THE SCIENCE OF RISK ASSESSMENT: IMPLICATIONS FOR FEDERAL REGULATION

HEARING

BEFORE THE

SUBCOMMITTEE ON ENERGY AND ENVIRONMENT

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Mr. EHLERS. Thank you, Dr. Gray.
Dr. Gold.

TESTIMONY OF LOIS GOLD, DIRECTOR, THE CARCINOGENIC POTENCY PROJECT, NATIONAL INSTITUTE OF ENVIRONMENTAL HEALTH SCIENCES, CENTER FOR ENVIRONMENTAL HEALTH SCIENCES, UNIVERSITY OF CALIFORNIA, BERKELEY, SENIOR SCIENTIST, LAWRENCE BERKELEY LABORATORY

Dr. GOLD. I am very pleased that the Committee is giving me an opportunity to speak today.

I'm going to discuss a bit more of the scientific background than the other three speakers based on my experience in doing cancer risk assessment over the past 20 years. I believe that we have some important lessons to learn about regulatory policy from cancer risk assessment because a series of misconceptions underlies current regulatory policy which was, as Dr. Carlo pointed out, based on a set of assumptions from the 1970's that have persisted in regulatory policy, in spite of new scientific information. And, like Dr. Omenn and Dr. Gray, I want to emphasize the importance of comparative risk assessment and, in my case, of setting priorities among a variety of risks.

Gee, we don't see too well on here but——

Mr. EHLERS. Yes, we're trying to get the lights on that end of the room turned off.

Dr. GOLD. My work has emphasized the importance——

Mr. EHLERS. Could you turn off the lights over here? There.

Dr. GOLD. My work has emphasized——

Mr. EHLERS. Thank you.

Dr. GOLD [continuing]. The importance of a broad perspective on cancer risk and I believe that the public, as well as risk assessment methodology, have a series of misconceptions about cancer risk and that these are driving a loop, essentially, of public fear, Congressional response, and agency response which confirms hypothetical risks and furthers public fear.

The idea that cancer rates are soaring is incorrect. Except for lung cancer, for which there is an epidemic, cancer rates have declined since the 1950's, about 15 percent overall, and life expectancy continues to increase.

Current regulatory policy is based on the idea that environmental synthetic chemicals are an important cause of human cancer. There is almost no evidence in humans to confirm such an assumption and, in fact, epidemiologic data gives us—and I won't—I hesitate to use these points—percentages after all that's been said but—within a range that's reasonable we can say about 35 percent of human cancer is due to cigarette smoking, about one-third is due to dietary imbalances which includes inadequate fruits and vegetables—and the public does not consume enough of these products and we have shown in 200 studies in epidemiologic work how important eating fruits and vegetables is, but only 20 percent of adolescents and one-third of adults eat 5 fruits and vegetables a day.

If we really wanted to prevent cancer in America, we would be doing research on why that's the case, what is it about the diet,

what is it about fruits and vegetables that are protective against cancer, rather than chasing after parts per billion of synthetic, industrial pollutants as causes of cancer.

A third misconception is that reducing pesticide residues is an effective way to prevent diet-related cancer. As I've said, fruits and vegetables are protective so the net impact is positive, not negative. In later discussion, I hope to describe to you some of the distorted estimates about pesticide residues which have driven policy based on hypothetical assumptions which are shown to be incorrect when residues are measured in the diet by FDA.

One of the bases of my work for the past 20 years in collaboration with Bruce Ames, also at UC-Berkeley, has been to gain a broad perspective on risk and here we have looked at the natural background of chemicals.

What most people don't realize is that 99.9 percent of the chemicals you're exposed to are naturally-occurring and because in the 1970's we had the idea that DDT was bad and therefore—although it did save millions of lives, hundreds of millions of lives—we came up with the idea that we would find a rare group of synthetic industrial chemicals that would be causing human cancer. We would test them in animals at very high doses so that we wouldn't miss any of these rare chemical carcinogens. And then, we would assume hypothetical estimates of exposure—which would be very conservative so that we would protect the public. But, all of these assumptions are wrong.

The hit rate on carcinogens in animal cancer tests is 60 percent when tested in rats and mice—60 percent of the chemicals tested are carcinogenic. The proportion that are carcinogens is the same between naturally-occurring chemicals in the diet and synthetic industrial chemicals. We in the United States don't test natural chemicals very much. The Japanese are much more concerned about testing them, for example, because they've had high rates of stomach cancer. But, the hit rate is the same and very high.

So, when we use this as a benchmark for tagging a synthetic chemical as a potential risk, we know that it isn't rare to find such an event and we know that we're tapping 0.01 percent or 0.1 percent of the chemicals that people are exposed to. So, we're missing the big picture here. Now, are naturally-occurring chemicals a hazard? I can say that of the chemicals identified in humans as carcinogens, two-thirds occur naturally. We would expect to find most things to be natural that are problematical because nearly everything you're exposed to is natural. So, this is a terrible gap in risk assessment.

I'm running out of time, I realize. I'll try and speed up. So, what is it we can learn from high dose animal cancer tests and how might they have led us astray? I think—the main thing I want to say is that: without ever conducting the study because of the way they're designed, I can tell you within a factor of 10 what the risk will be, what the one-in-a-million risk will be without ever doing the study. And this doesn't really change with the new EPA risk assessment guidelines which use a *"margin-of-exposure"* approach because I can give you—if you assume 1,000-fold safety factor, I can tell you from the dose given in the bioassay what the risk level will be.

So, we're not getting out of the problem by the new described risk assessment methodology. What we need is better science. Why does a chemical cause cancer, period. We have to look much further than the rodent bioassay. We're spending $3 million a chemical, one after another, to test these synthetic industrial pollutants.

I've given a list of naturally-occurring chemicals that occur at high amounts in the diet to NTP and they're going to consider testing them. But, there are a host of natural chemicals that we're paying little attention to and the one I just want to draw your attention to is medicinal herbs which are recommended at enormously high exposure levels. I've just looked into Quercetin which is a suspect rodent carcinogen and the amounts that are being recommended in pills per day are close to the dose that gave rodents cancer—on a milligram per kilogram body weight basis—and there's nobody out there "minding the store" about these chemicals at all.

I've gone over my time. I just want to draw your attention to the fact that I'd like an opportunity to discuss the HERP (Human Exposure Rodent Potency) table in my testimony at some point. It is a comparative risk ranking of chemicals that have been identified as rodent carcinogens. It is essentially a "margin-of-exposure" approach. It tells you what percent of the dose that gave a rat cancer does a human get on average in America, from various exposures. It includes workplace exposures, drug exposures, pollutants, and the natural background of chemicals in the diet.

They're vastly under-represented in this table because we don't test natural chemicals. We've tested—80 percent of the chemicals we've tested are synthetic, and 99.9 percent of the chemicals we're exposed to are natural. So, in spite of that, there's an enormous background of naturally-occurring chemicals in the diet that are rodent carcinogens that we get at very high doses compared to the carcinogenic dose in a rodent.

And, if you rank all of these—a one-in-a-million EPA risk would be something close to 0.0001 in this HERP table and you can see that there is a whole host of exposures that rank far above that, that we're not paying any attention to. Now, whether they're a real risk of cancer, I have no idea. Whether that's part of the problem in the diet, I don't know. I'd be much more likely to look for essential vitamins and minerals, the deficiency of those in being protective—in being carcinogenic, so that we might want to raise the levels of our RDA based on carcinogens. There's a whole area of research which has very little funding for doing that. So, I'll close my opening 5 minutes. Thank you.

[The prepared statement and attachments of Dr. Gold follow:]

Testimony, July 15, 1998
U.S. House of Representatives, Committee on Science
Lois Swirsky Gold and Bruce N. Ames

Dr. Lois Swirsky Gold is Director of the Carcinogenic Potency Project at the Environmental Health Sciences Center (NIEHS), University of California, Berkeley, and a Senior Scientist at the E.O Lawrence Berkeley National Laboratory. She has published 90 papers on the methodology of risk assessment, analyses of animal cancer tests, and the implications for cancer prevention and regulatory policy. Her Carcinogenic Potency Database (CPDB), published as a CRC handbook, analyzes the results of 5100 chronic, long-term cancer tests on 1300 chemicals. Dr. Gold's work has addressed many issues in the field of risk assessment: methodological issues such as validity problems associated with the use of limited data from animal cancer tests to estimate low-dose human cancer risks; reproducibility of results in near-replicate animal cancer tests; misconceptions about the causes of cancer, which underlie current regulatory policy; qualitative and quantitative extrapolation between species; target organs of carcinogenesis; ranking possible carcinogenic hazards of naturally-occurring and synthetic chemicals; and statistical issues in risk estimation. Dr. Gold has served on the Panel of Expert Reviewers for the National Toxicology Program, and the Board of the Harvard Center for Risk Analysis. She has been a member of the Harvard Risk Management Group and the Board of Directors of the Annapolis Center. [lois@potency.berkeley.edu University of California, Berkeley CA 94720-3202, (510) 486-7080 http://potency.berkeley.edu/cpdb.html]

Dr. Bruce N. Ames is a Professor of Biochemistry and Molecular Biology and Director of the Environmental Health Sciences Center (NIEHS), University of California. He is a member of the National Academy of Sciences and was on their Commission on Life Sciences. He was a member of the National Cancer Advisory Board of the National Cancer Institute (1976-82). His many awards include: the General Motors Cancer Research Foundation Prize (1983), the Tyler Prize for environmental achievement (1985) the Honda Foundation Prize for Ecotoxicology (1996) and the Japan Prize (1997). bnames@uclink4.berkeley.edu (510) 642-5165

Cancer risk assessments have been the basis of many regulatory decisions involving low-dose human exposures to synthetic chemicals. Our analyses during the past 20 years of the causes of cancer, animal cancer tests, and risk assessment methodology, underscore the importance of setting priorities in efforts to prevent human cancer, and of taking a broad perspective on risk, which includes epidemiological studies and examines that natural background of chemicals to which humans are exposed. The experience of cancer risk assessment since the 1970's offers valuable lessons for future regulatory policy. Our analyses indicate that various misconceptions about the relationship between environmental pollution and human disease, particularly cancer, drive regulatory policy.

