Canada 13,* (No 1 Supplement, January-February 1992); Aronson K.J.et al., "Surveillance of Potential Associations Between Occupations and Causes of Death in Canada, 1965-1991, *Occupational and Environmental Medicine 56* (1995), 265-269.

16. Cited in Proctor R.N., *Cancer Wars,* Basic Books, New York 1995, p 67, and quoted in Firth M., Brophy J. and Keith M., *Workplace Roulette, Gambling With Cancer,* Between the Lines, Toronto, 1997, page 12.

17. Famously, Breyer S., *Breaking the Vicious Circle, Towards Effective Risk Regulation,* Harvard University Press, Cambridge, 1993.

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