1. **Misconception: Cancer rates are soaring.**

Findings: There is no epidemic of cancer, except for lung cancer due to smoking. Cancer mortality rates have declined 16% since 1950 (excluding lung cancer).

2. **Misconception: Environmental synthetic chemicals are an important cause of human cancer.**

Findings: Neither epidemiology nor toxicology support this idea. The major causes of cancer are a) Smoking: About a third of U.S. cancer (90% of lung cancer); b) Dietary imbalances, e.g., lack of dietary fruits & vegetables: The quarter of the population eating the least fruits & vegetables has double the cancer rate for most types of cancer compared to the quarter eating the most; c) Chronic infections: mostly in developing countries; d) Hormonal factors: primarily influenced by life style.

3. **Misconception: Reducing pesticide residues is an effective way to prevent diet-related cancer.**

Findings: On the contrary, high consumption of fruits and vegetables is associated with lowered risk of cancer. But 80% of U.S. children and adolescents and 68% of adults did not meet the recommended intake of 5 a day. Half the public does not answer that fruits and vegetables are protective, when asked about the causes of cancer. If reduction of pesticide use results in higher costs of produce, then consumption will decline and cancer rates may rise.

4. **Misconception: Human exposures to carcinogens and other potential hazards are primarily to synthetic chemicals.**

Contrary to common perception, 99.9% of the chemicals humans ingest are natural. Plants produce natural pesticides to defend themselves, and Americans eat about 5,000 to 10,000 different natural pesticides, about 1,500 mg per person per day, which is about 10,000 times more than synthetic pesticide residues. Cooking food produces about 2000 mg of burnt material in food per day, compared to about 0.09 mg of pesticide residues consumed daily.

5. **Misconception: Cancer risks to humans can be assessed by standard high-dose animal cancer tests.**

Findings: Contrary to assumptions of the 1970's, chemicals that test positive in animal cancer tests are not rare. Half of all chemicals tested, whether occurring naturally or produced synthetically, are "carcinogens" Forty percent of the carcinogens are not mutagenic.

There are high-dose effects in rodent cancer tests that are not relevant to low-dose human exposures and which contribute to the high proportion of chemicals that test positive. Without additional data on mechanism of carcinogenesis for each chemical, the interpretation of a positive result in a rodent bioassay is highly uncertain; the effects may be limited to the high dose tested due to increased cell division caused by the high doses.

- Over 1000 chemicals have been described in coffee: 28 have been tested and 19 are rodent carcinogens.
- Although natural chemicals have not been a focus of cancer testing, of the 63 natural plant pesticides that have been tested, 35 are rodent carcinogens.
- Half the drugs in the Physician's Desk Reference (PDR) that report cancer test results, are positive.

Data from standard rodent bioassays is limited, and potency estimates are constrained to a narrow range about the high dose tested. Because of this constraint, the regulatory virtually safe dose at 1 in a million risk, can be estimated from the high dose tested without ever conducting the cancer test. Similarly, the benchmark dose risk proposed in the new EPA cancer guidelines can also be estimated from the high dose, whether the presumed model is linear or based on a safety factor approach assuming a threshold.

6. Misconception: Synthetic chemicals pose greater carcinogenic hazards than natural chemicals.

Gaining a broad perspective about the vast number of chemicals to which humans are exposed and comparing potential risks, is essential to setting research and regulatory priorities. We have used the strategy of ranking on a rough index of possible hazard from a wide variety of chemical exposures at levels that humans typically receive. Ranking is a critical first step for setting priorities for selecting chemicals for chronic bioassay or mechanistic studies, for epidemiological research, and for regulatory policy. Although one cannot say whether the ranked chemical exposures are likely to be of major or minor importance in human cancer, it is not prudent to focus attention on the possible hazards at the bottom off a ranking if, using the same methodology to identify hazard, there are numerous common human exposures with much greater possible hazards. Our analyses are based on the HERP index (Human Exposure/Rodent Potency), which indicates what percentage of the rodent carcinogenic potency (TD50 in mg/kg/day) a human receives from a given daily lifetime exposure (mg/kg/day) (SEE TABLE) A ranking based on standard regulatory risk assessment would be similar.

Overall, our analyses have shown that HERP values for some historically high exposures in the workplace and some pharmaceuticals rank high, and that there is an enormous background of naturally occurring rodent carcinogens in typical portions of common foods that cast doubt on the relative importance of low-dose exposures to residues of synthetic chemicals such as pesticides. The possible carcinogenic hazards from synthetic pesticides (at average measured exposures) are minimal compared to the background of nature's pesticides. Many ordinary foods would not pass the regulatory criteria used for synthetic chemicals, and no diet can be free of rodent carcinogens that are natural chemicals. For many natural chemicals the HERP values are in the top half of the table, even though natural chemicals are markedly underrepresented because so few have been tested in rodent bioassays. Caution is necessary in drawing conclusions from the occurrence in the diet of natural chemicals that are rodent carcinogens. It is not argued here that these dietary exposures are necessarily of much relevance to human cancer. Our results call for a re-evaluation of the utility of animal cancer tests in protecting the public against minor hypothetical risks.

7. Misconception: Regulating low, hypothetical risks advances public health.

There is no convincing evidence that synthetic chemical pollutants are important for human cancer. Regulations that try to eliminate minuscule levels of synthetic chemicals are enormously expensive: EPA has estimated that environmental regulations cost society $140 billion/year. It has been estimated that the median toxic control program costs 146 times more per life year saved than the median medical intervention.

Prevention of cancer will come from knowledge obtained from biomedical research, education of the public, and from lifestyle changes by individuals. A re-examination of priorities in cancer prevention, both public and private, seems called for.

Ranking Possible Carcinogenic Hazards from Average U.S. Exposures. [Chemicals that occur naturally in foods are in bold.] *Daily human exposure:* Reasonable daily intakes are used to facilitate comparisons. The calculations assume a daily dose for a lifetime. *Possible hazard:* The human dose of rodent carcinogen is divided by 70 kg to give a mg/kg/day of human exposure, and this dose is given as the percentage of the TD_{50} in the rodent (mg/kg/day) to calculate the *H*uman *E*xposure/*R*odent *P*otency index (HERP). TD_{50} values used in the HERP calculation are averages calculated by taking the harmonic mean of the TD_{50}s of the positive tests in that species from the Carcinogenic Potency Database. Average TD_{50} values, have been calculated separately for rats and mice, and the more potent value is used for calculating possible hazard.

Possible hazard: HERP (%)	Average daily US exposure	Human dose of rodent carcinogen	Potency TD_{50} (mg/kg/day)[a] Rats	Mice
140	EDB: workers (high exposure) (before 1977)	Ethylene dibromide, 150 mg	1.52	(7.45)
17	Clofibrate	Clofibrate, 2 g	169	
14	Phenobarbital, 1 sleeping pill	Phenobarbital, 60 mg	(+)	6.09
6.8	1,3-Butadiene: rubber workers (1978-86)	1,3-Butadiene, 66.0 mg	(261)	13.9
6.1	Tetrachloroethylene: dry cleaners with dry-to-dry units (1980-90)[b]	Tetrachloroethylene, 433 mg	101	(126)
4.0	Formaldehyde: workers	Formaldehyde, 6.1 mg	2.19	(43.9)
2.1	Beer, 257 g	Ethyl alcohol, 13.1 ml	9110	(–)
1.4	Mobile home air (14 hours/day)	Formaldehyde, 2.2 mg	2.19	(43.9)
0.9	Methylene chloride: workers (1940s-80s)	Methylene chloride, 471 mg	724	(918)
0.5	Wine, 28.0 g	Ethyl alcohol, 3.36 ml	9110	(–)
0.4	Conventional home air (14 hours/day)	Formaldehyde, 598 µg	2.19	(43.9)
0.1	Coffee, 13.3 g	Caffeic acid, 23.9 mg	297	(4900)
0.04	Lettuce, 14.9 g	Caffeic acid, 7.90 mg	297	(4900)
0.03	Safrole in spices	Safrole, 1.2 mg	(441)	51.3
0.03	Orange juice, 138 g	d-Limonene, 4.28 mg	204	(–)
0.03	Pepper, black, 446 mg	d-Limonene, 3.57 mg	204	(–)
0.02	Mushroom (Agaricus bisporus 2.55 g)	Mixture of hydrazines, etc. (whole mushroom)	–	20,300
0.02	Apple, 32.0 g	Caffeic acid, 3.40 mg	297	(4900)
0.02	Coffee, 13.3 g	Catechol, 1.33 mg	118	(244)
0.02	Coffee, 13.3 g	Furfural, 2.09 mg	(683)	197
0.009	BHA: daily US avg (1975)	BHA, 4.6 mg	745	(5530)
0.008	Beer (before 1979), 257 g	Dimethylnitrosamine, 726 ng	0.124	(0.189)
0.008	Aflatoxin: daily US avg (1984-89)	Aflatoxin, 18 ng	0.0032	(+)
0.007	Cinnamon, 21.9 mg	Coumarin, 65.0 µg	13.9	(103)
0.006	Coffee, 13.3 g	Hydroquinone, 333 µg	82.8	(225)
0.005	Saccharin: daily US avg (1977)	Saccharin, 7 mg	2140	(–)
0.005	Carrot, 12.1 g	Aniline, 624 µg	194[c]	(–)
0.004	Potato, 54.9 g	Caffeic acid, 867 µg	297	(4900)
0.004	Celery, 7.95 g	Caffeic acid, 858 µg	297	(4900)
0.004	White bread, 67.6 g	Furfural, 500 µg	(683)	197
0.003	Nutmeg, 27.4 mg	d-Limonene, 466 µg	204	(–)
0.003	Conventional home air (14 hour/day)	Benzene 155 µg	(169)	77.5
0.002	Carrot, 12.1 g	Caffeic acid, 374 µg	297	(4900)
0.002	Ethylene thiourea: daily US avg (1990)	Ethylene thiourea, 9.51 µg	7.9	(23.5)
0.002	[DDT: daily US avg (before 1972 ban)]	[DDT, 13.8 µg]	(84.7)	12.3
0.001	Plum, 2.00 g	Caffeic acid, 276 µg	297	(4900)
0.001	BHA: daily US avg (1987)	BHA, 700 µg	745	(5530)
0.001	Pear, 3.29 g	Caffeic acid, 240 µg	297	(4900)

Possible hazard: HERP (%)	Average daily US exposure	Human dose of rodent carcinogen	Potency TD_{50} (mg/kg/day)[a] Rats	Mice
0.001	[UDMH: daily US avg (1988)]	[UDMH, 2.82 µg (from Alar)]	(–)	3.96
0.0009	Brown mustard, 68.4 mg	Allyl isothiocyanate, 62.9 µg	96	(–)
0.0008	[DDE: daily US avg (before 1972 ban)]	[DDE, 6.91 µg]	(–)	12.5
0.0007	TCDD: daily US avg (1994)	TCDD, 12.0 pg	0.0000235	(0.000156)
0.0007	Bacon, 11.5 g	Diethylnitrosamine, 11.5 ng	0.0237	(+)
0.0006	Mushroom (Agaricus bisporus 2.55 g)	Glutamyl-p-hydrazino-benzoate, 107 µg		277
0.0005	Jasmine tea, 2.19 g	Benzyl acetate, 504 µg	(–)	1440
0.0004	Bacon, 11.5 g	N-Nitrosopyrrolidine, 196 ng	(0.799)	0.679
0.0004	Bacon, 11.5 g	Dimethylnitrosamine, 34.5 ng	0.124	(0.189)
0.0004	[EDB: Daily US avg (before 1984 ban)]	[EDB, 420 ng]	1.52	(7.45)
0.0004	Tap water, 1 liter (1987-92)	Bromodichloromethane, 13 µg	(72.5)	47.7
0.0003	Mango, 1.22 g	d-Limonene, 48.8 µg	204	(–)
0.0003	Beer, 257 g	Furfural, 39.9 µg	(683)	197
0.0003	Tap water, 1 liter (1987-92)	Chloroform, 17 µg	(262)	90.3
0.0003	Carbaryl: daily US avg (1990)	Carbaryl, 2.6 µg	14.1	(–)
0.0002	Celery, 7.95 g	8-Methoxypsoralen, 4.86 µg	32.4	(–)
0.0002	Toxaphene: daily US avg (1990)	Toxaphene, 595 ng	(–)	5.57
0.00009	Mushroom (Agaricus bisporus, 2.55 g)	p-Hydrazinobenzoate, 28 µg		454[c]
0.00008	PCBs: daily US avg (1984-86)	PCBs, 98 ng	1.74	(9.58)
0.00008	DDE/DDT: daily US avg (1990)	DDE, 659 ng	(–)	12.5
0.00007	Parsnip, 54.0 mg	8-Methoxypsoralen, 1.57 µg	32.4	(–)
0.00007	Toast, 67.6 g	Urethane, 811 ng	(41.3)	16.9
0.00006	Hamburger, pan fried, 85 g	PhIP, 176 ng	4.29[c]	(28.6[c])
0.00005	Estragole in spices	Estragole, 1.99 µg		51.8
0.00005	Parsley, fresh, 324 mg	8-Methoxypsoralen, 1.17 µg	32.4	(–)
0.00003	Hamburger, pan fried, 85 g	MeIQx, 38.1 ng	1.99	(24.3)
0.00002	Dicofol: daily US avg (1990)	Dicofol, 54[c] ng	(–)	32.9
0.00001	Beer, 257 g	Urethane, 115 ng	(41.3)	16.9
0.000005	Hamburger, pan fried, 85 g	IQ, 6.38 ng	1.89[c]	(19.6)
0.000001	Lindane: daily US avg (1990)	Lindane, 32 ng	(–)	30.7
0.0000007	Pineapple, 5.75 g	Ethyl acrylate, 57.5 ng	120	(324)
0.0000004	PCNB: daily US avg (1990)	PCNB (Quintozene), 19.2 ng	(–)	71.1
0.0000001	Chlorobenzilate: daily US avg (1989)	Chlorobenzilate, 6.4 ng	(–)	93.9
<0.00000001	Chlorothalonil: daily US avg (1990)	Chlorothalonil, <6.4 ng	828[d]	(–)
0.000000008	Folpet: daily US avg (1990)	Folpet, 12.8 ng		2280[d]
0.000000006	Captan: daily US avg (1990)	Captan, 11.5 ng	2690[d]	(2730[d])

[a]"..." = no data in CPDB; a number in parentheses indicates a TD_{50} value not used in the HERP calculation because TD_{50} is less potent than in the other species. (–) = negative in cancer test; (+) = positive cancer test(s) not suitable for calculating a TD_{50}.

[b]This is not an average, but a reasonably large sample (1027 workers).

[c]TD_{50} harmonic mean was estimated for the base chemical from the hydrochloride salt.

[d]Additional data from the EPA that is not in the CPDB were used to calculate these TD_{50} harmonic means.

Attachment 1

Misconception: Cancer rates are soaring.

Misconception: Environmental synthetic chemicals are an important cause of human cancer.

Misconception: Reducing pesticide residues is an effective way to prevent diet-related cancer.

Misconception: Human exposures to carcinogens and other potential hazards are primarily to synthetic chemicals.

Misconception: Cancer risks to humans can be assessed by standard high-dose animal cancer tests.

Misconception: Synthetic chemicals pose greater carcinogenic hazards than natural chemicals.

Misconception: Regulating low, hypothetical risks advances public health.

Attachment 2

Ranking Possible Carcinogenic Hazards from Average U.S. Exposures. [Chemicals that occur naturally in foods are in bold.] *Daily human exposure*: Reasonable daily intakes are used to facilitate comparisons. The calculations assume a daily dose for a lifetime. *Possible hazard:* The human dose of rodent carcinogen is divided by 70 kg to give a mg/kg/day of human exposure, and this dose is given as the percentage of the TD_{50} in the rodent (mg/kg/day) to calculate the *H*uman *E*xposure/*R*odent *P*otency index (HERP). TD_{50} values used in the HERP calculation are averages calculated by taking the harmonic mean of the TD_{50}s of the positive tests in that species from the Carcinogenic Potency Database. Average TD_{50} values, have been calculated separately for rats and mice, and the more potent value is used for calculating possible hazard.

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0.03	Safrole in spices	Safrole, 3.2 mg	(441)	51.3
0.03	Orange juice, 138 g	d-Limonene, 4.28 mg	204	(–)
0.03	Pepper, black, 446 mg	d-Limonene, 3.57 mg	204	(–)
0.02	Mushroom (Agaricus bisporus 2.55 g)	Mixture of hydrazines, etc. (whole mushroom)	–	20,300
0.02	Apple, 32.0 g	Caffeic acid, 3.40 mg	297	(4900)
0.02	Coffee, 13.3 g	Catechol, 1.33 mg	118	(244)
0.02	Coffee, 13.3 g	Furfural, 2.09 mg	(683)	197
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0.008	Aflatoxin: daily US avg (1984-89)	Aflatoxin, 18 ng	0.0032	(+)
0.007	Cinnamon, 21.9 mg	Coumarin, 65.0 µg	13.9	(103)
0.006	Coffee, 13.3 g	Hydroquinone, 333 µg	82.8	(225)
0.005	Saccharin: daily US avg (1977)	Saccharin, 7 mg	2140	(–)
0.005	Carrot, 12.1 g	Aniline, 624 µg	194[c]	(–)
0.004	Potato, 54.9 g	Caffeic acid, 867 µg	297	(4900)
0.004	Celery, 7.95 g	Caffeic acid, 858 µg	297	(4900)
0.004	White bread, 67.6 g	Furfural, 500 µg	(683)	197
0.003	Nutmeg, 27.4 mg	d-Limonene, 466 µg	204	(–)
0.003	Conventional home air (14 hour/day)	Benzene, 155 µg	(169)	77.5
0.002	Carrot, 12.1 g	Caffeic acid, 374 µg	297	(4900)
0.002	Ethylene thiourea: daily US avg (1990)	Ethylene thiourea, 9.51 µg	7.9	(23.5)
0.002	[DDT: daily US avg (before 1972 ban)]	[DDT, 13.8 µg]	(84.7)	12.3
0.001	Plum, 2.00 g	Caffeic acid, 276 µg	297	(4900)
0.001	BHA: daily US avg (1987)	BHA, 700 µg	745	(5530)
0.001	Pear, 3.29 g	Caffeic acid, 240 µg	297	(4900)

Possible hazard: HERP (%)	Average daily US exposure	Human dose of rodent carcinogen	Potency TD_{50} (mg/kg/day)[a] Rats	Mice
0.001	[UDMH: daily US avg (1988)]	[UDMH, 2.82 µg (from Alar)]	(–)	3.96
0.0009	Brown mustard, 68.4 mg	Allyl isothiocyanate, 62.9 µg	96	(–)
0.0008	[DDE: daily US avg (before 1972 ban)]	[DDE, 6.91 µg]	(–)	12.5
0.0007	TCDD: daily US avg (1994)	TCDD, 12.0 pg	0.0000235	(0.000156)
0.0007	Bacon, 11.5 g	Diethylnitrosamine, 11.5 ng	0.0237	(+)
0.0006	Mushroom (Agaricus bisporus 2.55 g)	Glutamyl-p-hydrazino-benzoate, 107 µg	.	277
0.0005	Jasmine tea, 2.19 g	Benzyl acetate, 504 µg	(–)	1440
0.0004	Bacon, 11.5 g	N-Nitrosopyrrolidine, 196 ng	(0.799)	0.679
0.0004	Bacon, 11.5 g	Dimethylnitrosamine, 34.5 ng	0.124	(0.189)
0.0004	[EDB: Daily US avg (before 1984 ban)]	[EDB, 420 ng]	1.52	(7.45)
0.0004	Tap water, 1 liter (1987-92)	Bromodichloromethane, 13 µg	(72.5)	47.7
0.0003	Mango, 1.22 g	d-Limonene, 48.8 µg	204	(–)
0.0003	Beer, 257 g	Furfural, 39.9 µg	(683)	197
0.0003	Tap water, 1 liter (1987-92)	Chloroform, 17 µg	(262)	90.3
0.0003	Carbaryl: daily US avg (1990)	Carbaryl, 2.6 µg	14.1	(–)
0.0002	Celery, 7.95 g	8-Methoxypsoralen, 4.86 µg	32.4	(–)
0.0002	Toxaphene: daily US avg (1990)	Toxaphene, 595 ng	(–)	5.57
0.00009	Mushroom (Agaricus bisporus, 2.55 g)	p-Hydrazinobenzoate, 28 µg	.	454[c]
0.00008	PCBs: daily US avg (1984-86)	PCBs, 98 ng	1.74	(9.58)
0.00008	DDE/DDT: daily US avg (1990)	DDE, 659 ng	(–)	12.5
0.00007	Parsnip, 54.0 mg	8-Methoxypsoralen, 1.57 µg	32.4	(–)
0.00007	Toast, 67.6 g	Urethane, 811 ng	(41.3)	16.9
0.00006	Hamburger, pan fried, 85 g	PhIP, 176 ng	4.29[c]	(28.6[c])
0.00005	Estragole in spices	Estragole, 1.99 µg	.	51.8
0.00005	Parsley, fresh, 324 mg	8-Methoxypsoralen, 1.17 µg	32.4	(–)
0.00003	Hamburger, pan fried, 85 g	MeIQx, 38.1 ng	1.99	(24.3)
0.00002	Dicofol: daily US avg (1990)	Dicofol, 544 ng	(–)	32.9
0.00001	Beer, 257 g	Urethane, 115 ng	(41.3)	16.9
0.000005	Hamburger, pan fried, 85 g	IQ, 6.38 ng	1.89[c]	(19.6)
0.000001	Lindane: daily US avg (1990)	Lindane, 32 ng	(–)	30.7
0.0000007	Pineapple, 5.75 g	Ethyl acrylate, 57.5 ng	120	(324)
0.0000004	PCNB: daily US avg (1990)	PCNB (Quintozene), 19.2 ng	(–)	71.1
0.0000001	Chlorobenzilate: daily US avg (1989)	Chlorobenzilate, 6.4 ng	(–)	93.9
<0.00000001	Chlorothalonil: daily US avg (1990)	Chlorothalonil, <6.4 ng	828[d]	(–)
0.000000008	Folpet: daily US avg (1990)	Folpet, 12.8 ng	.	2280[d]
0.0000000006	Captan: daily US avg (1990)	Captan, 11.5 ng	2690[d]	(2730[d])

[a]"." = no data in CPDB; a number in parentheses indicates a TD_{50} value not used in the HERP calculation because TD_{50} is less potent than in the other species. (–) = negative in cancer test; (+) = positive cancer test(s) not suitable for calculating a TD_{50}.

[b]This is not an average, but a reasonably large sample (1027 workers).

[c]TD_{50} harmonic mean was estimated for the base chemical from the hydrochloride salt.

[d]Additional data from the EPA that is not in the CPDB were used to calculate these TD_{50} harmonic means.

Attachment 3

Comparison of Potency and Exposure Measures

Pesticides Included in the TDS (FDA)	Ratio of Potency: Recalculated q_1^*/ ($\ln(2)/TD_{50}$)	Ratio of Exposure: EPA/FDA
Azinphosmethyl$^{D(E)}$	6.1	369
Permethrin^{C_q}	1.5	411
Acephate$^{NA(C_{nq})}$	0.7	552
Linuron$^{C_q(C_{nq})}$	2.5	3,880
Parathion$^{C_q(C_{nq})}$	2.6	11,700
Chlorothalonil$^{NA(B_2)}$	1.9	>99,100
Captan^{B_2}	1.7	114,000
Folpet^{B_2}	0.8	463,000
Alachlor^{B_2}	0.9	—[a]
Captafol^{B_2}	1.2	—
Cypermethrin$^{C_q(C_{nq})}$	2.2	—
Oxadiazon$^{B_2(C_q)}$	3.0	—
Pesticides Not Measured in the TDS (FDA)		
Asulam$^{NA(C_{nq})}$	2.5	NA[b]
Benomyl^{C_q}	2.2	NA
Chlordimeform^{B_2}	1.7	NA
Fosetyl Al$^{C_q(C_{nq})}$	1.8	NA
Glyphosate$^{C_q(E)}$	2.5	NA
Metolachlor$^{C_q(C_{nq})}$	1.8	NA
Oryzalin^{C_q}	2.5	NA

[a]FDA did not detect any residues, therefore no ratio could be calculated.

[b]Not applicable because not measured by FDA.

Dr. Lois Swirsky Gold
Biography

Dr. Lois Swirsky Gold is Director of the Carcinogenic Potency Project at the Environmental Health Sciences Center (NIEHS), University of California, Berkeley, and a Senior Scientist at the E.O. Lawrence Berkeley National Laboratory. She has published 90 papers on the methodology of risk assessment, analyses of animal cancer tests, and the implications for cancer prevention and regulatory policy. Her Carcinogenic Potency Database (CPDB), published as a CRC handbook, analyzes the results of 5400 chronic, long-term cancer tests on 1300 chemicals. Dr. Gold's work has addressed many issues in the field of risk assessment: methodological issues such as validity problems associated with the use of limited data from animal cancer tests to estimate low-dose human cancer risks; reproducibility of results in near-replicate animal cancer tests; misconceptions about the causes of cancer, which underlie current regulatory policy; qualitative and quantitative extrapolation between species; target organs of carcinogenesis; ranking possible carcinogenic hazards of naturally-occurring and synthetic chemicals; and statistical issues in risk estimation. Dr. Gold has served on the Panel of Expert Reviewers for the National Toxicology Program, and the Board of the Harvard Center for Risk Analysis. She has been a member of the Harvard Risk Management Group and the Board of Directors of the Annapolis Center.

TABLE OF CARCINOGENIC POTENCY

Mr. EHLERS. Thank you and I think you have also just had the time to explain your HERP chart.

Just one quick question on that—and, incidentally, just to explain the rules in case you're not familiar with them, you were each given roughly 5 minutes to make your opening statement, we will ask questions 5 minutes per Member of Congress here and it appears there are two here so we'll oscillate back and forth.

Just a quick question on this HERP table. I had read several times over the past decade that Bruce Ames did this work showing that peanut butter was appreciably carcinogenic and a quick glance didn't show it on this chart. Is it—did I miss it or is it not on there?

Dr. GOLD. It's not.

Mr. EHLERS. Or is it so small—is it smaller than the others——

Dr. GOLD. No, we subsumed it under Aflatoxin in this table because——

Mr. EHLERS. Oh.

Dr. GOLD. We tried to give average daily exposures. Here, we have a HERP of 0.008 for Aflatoxin exposure. It's primarily in peanut butter and certain corn products. On the other hand, we have good human data on Aflatoxin and the potency in humans is about ten-fold lower than it is in rats and so we would—this would actually be coming down if it were a human risk-based risk assessment.

Mr. EHLERS. And how do you get good human data?

Dr. GOLD. On Aflatoxin, not in this country——

Mr. EHLERS. Well, on anything——

Dr. GOLD. Because the amounts we're exposed to were so low. But, in Asia, where people get much higher doses we can look at the risk of liver cancer, and we also know that there's a synergism between Hepatitis B virus—which is very common in the Third World—and Aflatoxin intake, and that this multiplies the effect on liver cancer.

S. 981—THE REGULATORY IMPROVEMENT ACT OF 1997

Mr. EHLERS. All right. Thank you.

I have one general question for all of you to start things off. We've heard several comments here about the Levin-Thompson bill and most seem favorable to the bill but also I saw in the written testimony some reservations about it. I would be interested in the reactions of each of you to that bill as it stands now and ways in which you would recommend improving it. If there's not sufficient time for you to give your answer here, we'd be happy to have your answers in writing because, presumably, we will—if that bill passes the Senate, we will have to deal with it in the House. So, we'll go right down the line. Dr. Omenn, you had a key role in generating that bill.

Dr. OMENN. Yes, well, I've had—and Gail Charnley who is here—Dr. Gail Charnley who is the Executive Director of the Commission. We spent a lot of time with the Committee staff and we're pleased to see the evolution of S. 981. I do support it in its present version. I do think it could be somewhat further focussed and improved. The short version of an answer to your question is the submission included in the Senate report from Franklin Raines, OMB Director, contains several specific changes in the language that would make much more focused the judicial review, the peer review process, and the burden on OMB and OSTP to generate an extensive review that somehow covers all the diverse kinds of risks across the many agencies.

There's also the Minority report that suggests we ought to make crystal clear that environmental health and safety has boundaries around it and regulations—like regulations to protect civil rights which have been raised by the Minority clearly outside. I'm sure that's the intent of the bill.

Mr. EHLERS. All right, thank you. Dr. Carlo.

Mr. CARLO. We're generally supportive of the bill as a first step. However, the problem from my perspective is that the secondary and tertiary prevention aspect—secondary prevention being, you know, screening aimed at early diagnosis of problems; tertiary prevention being treatment and rehabilitation—are not included. And in order to include those things, you have to cross agency boundaries to include, incorporate, the CDC and the NIH, several agencies within NIH, to cover the secondary prevention, and tertiary prevention treatment and rehabilitation, really, is the medical community of both the national and the local level as well as the public health community.

So, we're supportive of the concept of the bill that moves us in the right direction in terms of the integrity of science and the use of science for helping the decision making process, however, I still have reservations about this type of bill being able to solve the problem of being disingenuous in some degree with the public in terms of public health protection, because without secondary and tertiary prevention included, you're just not going to have a major impact on public health protection at this time.

Mr. EHLERS. Thank you. Dr. Gray.

Mr. GRAY. Well, my general opinion is that it is a good bill that lays out a proper approach using benefit-cost analysis as just one input into the regulatory decision making process; that it considers a broad range of benefits, both that are quantifiable and those that are not quantifiable.

My concern is that the risk assessment provisions may not be strong enough—and I mentioned this in my written testimony. The question of doing a better job of discussing the need for ranges of risk for quantification of uncertainty and variability and for full use of the scientific information that is available. Those sorts of things are very important given that risk assessment is the tool that will be used to estimate at least some of the benefits of any particular action that's being studied, and we need to make sure that we're doing a good job of the risk assessment to know that we're getting that side of the equation done well.

Mr. EHLERS. Thank you. Dr. Gold.

Dr. GOLD. I think the bill needs to emphasize the importance of educating the public about the known causes of human cancer, and by that I mean that we need to put this big perspective on cancer risk, because it's been cancer risk that's driven the risk assessment policies, the risk management policies. So, I think the lifestyle factors of Americans—sedentary, obesity, few fruits and vegetables, continuing to smoke. Anytime we're talking about risk and cancer,

we have to talk about how we're going to educate the public, and I think that we need a lot of research into that; how to get the message across so that somehow there's a behavioral change.

And, second, I think the bill should say something about trying to encourage the science to—I don't want to be in the position of saying we should put more money into science research, because that would sort of look like a turf issue for me—but I think that we're kind of on the pinnacle of some very important breakthroughs like Dr. Carlo was mentioning, and I just think that this ought to be the emphasis. How does Trichloroethylene cause cancer in a mouse liver? I mean, we need to know about that. And how does a human differ from the mouse or how does the rat differ from the mouse? We can't just continue to test at a potency and assess risk.

Mr. EHLERS. Dr. Omenn, you wanted to——

Dr. OMENN. May I just add, there's one glaring omission that strikes me in this bill which would reflect the comments of my colleagues. There is really *no public health context or even ecological context theme.* The bill is still based upon risk assessment plus cost-benefit analysis plus review plus more review of chemical-by-chemical, risk-by-risk regulation. What we're all saying here—and you illustrated in your opening remarks, Mr. Chairman—is that it would be much more understandable to the public and much more likely to be an effective way of protecting public health and environment if we could say we're looking at childhood asthma. We know it's on the rise both in incidences and mortality or lung cancers or birth defects or any other specific health problem and try to attribute the incidence rates of that problem to various causes—this is what Dr. Gold tried to say about the cancer exposures of many kinds. And then the question is how can you most make a difference in children's health? How can you make a difference in incidence and in mortality of asthma? How can you make a difference in every kind of cancer rate? In lung problems?

If we're worried about air pollution, we say in this report, for example, with regard to the tiered approach to section 112, Clean Air Act Hazardous Air Pollutants, that you ought to look at all sources of those prominent hazardous air pollutants. Start with benzene but put it in a class with polycyclic aromatic hydrocarbons. Then compare that with the health risks of criteria air pollutants and figure out where would you make a bigger difference for human health. It's very awkward to put this into statutory language, because soon it's translated, and it's subject to judicial review whether it was done exactly the way that Congress intended. I know from conversations that this is one reason why the Senate drafters have been reluctant to step up to this contextual opportunity. But it is so important, and I think that Congress must find a way to encourage the agencies to do it without putting them at risk for having nit-pickers take them apart on every little detail of how it could have been done through a judicial and regulatory review.

Mr. EHLERS. That is extremely important. What makes it even more complicated, of course, is the possible synergistic effects between these various risk elements. Dr. Carlo, you wanted to add something?

Mr. CARLO. I just wanted to add, Mr. Chairman, that our guess would be that most of the programs are already in place somewhere in the government to effectuate the types of public health protection steps that we are, I think, all talking about now, and the difficulty is that if something is going on within NCI for cancer screening, it is very difficult to get that incorporated into something that is the purview of EPA, and it is a—I think it's a difficult administrative problem that has to be dealt with if, again, we are going to be giving the full modicum of public health protection. I think those raw materials are in place; if not all of them, most of them.

Mr. EHLERS. Thank you all for your comments. My time is expired, and I would just add one little note: It's always amazed me, I've served at several levels of government and conducted many hearings on specific risks, and it's always puzzled and, in a sense, tragically amused me at the conclusion of a hearing to have someone talk to me vehemently opposed to a particular chemical of some such with 10 to the minus 6 risk level, something of that sort, and then afterwards, light up a cigarette and jump in the car to drive home, both of which have astronomical risks compared to the subject of the hearing. Mr. Lampson.

PUBLIC INVOLVEMENT IN RISK ASSESSMENT

Mr. LAMPSON. Thank you, Mr. Ehlers. We just went through some public meetings in Texas regarding the transport of Napalm after there was a huge concern expressed when we began to—or thought we were going to ship some some weeks or months back. The way we deal with the public and the way we communicate with them what our findings are—and, I guess, let me ask Dr. Omenn—one of the main conclusions of the Risk Commission is that there is a need to involve the public at all stages of risk assessment. Does that change risk assessment from a scientific process to a political one?

Dr. OMENN. No, it doesn't, Mr. Lampson. I think we documented in testimony before the Commission numerous compelling examples of valuable, technical input from ordinary citizens. As several of us have said, the exposure side of the hazard/exposure/outrage risk estimate is usually weak, and it's usually generalized and extrapolated through assumptions to some kind of a worst case scenario for the whole country.

Well, this country is made up of enormous heterogeneity in populations, in settings, in geography, and all that leads to variation in exposure. Many times, people were able to give information about how they are exposed, not hypotheticals, but how they are exposed through their patterns of eating. We have immigrant populations in this country who eat whole fish, so the notion that the head of a fish is irrelevant doesn't apply; they put it in a stew. We have examples of exposures in recreation as well as on the job that are remarkably high for some subgroups. Very often, those subgroups are extraordinarily reasonable—as was pointed out by George Carlo—about the remedies. They didn't necessarily want the whole country to have exposures reduced to such a level that even with extraordinary intakes they would still be perfectly safe. They were

willing to modify certain behaviors; have certain kinds of testing done, and other ways deal with this.

Another example of involving the community: the syndicated columnist, Neil Peirce, wrote many years ago about "The People's Park" in Seattle. It was the site of the former big gasworks, so it's no surprise that there's contamination with polycyclic aromatic hydrocarbons. About 8 years ago, there was observed by families using the site oozing of black material into the edge of the lake. So, the local health department came and took some samples, no big fuss. The regional EPA sent out their staff claiming to be protecting the staff in moon suits. Well, this was a terrific TV spot, and it scared the bejambers out of the neighborhood. So, the School of Public Health, University of Washington, and the local EPA all got together on this with a community group from, basically, the first afternoon to how to deal with what was now a media crisis and the possible horrible exposure to children and infants, and it worked out great.

Neil Peirce came back and wrote about the contamination in the park, and a few years later he wrote a third article about how the public process had worked, because people compared risks for the same kinds of chemicals that were coming from buses and cars stacked up right along the parking lots and pathways to the streets there. More importantly, measurements were made, real measurements on real contaminant levels, and they did find a lot of stuff at the edge, and they found a hot spot in a sand box, for goodness sake. Well, that was remedied in short order. Within a relatively—I don't know, maybe, 12 weeks or so—the park which had been closed was reopened with full participation of the community and the environmental groups and all the others who were worried about it. They were involved from defining the problem; to figuring out what studies should be done; to looking at the data as they came in, and to agreeing what comparisons were relevant and what comparisons were not relevant. For example—although I think it's very appropriate—they didn't want to put it to a comparison of smoking, but they were willing to compare being in the same kinds of chemicals.

That's just one example of many in which the engagement of the community makes a big difference in working the process. Even though it takes more time up front, it saves a tremendous amount of extended, protracted, bitter dispute at the later stages in many cases.

Mr. LAMPSON. I think it takes a special leadership, though, to pull that together and make sure that the emotionalism doesn't get into it, and making that happen, where does it come from? It's going to vary, I would assume, obviously, issue by issue and community by community.

Dr. OMENN. Exactly.

Mr. LAMPSON. Any thoughts other than that?

PUBLIC HEALTH PROTECTION OVER THE PAST 30 YEARS

Dr. OMENN. Well, the responsible parties, actually, are the public health officials. One of the problems in this country for the last 30 years, I regret to say, is that in the 1960's—with the Rachel Carson book in 1962 and leading up to Earth Day in 1970; the creation by

the Congress of EPA and OSHA and all the legislation that flowed—the public health community sort of defaulted. The notion was that these were hypothetical or emotional problems; that there were huge problems that needed to be maintained like protecting our food supply and restaurant inspections and really unglamorous stuff that we've come back to now that we realize we neglected those in recent years. Anyway, new agencies were formed, and the environmental agencies, as good as they are and as focused as they are on important issues for the public, *haven't been very strong about public health expertise or about working in a practical relationship with communities as opposed to regulatory regimes.* Where the local public health agency or the state public health agency is capable and where they work hand-in-glove with environmental agencies and the media and physicians and other health professionals—which is many places around the country, including Texas—this really can work.

Mr. EHLERS. Dr. Carlo, we'll extend your time a bit, so you can answer.

THE "80/20 RULE" IN PUBLIC HEALTH

Mr. CARLO. I wanted to add something, Mr. Lampson. What we have observed—and, again, I'm in full agreement with everything that Dr. Omenn has said here—but what we have observed is something that we call the 80/20 rule, and it seems to bear out, that with any issue whether it's Napalm transportation or environmental pollution, about 20 percent of the population has already made up their mind before you get there, so that when you do whatever it is you're going to do you have to focus on the 80 percent, and what we have found is that what those folks, the 80 percent who do have an emotional investment in any public health question or environmental question going in, what they're looking for is really two things: *they're looking for evidence that somebody is responsibly looking into the problem, and if there's a problem, it's going to be solved, and the second thing they're looking for is evidence that the government is watching to make sure it's done right.* And what we're finding this now is a new sort of concern in the public. Now, they don't really—that 80 percent, *they don't want to hear about numbers, because they want to go to a soccer game.* They've got to take their kids to school. *They want to know that the problem is being addressed by those responsible* and that the government is watching their best interests, and when you step into that group with risk assessment numbers or you start talking to them about animals, rats, and 10 to the 9th power risks and 10 to the 6th power risks, it not only creates a blank look on their face, it angers them. It causes agitation, *because you're now intruding on their time.* For them to be able to make a contribution to this debate they have to learn about rats; they have to learn about mice; they have to learn about numbers, and they want to go to a soccer game; they've got to worry about groceries. So, that is what the newest thing that we're finding, and, historically, what many of us have done in the public health community is that we have erroneously focused on the 20 percent, because those are the guys who are most noisy and who have the most impact up here—you know, the advocacy groups, and those types of folks and even the indus-

try; the industry is in that 20 percent too, you know. That slices 10 percent each way—10 percent of everything is bad, 10 percent of everything is good. So, you've got to make your policy decisions based on the masses, and what we're finding is that the masses only want two things: that somebody is taking responsibility for making sure that it's being looked at and if there's a problem, it's going to solved and that the government is playing a role as watchdog.

Mr. EHLERS. Will the gentleman yield?

Mr. LAMPSON. Sure.

THE "80/20 RULE" AND THE COST OF REDUCING EXPOSURE RISK TO ZERO

Mr. EHLERS. I just wanted to pick up on that, because in my personal experience—and I've worked at local and state governments as well—generally, the problem that I've encountered is that the 80 percent gets very concerned, because there are some Chicken Littles running around saying, "This is the end of the world, and don't trust anyone; just trust me." So, politically, I think that becomes the major problem. Apparently, you avoided that in the case in Seattle. But I've had cases where it's virtually impossible to get a solution, and, in fact, the entire State of Michigan developed that over the PBB contamination some time ago whereby the Governor eventually said the only solution is to kill every cow that had ingested it, and the State spent $10 million burying one ounce of PBB and several thousand cows that were killed and buried just to get rid of that one last ounce and say, "Okay, it's done."

PUBLIC HEALTH "BEDSIDE MANNER"

Mr. CARLO. Another thing that we've observed in some of this work along those lines, Mr. Chairman, is that when—you know, in most instances, by the time a responsible public health person comes on the scene with the public, they already have an answer. They say, "Well, this risk is not big or this is not a problem. This is what you have to do to fix it." Now if you step back and you look at how you would treat that individual as a patient in a doctor's office—because public health is really looking at the population as the patient—the first you would do is something called bedside manner. The first thing you'd do is you say, "All right, tell me what your concerns are. Talk to me about the problem." And what we do is we create a scenario where we walk into the doctor's office and say, "Ah-ha, Mr. Chairman, here's the solution to your problem", before you've even established to us—we haven't had a dialogue. And that's another thing that's very important that we're observing is that there has to be a concentrated effort on establishing a bedside manner, a level of trust, before you come in with your data; before you come in with your solutions.

Now, as Dr. Omenn points out, this is more probably in how the public health community implements this, and it's not really a regulatory concern, but the regulations and the legislation needs to accommodate some time for developing trust—we call it bedside manner; there may be a better word for it.

MARKET SURVEILLANCE AND "RAPID TRIGGER" RESPONSE TO PUBLIC HEALTH AND OTHER THREATS

Mr. EHLERS. Mr. Lampson has had to depart, apparently. Mr. Roemer has arrived, but I'll take my second round and then introduce Mr. Roemer if he wishes to ask any questions.

I have several—Mr. Carlo, while I was listening to you, I thought of a question I wanted to ask. You talked about the importance of first-market surveillance triggering intervention and so forth. Now, that works for a disease model, but that does not work—I mean, I wouldn't say it doesn't work, but there are a lot of other risks that we have to evaluate other than disease risks. Is that not correct? For example, trauma of various sorts, accidents.

Mr. CARLO. That's correct, yes.

Mr. EHLERS. Would you modify your statement relating to those or would you still say that applies?

Mr. CARLO. Well, I think that, in general, if you define whether it's trauma or whether it's a disease, the public health intervention approach applies. You know, primary, secondary, and tertiary prevention as basic principals apply pretty much across the board to anything that would be considered a public health threat.

THE RELATIONSHIP BETWEEN RISK ASSESSMENT AND COST-BENEFIT ANALYSIS

Mr. EHLERS. Okay, thank you, appreciate that. Dr. Omenn, one problem we run into here a lot, almost everyone that I meet in the Congress wants to tie risk-benefit analysis to cost-benefit analysis, and I've heard very little discussion about it—Dr. Gray, I think you mentioned something about that. Could you try to clarify for the Committee and for the record what the relationship is between risk-benefit analysis and cost-benefit analysis and when each should be used?

Dr. OMENN. Okay. In general, risks and benefits in this are the same. Let's say the benefit is the reduction of risk. So the problem, of course, is when——

Mr. EHLERS. Are you saying there's a direct math, medical inverse relationship?

Dr. OMENN. Exactly, when there's a trade-off, sometimes, in order to get one benefit we trigger other problems—George Gray and John Graham have written a nice case study book about trade-offs where, in fact, reducing one exposure leads to a substitution or leads to complications which generate new risks. That's a very important kind of analysis that the people can understand, whatever their feelings are about the expenditures of cash to comply with the regulation, because you're trading health problems for other health problems.

The cost-benefit analysis runs afoul of the problem of putting dollar signs on everything, and very imprecise estimates are often manipulated for a particular interest either to minimize the cost of complying or to maximize the complying. For example, you gave us $15 billion to $120 billion—that's quite a sizeable range. It's common for the economists who submit opinions in these cases to come up with a very precise number, sometimes to the pennies, and not to acknowledge the assumptions; the choice of the data set; all of

the things that are being asked of the risk assessment folks but are not asked of the economists.

In S. 981, in fact, in the bill itself, it just mentions do the cost-benefit analysis and report the results and consider them. The Senate report actually goes into much more detail and does suggest that the choice of data and the assumptions and the extrapolations should be revealed which is input that we have laid on them.

There's another big problem: certain health benefits are hard—benefits are much harder to put dollar signs on. So, how much do you value for the prevention of a death? At different ages, is it different? In different populations? By different causes? How much suffering goes along? And, of course, there are many non-death endpoints in health, as you just pointed out. So, putting dollar signs on is a huge problem. Then you have to bring the costs and the benefits together. I recall challenging Mr. Jim Miller when he was in the regulatory district role for OMB early in the Reagan period. "What is the decisional criteria?" And he said, "Regulatory actions should be governed by the benefit-cost ratio." I said, "Well, is it the ratio or is it a dollar more or two dollars more? By how much margin? With what uncertainty?" And he said, "Gil, we just haven't considered anything like that." Well, somebody has to consider it or you to have to back off as S. 981 does and said just "consider the costs" and make your judgment and justification in qualitative terms. I think that's unreasonable, actually.

In the case of the Executive Orders of five Presidents now, the regulatory officials have been under guidance to do this kind of evaluation of the costs, and, most importantly, the cost-effectiveness of alternative ways of achieving similar benefits. So, if the notion is to bring ozone below a certain maximum level on the worst day or the fourth worst day of the year, there are many different ways that could be done with control of vehicles and stationary sources.

Well, for the same health benefit, you would surely find agreement that you should do it for the least cost. That's the cost-effectiveness analysis (CEA), which the economists would classify as a subcategory of costs-and-benefits analysis. CEA doesn't require that you put a dollar value on the benefit. It just means to say you set a benefit that you want to achieve, and you find different ways of doing it, and choose the most cost effective.

It's a big subject, obviously. We have in our reports commissioned papers from Resources for the Future and from other sources. There's a vast literature. The bottom line is to try to make the regulators, the public, and the affected parties come to some common understanding of what the benefits are and what the costs are in a variety of ways and try to at least understand each other better. It's about all we can do at this stage, I would say.

Mr. EHLERS. Thank you. I think that's a good summary, and what is required so often is just plain good judgment and by educated minds, and my frustration with the process is that very often the ones making the final decision are not that educated in the field and try to take the safest possible approach, and then you no longer have good judgment.

Particularly bothersome to me is decisions which severely regulate one quantity without recognizing, as you pointed out, what will

people use for a substitute? And that's often the worst example, and I appreciate Dr. Gray's work on that.

I think my time has expired again. We'll recognize Congressman Roemer for—and, incidentally, without objection, your opening statement, if you have one, will be entered into the record.

STATE AND LOCAL GOVERNMENT RANKING VS. FEDERAL RANKING OF ENVIRONMENTAL RISKS

Mr. ROEMER. Thank you. I want to thank you, Dr. Ehlers, for this very important hearing, and we have such a distinguished panel of doctors before us, so I welcome your testimony and your expertise and counsel on a very important topic. I'm also very personally interested in this topic and not only have encouraged this Committee to look at this and evaluate this subject matter but also have offered amendments and bills on the Floor addressing risk analysis and risk management and risk assessment and so forth. So, I'm delighted to have all of you before our Science Committee today.

I also have to tell that as I made my way in from another meeting—and I apologize for being late—Nick Lampson was making his way out to a meeting, and he said to me, "Geez, you got to get in there, Roemer, it's very interesting today." That's not always the case.

[Laughter.]

That's a real compliment to all of you and to our Chairman that we're engaged in some informative and interesting and important debate for the direction of the country in a very important public policy area.

Let me start with I think a pretty basic question. There have been different experiences of state and local governments in this whole area in conducting comparative risk assessments. Should States and localities be permitted to set their own environmental priorities or should there be a federal ranking of environmental risks? Dr. Omenn, do you want to start?

Dr. OMENN. Sure. I think there absolutely should be a federal effort to bring things together. But, in fact, the federal effort, led by EPA but with involvement from many other agencies, has, in large part, been to stimulate state level and local level comparative risk activities. Now, we have a problem. In fact, in our report from the Risk Commission, we went to great lengths to distinguish *"comparative risk assessment" from "comparisons of risk"*, because they are used entirely differently. What you're asking about is sort of the agenda. What kinds of risk? What's described in S. 981 is the risk assessment for control of benzene from a particular source compared with control of benzene from other places or compared with pesticide risks or other causes of cancers. That's a chemical specific comparison, we would call "comparisons of specific risk."

What we're addressing and the question you've asked us all is what about air pollutants of various categories, water pollution, smoking, etc., I take it. We had such a process in Seattle called the Environmental Priorities Project of the Mayor. He put his OMB on it and his environmental guy on this; there was a very broadly drawn community group; there was a technical panel and a policy

panel, and we all came together; a few of us were on a steering committee.

In the Risk Commission report, I think we had something like 30-some States now have done projects—stimulated with money from EPA—of their own; looking at their own environments and their own public values and activists and industries and municipal-like problems and trying to understand where the problems are most salient and where they can make the most difference. The lists are different from region to region.

California had a very expensive process which ended up in, basically, chaos. They tried to make it so inclusive—let's say, sort of, their public welfare dimension of secondary effects of air pollutant language, and they wanted to have, of course—very important, recognized all around the country now—about equity considerations, environmental justice matters. *These social, economic, political, and ethical matters really energize these discussions, yet in a way that takes them far from the scientific base, and I think they're a central context. It's very important to be respectful of differences of views and values through this country; it's a very diverse population. In some way we have to respectfully consider all those values* in the combination of looking at the responsibilities of local government, state government, and the feds to control what's mandated by Congress under statute and implemented by the agencies under regulation and exhortation.

Mr. ROEMER. Anybody else? Dr. Carlo.

Mr. CARLO. Mr. Roemer, I also believe that there should be a federal framework for this type of program. I guess the most salient reason is to—this is becoming very, very complicated, and in order to pull the expertise together, in order to make the right decision, the right decision about this type of framework, it's more efficient to do it at the federal level. Conversely, the implementation of public health intervention programs, necessarily, is local, and this has to do with the need for bedside manner; the need to have the public be receptive to those who are trying to make these interventions work. And, we, for example, have observed in some of our work that when it comes to federal regulators with their recommendations for intervention or communications about risk, that they have very high credibility with bad news but almost no credibility with good news. So, that if you're talking about the Federal Government saying, "Look, there's a problem with air pollution," people believe that, but when the Federal Government says there isn't a problem with air pollution, nobody believes it, and——

Mr. ROEMER. How about if they come up with a solution to air pollution as good news, do they believe that or how skeptical——

Mr. CARLO. Well, the solution is something that requires bedside manner, and bedside manner—as difficult as it is to admit—at the federal level it's almost impossible to be warm and fuzzy. So, the implementation is the type of thing that really requires the local officials, the local public health people, the local medical community even with an intervention, so that if the role of the Federal Government is creating the framework; creating the pathway; creating the guidance, then that is probably an efficient balancing of local and federal input.

Mr. ROEMER. Dr. Gray.

Mr. GRAY. Well, thank you. I just have a brief comment, and I think your question—what can we learn from what's gone on in the comparative risk exercise as it's been undertaken to this point—is a very, very good one. And I think that it's clear that when local citizens are involved in a process; when they bring their values and their knowledge to the process, it helps a lot in getting education going both ways, both in them understanding what are some of the questions that the risk analysis or the, sort of, the technical community can bring, but the technical community also understanding what are the values that this group puts on different types of risk? What are things they want to know about?

And I think that there are examples, at this point, some other folks have tried to draw some of the lessons—it's in Dr. Omenn's commission; Rick Menard has written several pages; there's a book from Resources for the Future—and they really do tell us that in these *comparative risk exercises, we get communication; we get education, and often get considerable consensus about sizes and sources of risk in a community.*

Now, something that Dr. Omenn mentioned is that there are, indeed, differences across jurisdictions; across States; across localities in what are considered the big risks, and I think that suggests *that there may well be a need for local fixes.* But I also think that the Federal level, as the others have said, does have an important role in setting an agenda and in doing some things that are bigger picture, looking to the future. What are potential risks to technologies and that sort of thing?

Mr. ROEMER. So, Dr. Gray, you agree with Dr. Carlo's framing of the issue saying that the federal role should be kind of putting together a federal framework for this kind of assessment?

Mr. GRAY. Well, in many ways that has happened. As you know and others have mentioned, many of these comparative risk studies have been funded at this point by the EPA who has had a pretty well-defined framework for how these should take place. So, that there has been a framework even for comparing risk that's come from the federal level. Whether that's the best way to do it is open to question, but there are questions that are bigger; that are transboundary; that go across States; that are international and national that do require attention and fixes at the federal level, but in many cases, as Dr. Omenn's few stories have shown us—and there are many other examples—often, the happiest solutions come when things are done at the smallest level.

Mr. ROEMER. Dr. Gold.

Dr. GOLD. Yes, I think that one of the most important things at the federal level is to emphasize attributable risk; that is, what proportion of the total risk of some disease is due to the problem being defined? I think there is just a leadership role that the scientific community or the regulatory community needs to convey.

And I'd like to make one other comment about the federal level on regulatory policy that there really is an enormous range across-regulatory agencies in the way scientific data is dealt with in making risk assessments. I have done quite some work looking at OSHA regulations, and the permitted levels for workers are extraordinarily high compared to the doses that give rodents cancer. Let's take methylene chloride as an example. The regulatory level

permitted on a milligram per kilogram body weight basis for workers was a million times higher than the MCL for methylene chloride in drinking water. Recently, OSHA dealt with methylene chloride and reduced the PEL 20-fold, but it's still almost 100,000 times higher.

There needs to be some evaluation of scientific information in an interagency framework at the federal level, and I don't know that the standard has to be: 1 in 1,000 for workers; 1 in a million for drinking water or whatever, but the evaluation of the science is distorted across the Federal Government.

Dr. OMENN. I'd just add to Dr. Gold's comment about interagency reminds us that EPA is hardly the whole picture of what needs to be done to protect health and environment, and at the local level there's an extraordinary opportunity for synthesis and integration. In the Environmental Priorities Project I mentioned at the city level, the group came to the realization that the big issues were transportation and growth management, because water pollution, air pollution, all kinds of other problems really came back to those societal processes, and, there, the local government has most of the action.

JUDICIAL REVIEW

Mr. ROEMER. Let me ask one final question—and I apologize if you addressed this, Dr. Omenn, in your initial remarks or your testimony, but I think you have addressed this in your report, but I'd like to get it on the record—one of the most controversial issues surrounding regulatory reform legislative proposals has to do with judicial review of scientific matters such as risk assessments. Should courts specifically review agency risk assessments?

Dr. OMENN. No. I agree that the final rule is up for judicial review. This is standard practice, and it's appropriate for the courts to consider the extent to which the agency gave due consideration to all factors; to proper analyses, and to consideration of views—technical, economic, and other—but most courts, and certainly the first district here, have held that they are reviewing the process and the responsibility of the agency, not the technical competence of the particular analysis. I think that's right.

Mr. ROEMER. Thank you. If you're going to agree with me, Dr. Gray, I'd be happy to recognize you.

[Laughter.]

I happen to agree with Dr. Omenn, but go ahead. I think we're going to get some other opinion on the record here. Go ahead, Dr. Gray.

Mr. GRAY. No, we're not.

Mr. ROEMER. Oh, good.

JUDICIAL AND PEER REVIEWS

Mr. GRAY. In fact, I'm going to agree with you as well and say, in fact, that what's been in a lot of the regulatory reform legislation that will serve a lot of that same function and accomplish the ends that people are interested in in getting sound science and risk assessment is *a strong peer review function. If that happens up front and it's the scientists who are evaluating the technical merits, and the courts can evaluate the process.*

Mr. ROEMER. I appreciate it. Dr. Carlo.

Mr. CARLO. I'll agree with you too.

Mr. ROEMER. Good.

Mr. CARLO. And, again, to underscore what Dr. Gray said, that the currency of science, the language of science, is peer review, and we do that a certain way and within the framework of scientists talking, sometimes it's bloody, and sometimes it's nasty, but it really needs to be kept in the family, and the process works; it's been working 300, 400 years. So, when you interject something like a judicial review on a process that has its own language, it has it's own process, I think you're asking for trouble.

Mr. ROEMER. Thank you. Thank you.

RISKS FROM NATURAL AND SYNTHETIC CHEMICALS COMPARED

Mr. EHLERS. The gentleman's time has expired. Just for your information, I agree also, but I'm not sure we can ever keep it all in the family in our present judicial system.

Just a few quick questions, then I'll be finished for the day. First of all, Dr. Gold, can you just give me a rough estimate of the danger of cancer from what you referred to as natural chemicals as compared to that from synthetic chemicals for the average citizen today?

Dr. GOLD. I would say that for the average citizen, the risk from synthetic industrial chemicals is minute, maybe up to 3 percent of cancer in the United States; probably not more than that, because the exposures are so low. That doesn't include the workplace. Maybe a few more percent is possible from workplace exposures, but even among the workplace, chemicals that have been identified as human carcinogens, nearly two-thirds of them are naturally occurring; they're in an industrial context. However, I'm including cigarettes and tobacco as a natural chemical—a series of natural chemicals which they are, and burning is also a natural event, so you might have certain reservations about those categorizations, but since we know that the causes of human cancer are cigarette smoking, dietary imbalances, endogenous hormones, chronic infections, all of those are naturally occurring. So, that's where the big risks are.

FEDERAL "RIGHT-TO-KNOW" INITIATIVES—EPA'S TOXIC RELEASE INVENTORY (TRI)

Mr. EHLERS. All right, thank you. And, Dr. Gray, I just want your opinion about government right-to-know initiatives, and if others want to comment on that too, that would be fine, but the time is getting late. I'm thinking about the EPA's toxic release inventory which has been immensely popular among some groups and some individuals, and do you see this is an effective way to communicate relative risks to the public? How do these requirements affect industry? How do they inform or misinform the public and create apprehension or appreciation? And is there a better way of communicating risks other than the toxic release inventory? I'd appreciate your comments on that.

Mr. GRAY. Well, thank you very much for the question. I guess maybe you don't have something else to do, because you've asked me that question to keep me busy for the rest of the afternoon.

[Laughter.]

But very briefly——

Mr. EHLERS. Well, I have a 12 o'clock meeting.

Mr. GRAY. Okay, we'll make sure you make it. Very briefly, I've written and spoken on this many times. I think that one of the problems we have is that the information in right-to-know types of programs frequently isn't of the sort we would like for comparative risks because it's not risk.

The toxic release *inventory reporting pounds of material emitted, even materials accounting sorts of approaches that are being advocated as additions to toxic release inventory would focus on pounds of material used don't give us the context, don't give us the information to really make the sorts of comparative risk that would help citizens understand where this falls in the range of risks to their health.* So, that the short answer is these sorts of things, *at best, are confusing; at worst, are misleading and really the only way to fix these things is to try to get them back on some sort of a risk basis where we consider both exposure and the inherent potency of the compounds.*

Mr. EHLERS. Well, let me expand the question just a bit. It's been proposed that we expand the toxic release inventory to also include chemicals on site, kept on site at every plant in the United States. I would judge your answer would be that would not be useful also?

Mr. GRAY. Unless it was somehow continued to think about what on earth would be the risk of this being on site? The presence of something isn't a risk; it's exposure to it.

Mr. EHLERS. Dr. Carlo?

Mr. CARLO. I'd like to add—excuse me, another thing that we've found is that the information threshold for the general public is very low, and in that 80/20 breakdown, that 80 percent of the population, when you present them with a list of chemicals, like in a right-to-know or a toxic release inventory, what you do is you begin to move that 80 percent into the 10 percent on either side. Just by having that, causes agitation; by having that, causes fear among the population, and I know this is a very difficult area, because it sounds like keeping information from the public, but what we've identified—we don't quite have a solution for it yet—is that by releasing information you create public health anxieties. It's not public health education that this is promoted as. It is agitation among the public, because they don't understand what it means, and that in and of itself becomes a public health problem.

Dr. OMENN. I'm an advocate, and I've always been an advocate of TRI. Lester Lave, an economist at Carnegie Mellon, and I worked with the National Academy and multiple environmental organizations in the eighties when TRI was coming out to try to help people understand that these pounds are not risks; that's exactly right, and that you have to have some way of categorizing chemicals as high, medium, and low potential hazard, but as long as it's a not too rapidly moving target, it's actually useful to give some data to sort in the local communities.

I've chaired for many years the Environmental Advisory Council for Rohm and Haas Company, a big chemical company in this country, and every single one of their plants around the United States and around the world, they release this information, and they have

community action councils with interested, local people including the fire chief and other people who have a reason to want to know. TRI is a basis for discussion of what the chemical processes are, what is released, and what's not released.

I think the proposal you've mentioned is very unfortunate, *because under the banner of toxic release, it's talking about chemicals properly managed on site.* They already have chemical by-products properly disposed of included in TRI, and that's not appropriate either. What companies have done is to list separately what's properly disposed of in pre-mix facilities and other off-site and on-site facilities and what goes into the air and anything that goes into the water; anything that goes anywhere else. And that is appropriate. I think it's been used well in many communities. It's been distorted in many headlines, which is agitating to people trying to keep rational discussion, but, on balance, it's been useful; it's opened windows for discussion, and I support it.

Mr. EHLERS. Thank you. Dr. Gold.

IMPROVEMENTS TO THE TOXIC RELEASE INVENTORY

Dr. GOLD. Yes, I support the idea of the right to know, but I think that the right to know should always be more rather than less, so I would say for the toxic release inventory, there ought to be a production volume, a released volume, and a human exposure estimate.

And I just want to take this opportunity to describe the importance of the exposure estimate. This overhead is from a paper I recently did on pesticide risk, and I looked at the two components of risk, carcinogenic potency from a rodent study and exposure assessment. I used the National Academy Report from 1987, *The Delaney Paradox, Regulating Pesticides in Foods,* and I looked at the EPA toxicology branch memos and how the risk assessment for carcinogenic potency was derived. I recalculated potency using my measure of potency which is just the inverse more or less of the EPA Q1*. I found that potency estimates were close no matter what, within a factor of two most of the time—a few discrepancies based on whether life table data was used and a few misprints. But when you look at the ratio of exposure assessment, you get an enormous range. Here, I compared the EPA's theoretical maximum residue contribution of the exposure to people in the food supply for various rodent carcinogens that are used as pesticides, and I compared that to the FDA's measured Total Diet Study estimate of residues. The FDA conducts a survey every year for the last 25 years in which they take samples from the supermarket in different geographic areas in the country and mash it all together, and they can detect at one part per billion. It's not a perfect system. There are certain design problems and so on, but over the years, we've got very standard low, low levels. All of these risks were defined as greater than one in a million by EPA, using a theoretical maximum. Now, the theoretical maximum tells you—let's say there are 54 chemicals allowed on tomatoes in California—which is the case—the theoretical maximum assumes that every farmer uses all 35 pesticides on 100 percent of his crops and that the person gets in his food supply what there is on the plant when it is harvested; all of those as-

sumptions are so drastically wrong. No farmer uses more than five, and one-third of them don't use any. It's just so distorted.

This last column in the overhead reflects that distortion. The four chemicals at the bottom of the top section—Alachlor, Captafol, Cypermethrin, and Oxadiazon—were never detected by FDA in 25 years at one part per billion. They were never detected in the food supply, but the risk estimate based on the theoretical maximum is more than one in a million for cancer risk. For those that were detected, there's a range of 100,000 to 600 times difference in the risk estimate based on measured exposures.

So, I think that if you're going to have the toxic release inventory, you need to give that kind of a measured exposure estimate, because, otherwise, people look at how many tons of pesticides are used, and they think that they're first getting—the amount of that pesticide they get is somehow relevant to the relative ranking of the amounts that are used, which isn't the case at all. One pesticide may have many more tons released, but people may be getting much less of it. So, you get a distorted view if you don't add in some exposure assessment. Thank you.

ORGANOPHOSPHATE PESTICIDES AND FRUIT AND VEGETABLE PRICES—RELATIVE CANCER RISKS

Mr. EHLERS. I agree with your comments, and I assume that you would not think it wise for the EPA to do as they propose to do and that is to ban a whole group of pesticides, the organophosphates, which the fruit farmers and vegetable farmers all say will result in greatly increasing fruit and vegetable prices?

Dr. GOLD. Right. So, I would say two things about that: first, we all carry around in our tissues lots of organophosphates that we get which are naturally occurring in potatoes.[1] We eat solanine and chaconine, and we all have it in our tissues. So, you need to look at those amounts that we're taking and compare to the pesticide residues. Those two are among the chemicals I proposed to NTP to be tested for carcinogenicity. And the second is the issue of cost. Clearly, poor people in this country spend a higher proportion of their income on food. One really doesn't want to increase the price of fruits and vegetables, because, (a) it falls disproportionately on the poor, and, (b) because it's probably going to lower intake, and we know that it's protective against cancer. I mean, that's a 50 percent decrease in cancer risk for most types of cancer among people that eat the highest amount of fruits and vegetables; the quarter of the population that eats the highest amounts has half the cancer risk of those that eat the lowest.

Mr. EHLERS. Fine. One final comment, Dr. Carlo, you observed that the Federal Government is not very warm and fuzzy. I would just like to have the record show that Members of Congress are very warm and fuzzy.

[Laughter.]

Mr. CARLO. All right.

Mr. EHLERS. Mr. Roemer, do you have any further questions?

Mr. ROEMER. I don't, but I certainly concur with Nick Lampson's statement to me on the way in and on his way out that this has

[1] This sentence should read: "We all carry around in our tissues chemicals that, like organophosphates, are neurotoxins, but these are naturally-occurring in potatoes."

been a very interesting and very informative panel, and we hope that you can come back sometime and continue to update us on the progress in these areas.

And also, Dr. Gold, I sit on the Education Committee where we're working on OSHA reform, responsible OSHA reform, which will continue to protect the working people but also reflect more common sense in that kind of reform, and where you have suggestions in that area, we'd be happy to work with you as well. So, thank you again, and thank you, Mr. Chairman, for your hard work in this area.

Mr. EHLERS. And I want to thank the panel. It's been an exceptionally good panel and a very good hearing. We certainly appreciate your words of wisdom and I hope that we have some success in this body and the one on the other side of the Rotunda in getting a good bill that deals properly with the entire topic of risk assessment. Thank you very, very much for sharing your knowledge with us and taking the time to do it.

This meeting is adjourned.

[Whereupon, at 12:48 p.m., the Subcommittee was adjourned.]

[The following material was received for the record:]